Palmitic acid (16:0) consumption in plant-based diets and its effect on metabolic health.

by

Thomas Hothersall

A thesis submitted in partial fulfilment for the requirements for the degree of Doctor of Philosophy at the University of Central Lancashire

February 2026

University of Central Lancashire
UCLan

RESEARCH STUDENT DECLARATION FORM

Type of Award	**Doctor of Philosophy**
School	**School of Health, Social Work and Sport**

Concurrent registration for two or more academic awards

I declare that while registered as a candidate for the research degree, I have not been a registered candidate or enrolled student for another award of the University or other academic or professional institution.

Material submitted for another award

I declare that no material contained in the thesis has been used in any other submission for an academic award and is solely my own work.

Collaboration

The Gas Chromatography Fatty Acid Analysis (Chapter 4) was a collaborative project with the School of Health and Agricultural Sciences at University College Dublin. I performed all investigational, experimental and analytical procedures, with guidance and support from Dr Marco Garcia-Vaquero and Dr Raquel Cama-Moncunill.

Use of a Proof-reader

No proof-reading service was used in the compilation of this thesis.

Additional declaration(s):

I have been practicing veganism/consuming a plant-based diet for many years. However, I have (to the best of my ability) maintained objectivity throughout and ensured no personal biases have influenced any part of study design/methodologies, nor interpretation of results.

Signature of Candidate:

Print name: Thomas Hothersall

Abstract

Palm oil (PO) contains approximately 50% saturated fatty acids (44% palmitic acid) and represents one of the most widely utilised vegetable oils in the world. Growing evidence suggests that palmitic acid may exacerbate metabolic inflammation associated with a range of chronic diseases. The 'Western diet', or 'Western dietary pattern' is the most common diet in the United Kingdom, suggesting that palmitic acid may make a significant contribution to overall dietary lipid intake. The main objectives of this research were to 1: evaluate the evidence on the role of palm oil and palmitic acid (16:0) on metabolic health and the gut microbiome, 2: assess the awareness of the United Kingdom public of palm oil and palmitic acid; their existence, their prevalence, as well as their potential health effects, 3: analyse the fatty acid composition of common food items, with an emphasis on palmitic acid content and 4: screen a panel of gene expression biomarkers in participants with different levels of dietary palmitic acid intake and assess their potential role in metabolic health.

A systematic review was performed to examine the evidence on the role of palm oil and palmitic acid (16:0) on metabolic health and the gut microbiome. A total of 132 studies are included in the final review. A total of 251 members of the United Kingdom public completed a questionnaire containing demographic information, details regarding their anthropometry, exercise frequency/intensity, sleeping patterns and dietary habits, as well as their perceptions, behaviours and beliefs towards palm oil and palmitic acid. A total of 62 of these 251 respondents also provided a 4-day food diary. A total of 70 food samples, representing common food groups (vegetable/seed oils, palm-based foods, animal-origin foods, dairy foods, plant-based/vegan alternatives, nut/seed-based foods, miscellaneous/snack food items) were analysed for their fatty acid composition. A fatty acid profile of 38 different fatty acids was developed using gas chromatography. A total of 18 participants provided one intravenous blood sample for gene expression analysis, as well as anthropometry and food diary (4-day) data, before being allocated into one of the palmitic acid intake groups (low, medium and high) based on their daily palmitic acid intake.

The outcomes of the review concluded that the majority of studies provide evidence to suggest that palm oil, or more specifically palmitic acid intake, contributes significantly to various chronic diseases associated with metabolic syndrome, such as type 2 diabetes, cardiovascular disease(s), stroke, and nutrition-related cancers. The survey study provided evidence to suggest

that palm oil/palmitic acid awareness should be recognised as an important component of health and nutrition literacy. Fatty acid analysis of several food items across multiple food groups highlighted the abundance of palmitic acid within the diet, regardless of dietary inclination. Assessment of mRNA expression provided evidence to suggest that palmitic acid intake is positively associated with an undesirable gene expression profile, although metabolic adaptation(s) may occur above a certain threshold or due to long-term exposure of palmitic acid. Prospective randomized control trials are critical in investigating these suppositions further.

Table of Contents

Chapter 1. Introduction and Review of Literature _____ 1

1.1 Background _____ 1

1.1.1 Global Epidemic of Chronic Diseases _____ 1

1.1.2 COVID and Chronic Diseases _____ 1

1.1.3 Role of Diet in Chronic Diseases _____ 2

1.1.4 Plant-based Diet/Veganism _____ 3

1.2 Lipids in food _____ 4

1.2.1 Dietary Lipids _____ 4

1.2.2 Oil/Lipids in industrial food processing _____ 5

Review of Literature _____ 8

1.3 Chronic diseases _____ 8

1.3.1 Metabolic syndrome _____ 8

1.3.2 Overweight and obesity _____ 10

1.3.3 Type-2 diabetes _____ 11

1.3.4 Cardiovascular disease _____ 13

1.3.5 Metabolic dysfunction-associated steatotic liver disease (MASLD) _____ 14

1.3.6 Cancer _____ 15

1.3.7 Neurodegenerative disease _____ 17

1.4 Pathophysiology of chronic disease _____ 18

1.4.1 Chronic inflammation _____ 18

1.4.2 Oxidative stress _____ 22

1.4.3 Glucose homeostasis _____ 25

1.4.4 Dyslipidaemia _____ 27

1.4.5 Gut microbiology _____ 29

1.5 Role of dietary habits in health _____ 32

1.5.1 Lipids in health and disease _____ 33

1.5.2 Lipids in food processing and preservation _____ 36

1.5.3 Entry of Palm oil into the food chain _____ 38

1.5.4 Palm oil, palmitic acid, and metabolic health _____ 44

1.6 Research Gaps/Limitations 53

1.7 Conclusions 53

Chapter 2. Effects of palm oil, palmitic acid, and their derivatives on metabolic health: a systematic review 54

2.1 Chapter Overview 54

2.2 Introduction 55

2.3 Methodology 57

2.3.1 Validity statement 57

2.3.2 Literature sources and search criteria 58

2.3.3 Data extraction 58

2.3.4 Eligibility assessment 58

2.3.5 Risk of bias assessment 59

2.3.6 Sorting of literature 60

2.3.7 Meta-analysis 61

2.4 Results 61

2.4.1 Studies included 61

2.4.2 Presence of bias 63

2.5 Summary of Findings 72

2.6 Discussion 113

2.6.1 Limitations of the available literature 118

2.6.2 Strengths and limitations of the review 119

2.7 Conclusions 120

2.7.1 Conflicting interests and funding statement 120

Chapter 3. Awareness and perceptions of the health effects of palm oil and palmitic acid 121

3.1 Chapter Overview 121

3.2 Introduction 122

3.3 Methodology 126
3.3.1 Ethics application 126
3.3.2 Survey on awareness of palm oil/palm oil derivatives 126
3.3.3 Dietary intake of palm oil/palmitic acid derivatives 128
3.3.4 Palmitic acid intake 128
3.3.5 Statistical analysis 129
3.4 Results 130
3.4.1 Participant characteristics and lifestyles 130
3.4.2 Participants awareness of palm oil/palmitic acid and their derivatives 133
3.4.3 Participant demographic/lifestyle together with palm oil health perceptions 134
3.4.4 Dietary data 139
3.4.5 Palmitic acid content 141
3.5 Discussion 142
3.5.1 Limitations 146
3.6 Conclusions 146
Chapter 4. Fatty acid composition of common foods consumed in Westernised diets 148
4.1 Chapter Overview 148
4.2 Introduction 149
4.3 Methodology 152
4.3.1 Rationale 152
4.3.2 Selection of food samples for fatty acid composition analysis 153
4.4 Fatty acid analysis 162
4.4.1 Preparation of food samples 162
4.4.2 Preparation of Fatty Acid Methyl Esters (FAMEs) 168
4.4.3 Extraction of Fatty Acid Methyl Esters (FAMEs) 169
4.4.4 Analysis of Fatty Acid Methyl Esters (FAMEs) 170
4.4.5 Expression of fatty acid content based on dry matter content 174
4.4.6 Fatty acid composition data 174

4.5 Results 175
4.5.1 Edible vegetable oils 175
4.5.2 Dairy foods 179
4.5.3 Nut/seed-based foods 183
4.5.4 Animal-origin foods 187
4.5.5 Plant-based/vegan food alternatives 190
4.5.6 Miscellaneous/snack food items 197
4.5.7 Palm-based foods 202
4.6 Discussion 206
4.7 Conclusions 213
4.7.1 Acknowledgements 214
Chapter 5. An assessment of gene expression biomarkers linked to metabolic health in participants with different levels of dietary palmitic acid consumption 215
5.1 Chapter Overview 215
5.2 Introduction 216
5.3 Methodology 219
5.3.1 Ethics 219
5.3.2 Participants 219
5.3.3 Anthropometry 220
5.3.4 Diet diary and assessment of palmitic acid intake 220
5.3.5 Dietary palmitic acid intake categories 221
5.3.6 Statistical analysis of dietary data 221
5.3.7 Collection of Blood samples and RNA extraction 221
5.3.8 Gene expression profiling 222
5.4 Results 222
5.4.1 Participant characteristics 222
5.4.2 Palmitic acid content 224
5.4.3 Anthropometry 225

5.4.4 Dietary intake of nutrients 226
5.5.5 Gene expression 228
5.5.6 Comparison of gene expression profiles in low vs high palmitic acid intake 234
5.5.7 Comparison of gene expression profiles in low vs medium palmitic acid intake 234
5.5.8 Comparison of gene expression profiles in medium vs high palmitic acid intake 235
5.6 Discussion 236
5.7 Conclusions 245
Chapter 6. General Discussion 246
6.1 Research Overview 246
6.2 Synthesis of Findings 247
6.3 Original contributions to the research field 250
6.4 Future recommendations 251
References 252
Appendices 1
Appendix 1.1. Animal-based foods, plant-based foods and plant-based alternatives' fatty acid composition. 1
Appendix 2.1. Prisma 2020 for Abstracts Checklist. 2
Appendix 2.2. Prisma 2020 for Systematic Reviews Checklist. 4
Appendix 3.1. Participant information sheet for the survey and food diary data collection. 8
Appendix 3.2. Participant consent form for the survey and food diary data collection. 12
Appendix 3.3. Data protection checklist for the survey and intake study. 16
Appendix 3.4a. Recruitment poster for the survey and food diary data collection. 29
Appendix 3.4b. Recruitment social media post for the survey and food diary data collection. 30
Appendix 3.5. Participant information and palm oil awareness survey/questionnaire. 31
Appendix 3.6. Participant food/diet diary. 35
Appendix 3.7a. Respondent dietary intake (4-day mean) of calories and carbohydrates. 43

Appendix 3.7b. Respondent dietary intake (4-day mean) of fats. 46
Appendix 3.7c. Respondent dietary intake (4-day mean) of protein and vitamins A, D, E, K. 49
Appendix 3.8. Respondent dietary intake (mean) per dietary habit. 52
Appendix 4.1. Protocol for FAME preparation for gas chromatography analysis. 56
Appendix 4.2. Dry matter sampling protocol. 63
Appendix 4.3. Dry weight of food items within the dataset. 64
Appendix 4.4. Fatty acid composition of edible oils (dry matter content). 66
Appendix 4.5. Fatty acid composition of dairy-based foods (dry matter content). 68
Appendix 4.6. Fatty acid composition of nut/seed-based food items (dry matter content). 70
Appendix 4.7. Fatty acid composition of animal-origin foods (dry matter content). 72
Appendix 4.8a. Fatty acid composition of plant-based/vegan food alternatives (dry matter content). 74
Appendix 4.8b. Fatty acid composition of plant-based/vegan food alternatives (dry matter content). 76
Appendix 4.9. Fatty acid composition of miscellaneous/snack food items (dry matter content). 78
Appendix 4.10. Fatty acid composition of palm-based foods (dry matter content). 80
Appendix 4.11. Comparison of fresh weight (mg/g) of PA within food items between Chapter 4 and McCance and Widdowson's (CoFID) dataset. 82
Appendix 4.12a. Source information of failed food samples. 84
Appendix 4.12b. Sample preparation requirements of failed food samples. 84
Appendix 5.1. Participant information sheet for the biomarkers assessment. 85
Appendix 5.2. Participant consent form for the biomarkers assessment. 89
Appendix 5.3. Safety/COSHH form for the biomarkers assessment. 91
Appendix 5.4. Risk assessment form for the biomarkers assessment. 97
Appendix 5.5. Data protection checklist for the biomarkers assessment. 102
Appendix 5.6. Recruitment poster for the biomarkers assessment. 115

Acknowledgements

Firstly, I would like to thank the University of Central Lancashire, in particular the supervisory team of Dr Bojlul Bahar, Dr Jonathan Sinclair and Dr Stephanie Dillon for their invaluable support and direction. I would also like to extend my thanks to Dr Howard Hurst, Dr Robert Allan, Dr Belinda Hornby and Senior Laboratory Demonstrator Gareth Shadwell of UCLan, as well as Dr Marco Garcia-Vaquero and Dr Raquel Cama-Moncunill of University College Dublin (UCD), for their guidance and assistance throughout. Secondly, I am sincerely grateful to all participants for their contributions to the research. Lastly, I would like to give special thanks to my family for their patience, strength, and encouragement over the years. Thank you.

List of Figures

Figure 1.1. Overview of thesis chapters. 7
Figure 1.2. MetS-related deaths in years 2000-2019. 9
Figure 1.3. Incidences of global cardiovascular disease mortality. 9
Figure 1.4. Progression of insulin resistance to type 2 diabetes mellitus. 11
Figure 1.5. Mechanisms involved in high-fat diet-induced cardiovascular disease. 14
Figure 1.6. Pathophysiology of neurodegeneration. 18
Figure 1.7. Pro-inflammatory genes in acute and chronic inflammation. 19
Figure 1.8a. The NF-κB system and associated molecular and cellular signalling pathways. 22
Figure 1.8b. NF-κB targeted therapeutics in inflammatory disease. 22
Figure 1.9. The Nrf2 regulation pathway. 23
Figure 1.10. Homeostasis and imbalance outcomes within oxidative stress. 24
Figure 1.11. Post-prandial effects of carbohydrates on blood glucose. 25
Figure 1.12. Blood glucose homeostasis feedback within the liver. 26
Figure 1.13. Excess palmitic acid (palmitate) role in lipotoxicity. 28
Figure 1.14. Adverse effects of dietary imbalances (high-fat, high-sugar) on gut health. 30
Figure 1.15. Effects of plant-based versus animal-based fats on gut microbiota. 32
Figure 1.16. Chemical structure of example saturated and unsaturated fatty acids. 34
Figure 1.17. Timeline of important milestones in dietary fat evolution. 38
Figure 1.18. The range of yield (t/ha per year) of various edible oils. 40
Figure 1.19. The creation of palm oil derivatives for human use. 42
Figure 1.20. Content of palmitic acid (16:0) in common food groups consumed in the UK. 43
Figure 1.21a. Palmitate-mediated pathways in the immune response. 48
Figure 1.21b. Innate immune response amid palmitic-induced inflammation. 48
Figure 1.22. Sn positioning within palm oil triacylglycerols. 50
Figure 1.23. Changes in palmitic acid stereospecific numbering post-interesterification. 52
Figure 2.1. Included studies' various countries of origin, by number of studies. 62

Figure 2.2. The PRISMA flow diagram applied to extract literature on the effects of palm oil, palmitic acid and their derivates on metabolic health. 63
Figure 2.3. Risk of bias assessment of randomised human infant intervention trials (RoB2). 65
Figure 2.4a Risk of bias assessment of human adult intervention trials (RoB2). 66
Figure 2.4b Risk of bias assessment of human adult intervention trials (RoB2). 67
Figure 2.5. Risk of bias assessment of non-randomised human adult intervention trials (ROBINS-I). 68
Figure 2.6a. Risk of bias assessment of animal trials (SYRCLE). 70
Figure 2.6b. Risk of bias assessment of animal trials (SYRCLE). 70
Figure 2.7. Risk of bias assessment of in vitro trials (OHAT). 71
Figure 2.8. Total number of observed health outcomes of palm oil-related interventions. 72
Figure 3.1. Example food item entry within the Composition of Foods Integrated Dataset. 129
Figure 4.1. Esterification, saponification and extraction of FAMEs from food samples. 170
Figure 4.2. Example Chromatogram. 173
Figure 4.3. Formula used to calculate fatty acid content (mg/g). 174
Figure 4.4. The mean (mg/g, %) palmitic acid content per food group category. 207
Figure 4.5. Palmitic acid content of animal-origin foods and their plant-based alternative. 211
Figure 6.1. An overview of the main findings from each chapter within the thesis. 248
Figure 6.2. Reported health outcomes of palmitic acid (systematic review), alongside the general public's perceptions of palm oil/palmitic acid health outcomes. 249
Figure 6.3. Approximate dietary palmitic acid content (grams per day) of common UK dietary habits. 250

List of Tables

Table 1.1. Identifiable criteria of metabolic syndrome (MetS). 8
Table 1.2. BMI (kg/m^2) ranges in adults. 10
Table 1.3. Blood glucose levels threshold levels in diagnosing diabetes. 12
Table 1.4. Nutrients and their association with cancer development or prevention. 16
Table 1.5. Identifiable biomarkers of inflammation. 20
Table 1.6. Risk factors of acute and chronic microbial inflammation. 30
Table 1.7. Global percentage and figures share of palm oil production for 2023/24. 39
Table 1.8. Typical fatty acid composition of Malaysian palm oil. 44
Table 1.9. Palmitic acid content of common dietary fat sources. 44
Table 1.10. Full fatty acid composition of palm kernel oil. 45
Table 1.11. Stereospecificity of dominant triacylglycerol species in edible oils and natural fats. 50
Table 2.1. Domain(s) of biases evaluated within each risk assessment tool. 60
Table 2.2. Full terms for any abbreviations used within the characteristic's tables. 74
Table 2.3. Glossary of included interventions. 76
Table 2.4. Characteristics of studies investigating palm oil, palmitic acid, and their derivatives' health effects in human infants. 79
Table 2.5. Characteristics of studies investigating palm oil, palmitic acid, and their derivatives' health effects in human adults. 81
Table 2.6. Characteristics of studies investigating palm oil, palmitic acid, and their derivatives' health effects in animal trials. 96
Table 2.7. Characteristics of studies investigating palm oil, palmitic acid, and their derivatives' health effects in vitro. 106
Table 3.1. Fatty acid composition (% total) of common edible oils. 123
Table 3.2. Respondent selection(s) within baseline demographic/characteristics parameters. 131

Table 3.3. Respondent selection(s) within physical activity and sleep parameters. 132
Table 3.4. Respondent selection(s) within dietary habit parameters. 133
Table 3.5. Respondent selection(s) within awareness and preferences on palm oil parameters. 133
Table 3.6. Respondent selection of palm oil awareness group within demographic parameters. 135
Table 3.7. Respondent selection of palm oil awareness group within physical activity and sleep parameters. 136
Table 3.8. Respondent selection of palm oil awareness group within dietary parameters. 137
Table 3.9. Respondent selection of palm oil awareness group within palm oil awareness parameters. 138
Table 3.10. Respondent selection of palm oil awareness group within dietary parameters. 139
Table 3.11. Respondent palmitic acid daily intake per awareness group. 141
Table 3.12. Palmitic acid intake (grams per day) by dietary habit groups. 142
Table 3.13. Palmitic acid intake (grams per day) between two dietary groups (meat/dairy; non-meat/non-dairy). 142
Table 4.1. List of food items chosen within the selection phase. 155
Table 4.2a. List of sourced food items for analysis. 158
Table 4.2b. List of sourced food items for analysis (continued). 160
Table 4.3. Food sample weight requirements for analysis. 163
Table 4.4a. Food sample fat content, weight requirements and sample weights. 164
Table 4.4b. Food sample weight requirements and food sample weights (continued). 166
Table 4.5. List of reagents used within the preparation of FAMEs phase. 169
Table 4.6. FAMEs (38) detected during GC analysis. 172
Table 4.7. Fatty acid composition of edible oils. 177
Table 4.8. Fatty acid composition of dairy-based foods. 181
Table 4.9. Fatty acid composition of nut/seed-based food items. 185
Table 4.10. Fatty acid composition of animal-origin foods. 188
Table 4.11a. Fatty acid composition of plant-based/vegan food alternatives. 193

Table 4.11b. Fatty acid composition of plant-based/vegan food alternatives (continued). 195
Table 4.12. Fatty acid composition of miscellaneous/snack food items. 199
Table 4.13. Fatty acid composition of palm-based foods. 204
Table 5.1. Participant baseline demographic/characteristics parameters. 223
Table 5.2. Participant baseline demographic/characteristics per PA intake group. 223
Table 5.3. Participant palmitic acid intake (g) per dietary habit per PA intake group. 224
Table 5.4. Participant palmitic acid intake (g) per dietary habit. 224
Table 5.5. Participant bioimpedance analysis parameters per PA intake group. 225
Table 5.6. Participant dietary intake parameters per PA intake group. 227
Table 5.7. Gene identifiers (abbreviations) and their respective gene name. 229
Table 5.8. Differential expression of genes (by fold change) in cardiometabolic-related pathways between the three palmitic acid intake groups. 231
Table 5.9. The three foremost differentially expressed genes (by fold change) in the three palmitic acid intake groups. 243

Chapter 1. Introduction and Review of Literature

1.1 Background

1.1.1 Global Epidemic of Chronic Diseases

According to the World Health Organization (2023), chronic disease is responsible for 74% of annual global deaths. Oxidative stress and inflammation are key features of chronic health conditions, particularly those associated with metabolic syndrome (MetS) such as obesity, diabetes, cardiovascular disease, fatty liver disease, and cancer (Rochlani et al., 2017). A key feature of MetS is the disruption of the body's energy homeostasis, caused by the discrepancy between energy intake from food and energy expenditure. Weight gain and related health issues occur when there is an excess of energy, whereas a deficit of energy leads to weight loss. Long-term health and wellbeing of an individual are compromised by either extreme state.

Over the past few decades, there has been an exponential rise in the diseases of the MetS spectrum. Worldwide, there are 2.5 billion overweight adults and 890 million with obesity in 2022 (World Health Organization, 2022). It is reported that, as of 2022, the overweight and obesity population in the UK stands at 64% (NHS England, 2024). Projections indicate that obesity rates in the United States (Wang et al., 2008) and the United Kingdom (McPherson et al., 2007) will exceed 50% by 2030 and 2050, respectively. Numerous reports also predict that by 2050, MetS cases, including diabetes, cardiovascular disease, and nutrition-related cancers, will escalate to epidemic levels (Abdul et al., 2020, Jóźwiak et al., 2023; Li et al., 2021; Nedkoff et al., 2023).

1.1.2 COVID and Chronic Diseases

A contemporary health issue with universal impact; COVID-19, has strong links to MetS. Poor metabolic health: specifically, obesity, widely reported to impair immune function, decrease lung capacity, and increase the risk of viral infection, was positively associated with COVID-19 severity (Albashir, 2020). Unfortunately, the very nature of the pandemic itself created extremely obesogenic environments for millions of citizens worldwide. Lifestyle (lack of physical activity) and mental health status (Hossain et al., 2020), as well as diet quality (increased snacking, alcohol consumption) decreased exponentially (González-Monroy et al., 2021) due to the recommendations, or sometimes regulations, of simply staying indoors.

Patients with MetS reportedly had an increased mortality risk of 81%, due to COVID-19 (Wu et al., 2022). The long-term impact of COVID-19 and metabolic syndrome related disease cannot yet be fully understood. However, comprehensibly, proliferation of health risks throughout the duration of the pandemic (January 2020 - May 2023) acted synergistically with the COVID-19 virus to increase morbidity and mortality rate, creating an even more significant, and serious global epidemic. Many factors influence incidences of chronic disease; from demographic factors such as age, sex, and ethnicity (Kunnath et al., 2024), to socioeconomic status (Allen at al. 2017; Mielck et al., 2014). However, aside from smoking (Steinberg et al., 2010) and increased alcohol consumption (Shield et al., 2014), the principal behavioural or environmental components are lifestyle (physical activity) (Nyberg et al., 2020) and overall diet quality (nutrition) (Fanelli et al., 2020).

1.1.3 Role of Diet in Chronic Diseases

Convincing evidence suggests that unhealthy dietary habits have played a major role in the growing incidence of chronic diseases and nutritional transition in recent decades. In general, a westernised diet is highly processed and contains elevated levels of saturated fat, salt, and refined carbohydrates, while being low in fibre, healthy fats, micronutrients, and phytochemicals (Fedacko et al., 2022). Consuming processed foods like meat and dairy, red meat, and refined grains such as white bread and white rice, while having low intakes of fruits, vegetables, whole grains, nuts, and seeds is highly prevalent in this dietary habit. Evidence suggests that foods containing high saturated fat increases the risks of obesity (Golay & Bobbioni, 1997), diabetes (Brøns et al., 2009), cardiovascular disease (Guasch-Ferré et al., 2015) and many nutrition-related cancers including bowel, lung, prostate, breast, and uterine cancers (Hayashi et al., 2018). High saturated fat intake increases the build-up of blood cholesterol (Hart et al., 2025; Siri-Tarino et al., 2010; Sun et al., 2015), particularly low-density lipoprotein cholesterol (LDL-C). Contemporary studies have linked LDL-C to the development of cardiovascular disease (Boren, 2020). Foods high in salt increase blood pressure by promoting the entry of excess water to the bloodstream (Juul et al., 2021, Mohan & Campbell et al., 2009).

Consuming whole grains and dietary fibre can reduce the risks of MetS by slowing down digestion and suppressing appetite (Ye et al., 2015). According to the International Agency for Research on Cancer (IARC), processed meat is carcinogenic, while red meat is likely carcinogenic (World Health Organization, 2015). To enhance flavour and preserve meat,

processing involves techniques such as curing, salting, or smoking, and the addition of nitrates and nitrites. The outcome of these processes is the creation of two metabolites, N-nitroso-compounds (NOC) and polycyclic aromatic hydrocarbons (PAH), both of which are highly carcinogenic (Rohrmann & Linseisen, 2016). Beef, pork, and lamb, as well as fish and poultry, contain heme iron that can also promote the development of carcinogenic compounds (Bastide et al., 2011).

1.1.4 Plant-based Diet/Veganism

With the awareness of the susceptibility of a highly processed westernised diet, there has been a transformation in the Western dietary pattern. From 2016 onwards, there was a remarkable increase in the popularity of the plant-based diet (PBD) (Grassian, 2020). In a plant-based diet, there is an emphasis on eating more vegetables, fruits, whole grains, legumes, nuts, and seeds, in place of animal-based foods such as meat or dairy (Richter et al., 2016). Although the term 'plant-based' is often used within the literature to describe diets containing reduced amounts of animal-origin food products - for the purposes of this thesis, the term 'plant-based diet' will be used hereafter to define the diet adopted by those who practice 'veganism', i.e., a diet containing strictly no animal-based foods (meat and/or dairy). Increasing evidence shows the health advantages of a plant-based or vegan diet. These health benefits are typically due to consuming more dietary fibre, antioxidants, and phytochemicals while consuming less unhealthy fats, cholesterol, and animal-derived proteins (Szabo et al., 2021). Some of the health benefits of a plant-based/vegan diet include weight reduction (Turner-McGrievy et al., 2017), optimal blood pressure (Tuso et al., 2013), healthy blood cholesterol profile (Yokoyama et al., 2017), lowering insulin resistance (Kuchta et al., 2016), reducing cardiovascular disease pathophysiology (Kim et al., 2019), and potential anti-ageing and anti-cancer effects (Bahar et al., in press).

Because of the increasing popularity of plant-based/vegan diets and recognising the market potential, food industries are introducing more plant-based/vegan friendly products. Although seen as a healthier option compared to the Western diet, a plant-based diet also includes refined or processed foods. The popularity of plant-based alternatives or imitation versions of animal-based products, such as 'meat-free' burgers, 'vegan' chocolate, or 'free-from' cheese, is growing (Zhao et al., 2023). Industrial processing of plant-based food products removes essential nutrients like protein, fibre, vitamins, and minerals to increase production speed and

reduce costs (Prescott et al., 2023). It is common for industrially processed vegan food to include saturated fats, salts, sugars, emulsifiers, artificial colours, and additives in order to enhance appearance, texture, mouthfeel, and flavour (Bogueva & McClements, 2023). These plant-based alternatives or analogues are highly processed or 'ultra-processed' foods, which share similar nutritional properties to non-plant-based processed foods. Chicken-free nuggets, ham-free slices, and dairy-free butter are some examples of ultra-processed plant-based food items.

Some vital micronutrients, such as vitamin D, vitamin B-12 (typically found in animal sources), calcium, iron, zinc, and long-chain omega-3 fatty acids, are often low in a plant-based/vegan diet (Craig, 2009). Fortified breakfast cereals and snack bars are considered highly processed foods, but they can also provide many of these micronutrients as supplements. Processed plant-based meat and dairy alternatives have less saturated fat and more fibre compared to animal-based counterparts, despite being relatively high in fat and saturated fat (Clegg et al., 2021; Nájera Espinosa et al., 2024). Processed plant-based alternatives like tofu, tempeh, and seitan can also provide essential nutrients and have a positive effect on metabolic health (Gastaldello et al., 2022). Therefore, it is important to evaluate the nutritional health implications critically when distinguishing between minimally processed and ultra-processed plant-based/vegan foods (Gibney et al., 2017; Lee at al., 2020; Messina et al., 2022).

1.2 Lipids in food

1.2.1 Dietary Lipids

Dietary lipids play a crucial role in the human body by providing calories, fatty acids, and transporting fat-soluble vitamins. About 15 kg of fat is found in a healthy human body (a 70 kg healthy male body with 20% body fat). It is recommended to consume 15-35% of daily calories from fats (World Health Organization, 2023). The main sources of dietary lipids are plant-derived lipids, animal-derived lipids, and to some extent industrially processed fats (hydrogenated oil/trans-fats). Animal sources such as meat and dairy contribute 40-60% of dietary fats in Western diets (Sanders, 2024). The nutritional quality of dietary lipids is influenced by the different proportions of monounsaturated fatty acids (MUFAs), polyunsaturated fatty acids (PUFAs), and saturated fatty acids (SFAs). Monounsaturated fatty acids (MUFAs) are commonly found in plant lipids, including olive oil, canola oil, sunflower

oil, pumpkin seeds, sesame seeds, avocados, almonds, cashews, and nut butters (Mattson, 1989). PUFAs are commonly present in plant and animal lipids such as fish (anchovies, herring, mackerel, salmon, sardines), nuts (walnuts), seeds (flaxseeds, sunflower), tofu, and soybeans (Duan et al., 2023). Saturated fatty acids (SFAs) are primarily present in animal-derived lipids like meats (beef, lamb, pork), poultry, lard, and dairy products (butter, whole milk, cream) (Perna & Hewlings, 2022). Tropical oils, such as coconut oil, palm oil, and palm kernel oil, are plant-based sources that are particularly rich in SFAs (Unhapipatpong et al., 2021). Palmitic acid, abundant in animal-based products like meat and dairy, is also found in plant-based sources like palm oil.

1.2.2 Oil/Lipids in industrial food processing

Fat plays an important role in the sensory properties, nutritional profile, shelf-life, and stability of food products during processing (Aceves-Martins et al., 2022). Dietary lipids serve multiple purposes, including enhancing flavour, texture, emulsification, and preservation. An important way that lipids contribute to food preservation is by creating a barrier against moisture and oxygen, which stops oxidation and rancidity. The double bonds in unsaturated fatty acids can react with oxygen, causing oxidative rancidity, the creation of peroxides and aldehydes, and a foul smell. Edible oils (like soybean oil) and animal fats (such as lard and tallow) are the most commonly used fats in the food industry. Hydrogenated, and partially hydrogenated vegetable oils, known as trans fatty acids, are altered fats that are used in the processed food industry for their longer shelf-life. Trans fats are commonly present in snack foods like cakes, cookies, and popcorn, as well as in fried foods such as fries, doughnuts, and fried chicken, and in margarine or shortening (Dhaka et al., 2011). The recommended maximum daily trans-fat consumption for adults, as per the UK government, is around 5g per day (NHS, 2023) while the limit is much lower (2 gm) by the EU countries.

Palm oil gained popularity in the processed food industry as a replacement for trans fats and hydrogenated vegetable oils (Kanagaratnam et al., 1995), as well as advancements in production processes in the early 1990s (Gordon, 1993). It offers various benefits, such as improving texture, stability, and shelf-life of foods, as well as serving as a suitable cooking oil for frying. Primarily, palm oil is known for its ability to resist oxidation (Sulaiman et al., 2022). Animal fats were previously the dominant dietary fat source (Huebbe & Rimbach, 2020). Although, vegetable oils, especially a versatile plant-based, trans-fatty acid free, saturated fat

oil, was needed for vegetarian/vegan foods, making palm oil the ideal choice. Palm oil cultivation and its impact on the climate and environment has sparked intense debate in recent decades (Teng et al., 2020), potentially causing consumers to avoid palm oil-containing products because of ethical and environmental concerns. While the nutritional implications of consuming palm oil or foods containing it have been challenged, its conclusive effects on human health are unclear. Given the high levels of palmitic acid in palm oil, it is reasonable to assume that consuming palm oil-based food may have a detrimental impact on metabolic health. Hence, the overall goal of this study was to gather evidence on the food containing palm oil/palmitic acid and relationship between consuming palm oil and the development of chronic diseases. This thesis has the following chapters:

Chapter 2

Effects of palm oil, palmitic acid, and their derivatives on metabolic health: a systematic review

To collect, collate and analyse the relevant data to denote a conclusive answer on the effects of palm oil/palmitic acid on various metabolic health disorders, chronic diseases and relevant co-morbidities.

Chapter 3

Awareness and perceptions of the health effects of palm oil and palmitic acid

To investigate the United Kingdom general publics' awareness, perceptions and beliefs of palm oil/palmitic acid – cross-referencing with participant background, dietary habit and lifestyle factors through questionnaire. Dietary analysis to determine daily palmitic acid consumption of typical dietary habits.

Chapter 4

Fatty acid composition of common foods consumed in Westernised diets

To analyse the fatty acid composition of commonly consumed food items within Westernised diets, with a focus on palmitic acid. Also, to determine palm oil/palmitic acid content within vegan/plant-based foods and meat/dairy alternatives (deficient in current available data).

Chapter 5

An assessment of gene expression biomarkers linked to metabolic health in participants with different levels of dietary palmitic acid consumption

To directly analyse the effects of different levels of daily palm oil/palmitic acid consumption on an assembly of metabolic health biomarkers, highlighting any potential optimal/sub-optimal range(s) - through peripheral blood analysis.

The overview of all thesis chapters, including Chapter 1: Introduction and Review of Literature, and Chapter 6: General Discussion and Conclusions, is presented in Figure 1.1.

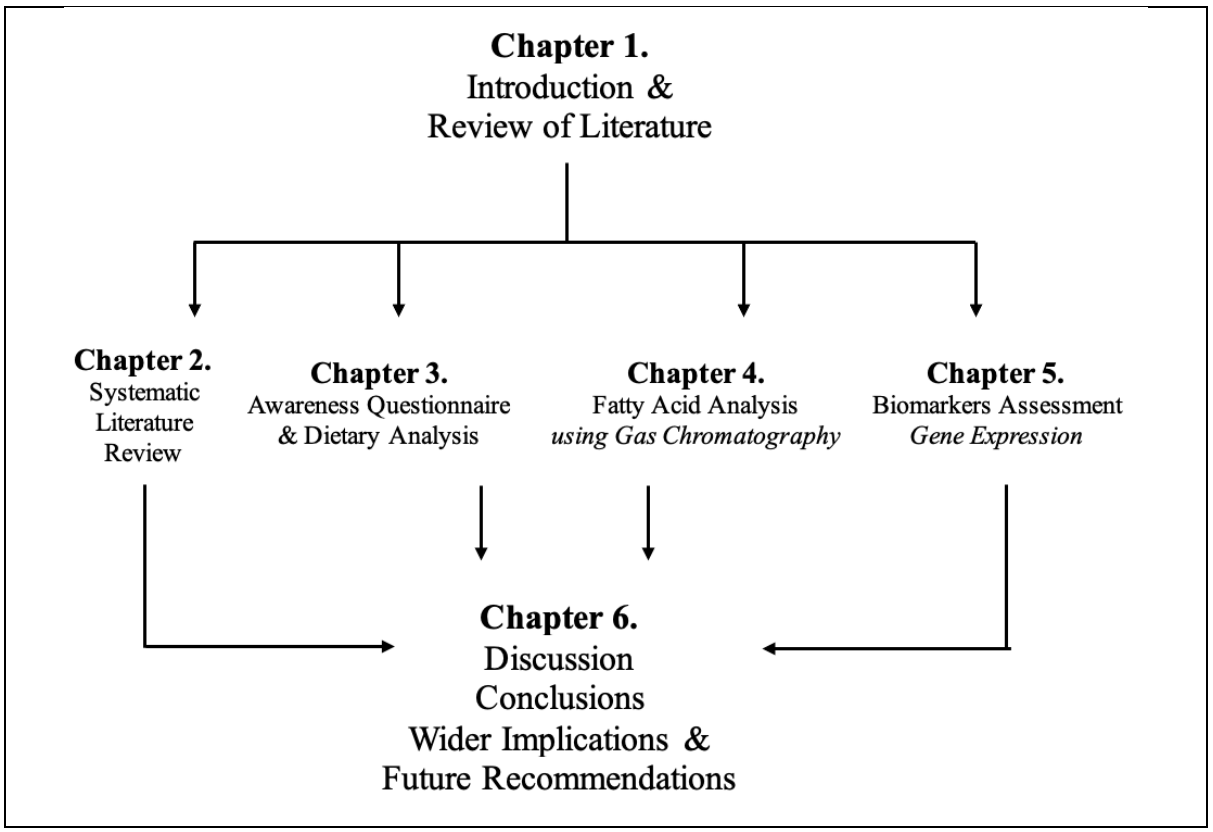


Figure 1.1. Overview of thesis chapters.

Review of Literature

1.3 Chronic diseases

1.3.1 Metabolic syndrome

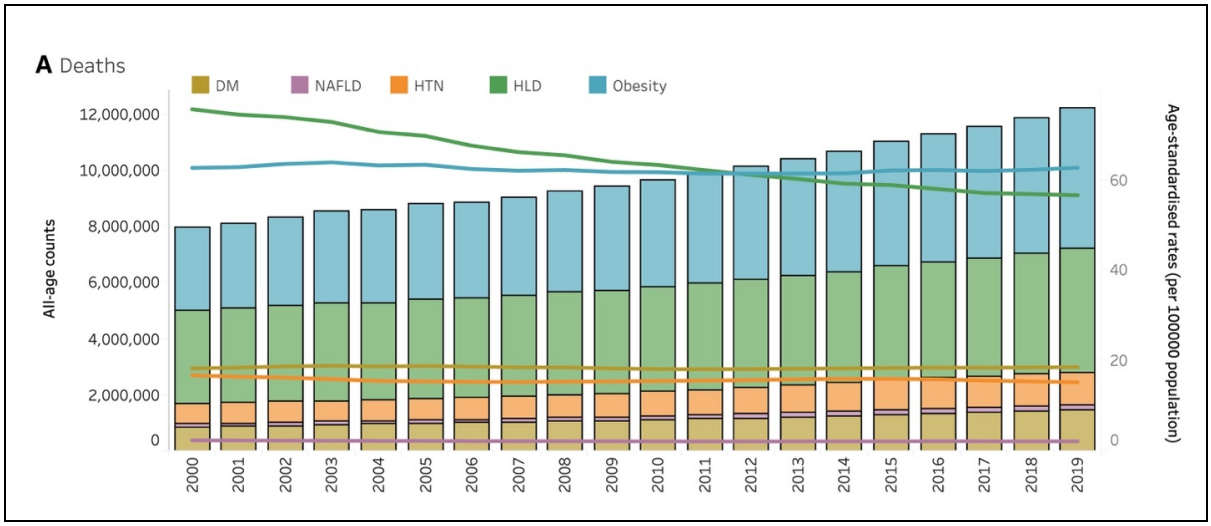
Metabolic syndrome (MetS) is a cluster of adverse health conditions, occurring simultaneously and increasing the risk of cardiovascular atherosclerotic disease and diabetes mellitus type 2 (Kassi et al., 2011). Markers of MetS include various risk factors including excess adiposity (central obesity), high blood sugar, impaired fasting glucose or insulin resistance (pre-diabetes), elevated blood pressure (hypertension), inflammation, high serum triglyceride levels, as well as abnormal levels of cholesterol (dyslipidaemia) - precisely: low levels of high-density lipoprotein cholesterol (HDL-C) (Cassells & Haffner, 2006). Diagnoses necessitate precise levels of these components (Table 1.1).

Table 1.1. Identifiable criteria of metabolic syndrome (MetS).
Note: Adapted from Ginsberg & MacCallum, 2009.

Metabolic Syndrome (MetS)	
Characterised by (at least) three of the following*:	
Central Obesity	Waist circumference: ≥88 cm/35 in (women) ≥102 cm/40 in (men)
Blood Pressure	≥130/80 mmHg
Insulin (abnormalities)	Fasting plasma glucose: ≥5.6 mmol/L
Triglycerides	≥1.7 mmol/L
Cholesterol	HDL: <1.3 mmol/L (women) <1.0 mmol/L (men)
**Must either meet the indicated criteria OR be on medication for a risk factor.*	

mmHg = millimetres of mercury. mmol/L = millimoles per litre.

Over the last twenty years, there has been a clear epidemic growth curve; with deaths progressively increasing (globally) year-by-year for MetS-related diseases, chiefly obesity, and type 2 diabetes mellitus (Figure 1.2), as well as cardiovascular disease (Figure 1.3).



DM = diabetes mellitus, NAFLD = non-alcoholic fatty liver disease, HTN = hypertension, HLD = hyperlipidaemia.

Figure 1.2. MetS-related deaths in years 2000-2019.

Note: From Chew et al., 2023.

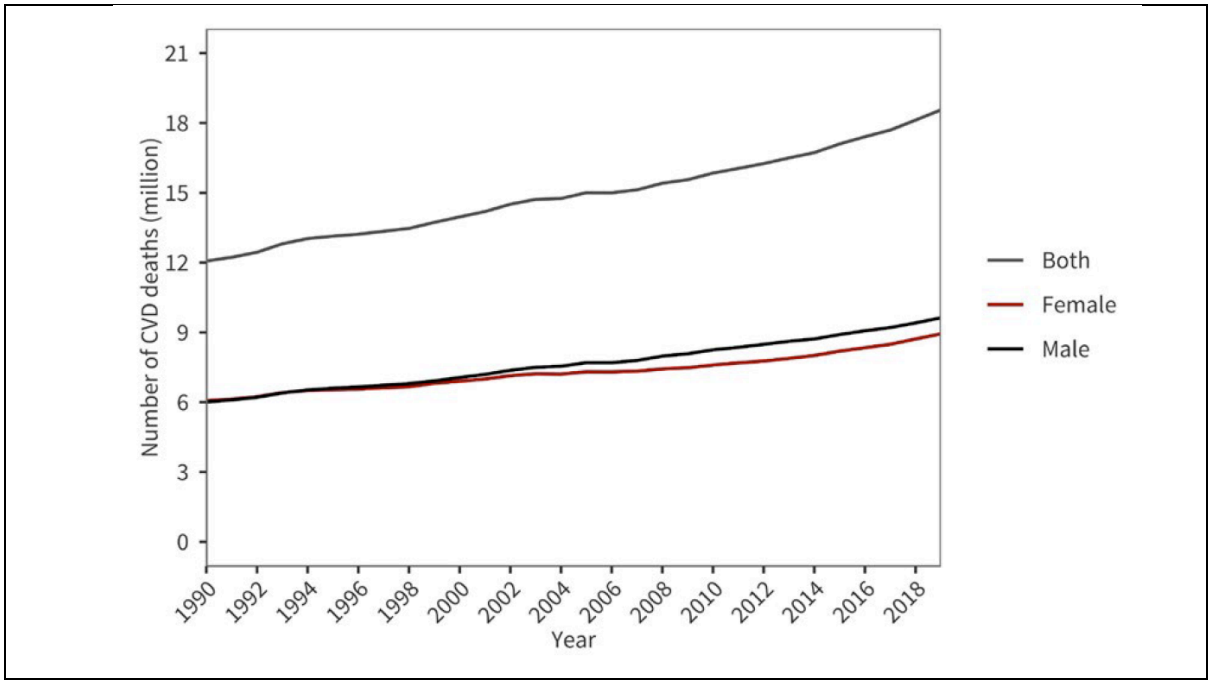


Figure 1.3. Incidences of global cardiovascular disease mortality.

Note: From World Heart Federation, 2023.

The following section(s) investigate major diseases within the MetS spectrum.

1.3.2 Overweight and obesity

Overweight and obesity are conditions characterized by excessive accumulation of body fat, to the point of developing health risks (Apovian, 2016). Consumption of excessive calories (hyperalimentation) and physical inactivity that results in a positive energy balance is the principal cause of overweight and obesity (Stein & Colditz, 2004). There are two types of body fat which contribute to obesity. These are visceral adipose tissue (VAT) that surrounds vital organs in the abdominal area such as the stomach, liver and intestines, and subcutaneous adipose tissue (SAT) which is fat stored underneath the skin. The VAT is associated with health risks (Tchernof & Després, 2013). Excessive accumulation of body fat is also associated with high blood pressure, high cholesterol, insulin resistance, hyperinsulinemia, dyslipidaemia, and other conditions, linking the diseases within the MetS spectrum to a common aetiology of positive energy balance. An individual is categorised as overweight or obese if their weight and height enter a specific range. Calculated using a body mass index (BMI), with the formula of weight divided by height squared. Overweight is determined by the weight/height range of 25-29.9 kilograms per square metre (kg/m^2), whereas obesity is defined as 30 kg/m^2 or higher (Table 1.2).

Table 1.2. BMI (kg/m^2) ranges in adults.
Note: Adapted from Jensen et al., 2014.

BMI in Adults ≥18 years	
BMI (kg/m^2)	**Weight Status**
<18.5	Underweight
18.5 - 24.9	Normal (Healthy) Weight
25 - 29.9	Overweight
>30	Obese

In 2022, 2.5 billion adults (aged 18 years and above) were classed as overweight. Of those, 890 million were classed as obese. These numbers have risen sharply from 2016, where the reported figures were 1.9 billion and 650 million respectively (Haththotuwa et al., 2020). Central obesity is a fundamental component of MetS, and arguably its' pathophysiology is the precursor to all other risk factors linked to related MetS, such as hypertension, insulin resistance and T2DM.

It has been proposed that there are two main lifestyle factors linked to obesity. These are consumption of highly processed food (containing high levels of dietary fat) and a sedentary lifestyle (Pagliai et al., 2021). The Western, high-fat diet pre-disposes the risk of developing overweight, obesity and associated co-morbidities.

1.3.3 Type-2 diabetes

Diabetes is a chronic disease, occurring either when the body cannot produce enough, or effectively use insulin, leading to elevated blood glucose levels (hyperglycaemia) (Fujimoto, 2000). Type-2 diabetes mellitus, described as elevated levels of blood glucose, dysregulation in the metabolism of lipids, proteins and fats, and insulin resistance (Egan & Dinneen, 2014), is estimated to be >90% of all cases of diabetes (Chatterjee et al., 2017). Insulin resistance (IR) is a condition where there is a sufficient insulin response to the elevated blood glucose levels, however, the cells are less responsive to the action of insulin (Wilcox, 2005). Insulin resistance within skeletal muscle, liver and adipose tissue, alongside pancreatic β-cell dysfunction, are the underlying causes of type-2 diabetes (Figure 1.4). β-cell dysfunction, or impaired pancreatic insulin secretion, occurs when β-cells are unable to produce adequate quantities of insulin (LeRoith, 2002).

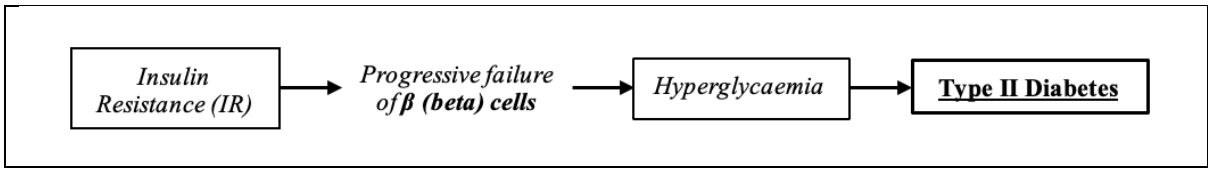


Figure 1.4. Progression of insulin resistance to type 2 diabetes mellitus.
Note: Adapted from Dunmore & Brown, 2013.

Diagnoses of diabetes impose specific ranges of blood glucose to determine classification, these threshold levels are typically measured in either millimoles per litre (mmol/L) (UK), or milligrams per decilitre (mg/dL) (US). The haemoglobin A1c (HbA1c) test is the most common clinical assessment of type 2 diabetes, measuring blood sugar attached to the body's haemoglobin, a protein located within red blood cells, tasked with oxygen transportation (Table 1.3).

Table 1.3. Blood glucose levels threshold levels in diagnosing diabetes.

Note: Adapted from World Health Organization, 2006.

Type 2 Diabetes Mellitus		
	Pre-prandial (fasting)	**Post-prandial**
Non-diabetic	5.6 mmol/L (<100 mg/dL)	7.8 mmol/L (<140 mg/dL)
Pre-diabetes	5.6 - 6.9 mmol/L (100 - 125 mg/dL)	7.8 - 11 mmol/L (140 - 199 mg/dL)
Diabetes	7.0 mmol/L (≥126 mg/dL)	11.1 mmol/L (≥200 mg/dL)

Impaired fasting glycaemia (IFG), oft-referred to as pre-diabetes, is raised blood sugar glucose levels within the body - however, as these levels are below the range(s) of diabetes, the term pre-diabetes is used to describe the body's glucose utilisation in an impaired state. Pre-diabetes is reported to affect approximately 5.1 million (~12% of) adults within the UK (Office for National Statistics, 2024). Globally, an estimated 462 million persons have type 2 diabetes, (Khan et al., 2020). There are reportedly more than 38 million people in the United States (Centers for Disease Control and Prevention, 2024), and more than five million people in the UK living with diabetes (Diabetes UK, 2024).

Increased adipose tissue mass plays a large role in insulin resistance, through elevated free fatty acid (FFA) levels, plasma leptin and tumour necrosis factor-α (TNF-α) (Leon & Wilding, 1999). Other factors can also contribute to β-cell failure; including ageing (Zhu et al., 2021), genetics (Asahara et al., 2022), glucagon-like peptide 1 (GLP-1) and gastric inhibitory polypeptide (GIP) resistance, or deficiency (Shu et al., 2009), lipotoxicity (Vilas-Boas et al., 2021), oxidative stress (Eguchi et al., 2021), inflammation and activation of inflammatory pathways (cytokine production) (Dludla et al., 2023), FFA abundance (Lytrivi et al., 2020), endoplasmic reticulum stress signaling (Shrestha et al., 2021). Prolonged exposure to inflammatory mediators interferes with and inhibits insulin signalling receptors in β-cells of both pancreatic islets and peripheral tissues, through the activation of signalling proteins (cytokines) (Berchtold et al., 2016).

Increased oxidative stress in peripheral tissues plays a key role in the pathophysiology of insulin resistance. Free radicals (including reactive oxygen species and reactive nitrogen species), physiologically generated from lipid oxidation are oxygen biomolecules that have roles in many pathways, including cellular signalling. Increased free radical generation disables or overwhelms the defence mechanisms of cells, leading to amplified oxidative stress. β-cell

function is impaired via increased oxidative stress in various ways, primarily reduced insulin production (Asmat et al., 2016).

1.3.4 Cardiovascular disease

Cardiovascular disease (CVD), a general term for conditions affecting the heart or blood vessels, has persisted as the leading cause of death worldwide for multiple decades (Mathers et al., 2005). There are various types of cardiovascular diseases: coronary heart disease (CHD), heart attack or heart failure; stroke or transient ischaemic attack (TIA); peripheral arterial disease; aortic disease - all involving the restriction or limitation of blood flow, through build-up of fatty deposits inside the arteries (atherosclerosis), to parts of the body including the brain, heart and limbs, and an increased risk of blood clots (Sara, 2018). Cardiovascular disease is also closely linked with thrombosis, an intravascular disorder (venous: veins, arterial: arteries) which occurs when blood vessels become blocked due to fatty deposits from foods (Zhang et al., 2024).

The modifiable risk factors of CVD include smoking/alcohol intake, stress, physical inactivity, and obesity (Nangia et al., 2016). A key risk factor in the development of CVD is blood pressure (BP), more specifically high blood pressure (HBP) (Fuchs & Whelton, 2020). BP is the force that the heart pumps blood around the body (Flint et al., 2018); measured using two values of pressure, systolic: BP in arteries when the heart beats, and diastolic: BP in-between beats (Magder, 2018). Hypotension, or low blood pressure, typically does not cause many health issues (Wessely et al., 1990), however hypertension (both stage 1 and stage 2), or high blood pressure, can lead to many cardiovascular diseases such as stroke and heart attack (Gosmanova et al., 2016). A normal or healthy blood pressure is typically <120/80mm Hg. In the UK, around 1 in 3 adults have high blood pressure (UK Data Service, 2015).

Dietary lipids play a role in the pathogenesis of CVD (Figure 1.5). Established risk factors of cardiovascular disease include increased adiposity, elevated apolipoprotein B-rich lipoproteins, hyperglycaemia, as well as hypertension - all synonymous with the effects of a high fat diet, particularly saturated and trans fats (Guasch-Ferré et al., 2015). Lipid accumulation exacerbates atherosclerosis progression within the blood vessels, leading to blood clots and an increased risk of heart attack (Malekmohammad et al., 2021).

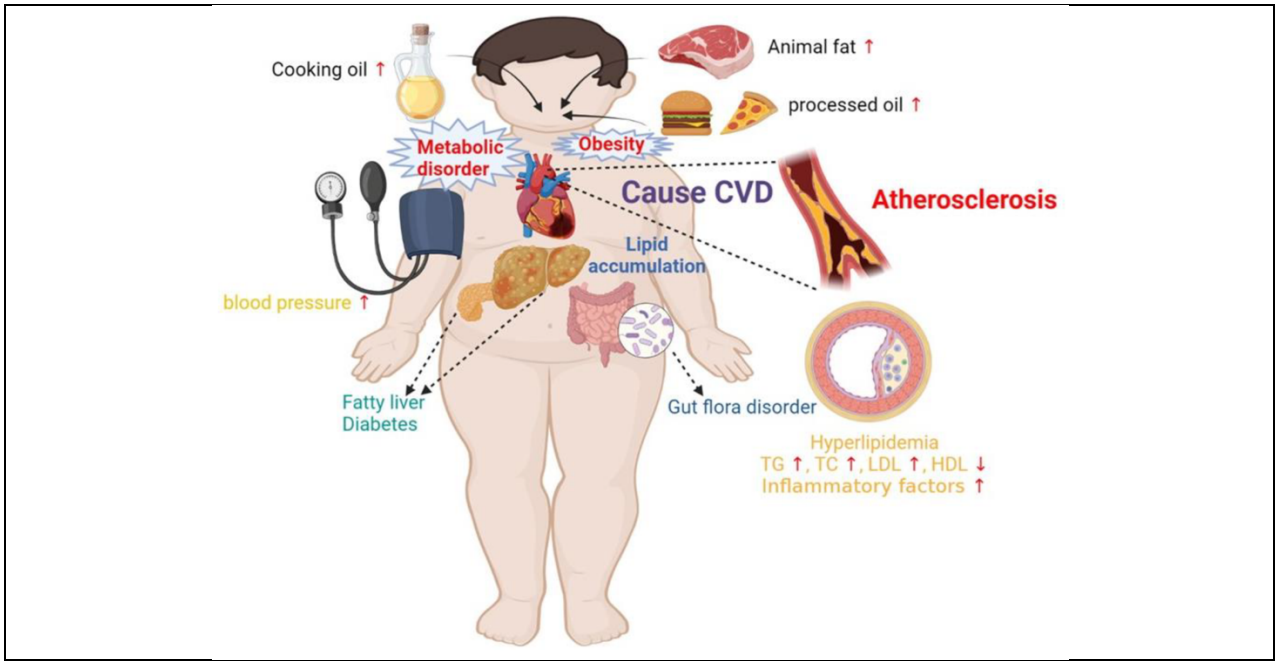


Figure 1.5. Mechanisms involved in high-fat diet-induced cardiovascular disease.

Note: From Weng et al., 2023.

1.3.5 Metabolic dysfunction-associated steatotic liver disease (MASLD)

Metabolic dysfunction-associated steatotic liver disease (MASLD), also known as non-alcoholic fatty liver disease (NAFLD) (Rinella et al., 2023), characteristically, develops in the liver of persons, though not alcohol-induced (Younossi et al., 2016). MASLD is defined as the accumulation of lipids in the liver, predominantly in the form of body fat constituents named triglycerides (TAGs). Dietary, circulating, and DNL-generated free fatty acids (FFA) esterify with glycerol, forming these TAGs. Metabolic syndrome (MetS) risk factors, specifically obesity and insulin resistance are commonly associated with MASLD (Sakurai et al., 2021). However, in some Asian countries (with lower BMI and seemingly much lesser incidences of insulin resistance), the disease can also manifest (Loomba & Sanyal, 2013). MASLD is also associated with major pathogenetic risk factors such as hepatic and systemic inflammation, lipotoxicity, as well as increased serum triglycerides and LDL-C, causing dyslipidaemia/hyperlipidaemia (Byrne & Targher, 2015).

Patients with MASLD have increased risks of cardiovascular mortality, with the majority of deaths occurring through CVD. Excess calories cause fat build up within the liver, resulting in the failure to process and break down fats. High-fat diets are positively correlated with obesity, highly prevalent in most cases of MASLD (Lian et al., 2020). High fat diets are also often used

in epidemiological studies to induce hepatic steatosis within animal models, outcomes closely resembling MASLD in humans (Im et al., 2021). The Western diet, containing typically high-fat (saturated) and carbohydrate-rich (fructose and sucrose) food sources, has been associated with many factors of MASLD (Figure 15). Although progress has been made, pathophysiology and pathogenesis of MASLD are not fully understood (Mantovani & Dalbeni, 2022), with sources post-2020 reporting that further research is essential (Lee et al., 2024). MASLD has grown to worldwide epidemic status in recent years, alongside obesity and type-2 diabetes mellitus (Estes, et al., 2018). MASLD is believed to affect approximately 25% of the global population; highest in the Middle East (32%) and lowest in Africa (13%), with rates at 23% in Europe (Maurice & Manousou, 2018) and approximately 32% in the US (Younossi et al., 2020).

1.3.6 Cancer

Cancer is a chronic disease which can affect most organs within the human body, occurring when mutations in cancer-susceptible genes lead to cells beginning to grow, act and spread, uncontrollably (Michor et al., 2004). There are many different types of cancer, with the most prevalent, and cause of most deaths, being lung, breast, prostate, and colorectal cancers (Mattiuzzi & Lippi, 2019). These cancers, principally colorectal, are the cancers most commonly associated with nutrition. 'High-fat' diets are typically defined as >35% (fat) of total energy intake (Office for Health Improvement and Disparities, 2025). However, the percentile range of a diet described as 'high-fat' can vary considerably - for example, a low-carbohydrate diet such as the ketogenic diet can consist of up to ~75% (fat) of total energy intake (Chung, 2023). A diet this high in fat (ketogenic) can influence various beneficial, as well as adverse, physiological and biochemical effects on metabolism, brain function and gut microbiota, due to adaptations within the body of now relying on fat as the primary energy source, in place of carbohydrates (glucose). Although, a ketogenic diet only accounts for ~2% of United Kingdom diets (Euromonitor International, 2024), with contemporary research indicating consumer dietary fat intake (on average) to be approximately 35% (Office for Health Improvement and Disparities, 2025). Microbial dysbiosis (an imbalance within the gut microbiome) contributes to colorectal tumour growth through the distribution of oncometabolites (Kato & Sun, 2023; Singh et al., 2023). It has been estimated that 15-20% of all cancers can be prevented by lifestyle and dietary measures alone (Stern et al., 2020). Many different types of foods, explicitly their unique nutrient structure or micronutrient composition, are believed to aid in the development or prevention of certain cancers, with negative effects

chiefly being attributed to high intakes of omega-6 (n-6/ω-6) fatty acids, or perhaps an imbalance between ω-3 and ω-6 fatty acids, as well as saturated fatty acids (Table 1.4).

Table 1.4. Nutrients and their association with cancer development or prevention.

Note: Adapted from Valdés-Ramos & Benítez-Arciniega, 2007.

Nutrient	Type of cancer
[1]ω-3 fatty acids	↓ Breast, colon, rectum, prostate
[1]ω-6 fatty acids	↑ Breast, prostate
Saturated fatty acids	↑ Colon, rectum, breast, prostate
Vitamin A	↓ Precancerous lesions
Vitamin C	↓ Oral cavity, oesophagus, stomach, colon
β-Carotene	↓ Lung, stomach, breast, colon, prostate
Selenium	↓ Lung, colon, rectum, prostate

[1]ω = omega.

These fatty acids are highly prevalent in high-fat, low-fibre diets (such as the Western diet). Dietary fat is associated with cancer development, whereas fibre is associated with cancer prevention (Faivre & Giacosa, 1998; McIntosh, 2001). Trans-fatty acids increase the risk of prostate and colorectal cancers (Michels et al., 2021). Palmitic acid, an incredibly prevalent saturated fatty acid found in most modern dietary habits, has been shown to promote colorectal cancer, through activation of cytokines colony stimulating factor 1 (CSF-1), transforming growth factor-β1 (TGF-β1), and interleukin-8 (IL-8/CXCL8) signalling (Deng et al., 2025). Increased dietary sugar intake has been positively associated with breast and prostate cancers (Debras et al., 2020). There is a strong correlation between the consumption of red meat such as beef, lamb and pork, and cancers such as breast and gastric (Sivasubramanian et al., 2023). Processed meats are also described as a significant risk factor in colorectal cancer development (Oostindjer et al., 2014). The presence of certain compounds, including heme iron, in red meat has been found to promote tumorigenesis (Bastide et al., 2011). Other carcinogens (cancer-causing substances) are located within animal-origin foods: heterocyclic amines and polycyclic aromatic hydrocarbons (smoked meats, high temperature grilled meats), as well as nitrites and nitrates (processed meats) (Soliman, 2018). High intakes of dairy products, specifically bovine (cow's) milk (and subsequently all dairy products containing it), have been linked to the increased risk of prostate cancer (Zhao et al., 2023).

Poor quality diet, alongside high energy intake is among the main risk factor(s) for cancer; significant positive associations were found between overweight, obesity and higher death rates for the following cancers: oesophagus, colon, liver, pancreas, kidney, gastric, prostate, breast, uterus, cervix, and ovary (Calle et al., 2003). There are also many other modifiable risk factors for cancer, including living a sedentary lifestyle, as well as alcohol and smoking intake (Stein & Colditz, 2004). Metabolic factors, or diseases associated with MetS, are largely believed to exacerbate the risks of mortality of cancer. Overweight and obesity is the second biggest cause of cancer in the UK, behind smoking (Brown et al., 2018). Diabetes is a risk factors for many cancers, including pancreatic, liver, colon, and breast cancer (Shahid et al., 2021). Regulation of gut microbiota through increased fibre, polyunsaturated fat, polyphenols, and probiotic consumption, is believed to be a therapeutic mechanism in cancer prevention (Rebersek et al., 2021).

1.3.7 Neurodegenerative disease

Cerebrovascular health, or cerebrovascular disease (CeVD), relates to disorders affecting the blood vessels of the brain (Portegies et al., 2016). Neurogenerative disease, an umbrella term for these disorders, is characterised by the progressive loss of, or damage to, brain neuron activity; aptly named neurodegeneration (Lamptey et al., 2022). As neuronal function continues to deteriorate, cognitive impairment occurs, ultimately resulting in induced apoptosis (cell death); commonly leading to neurodegenerative disorders such as Alzheimer's, Parkinson's, and Huntington's diseases, as well as multiple sclerosis (MS) and amyotrophic lateral sclerosis (ALS) (Gowthaman et al., 2007). Approximately 15% of the worldwide population has a neurodegenerative disease (Van Schependom et al., 2023). Neurodegeneration typically stems from damage to cells through oxidative stress, neuroinflammation or the most important intrinsic risk factor; age (Hou et al., 2019). Many factors can influence neurodegeneration (Figure 1.6).

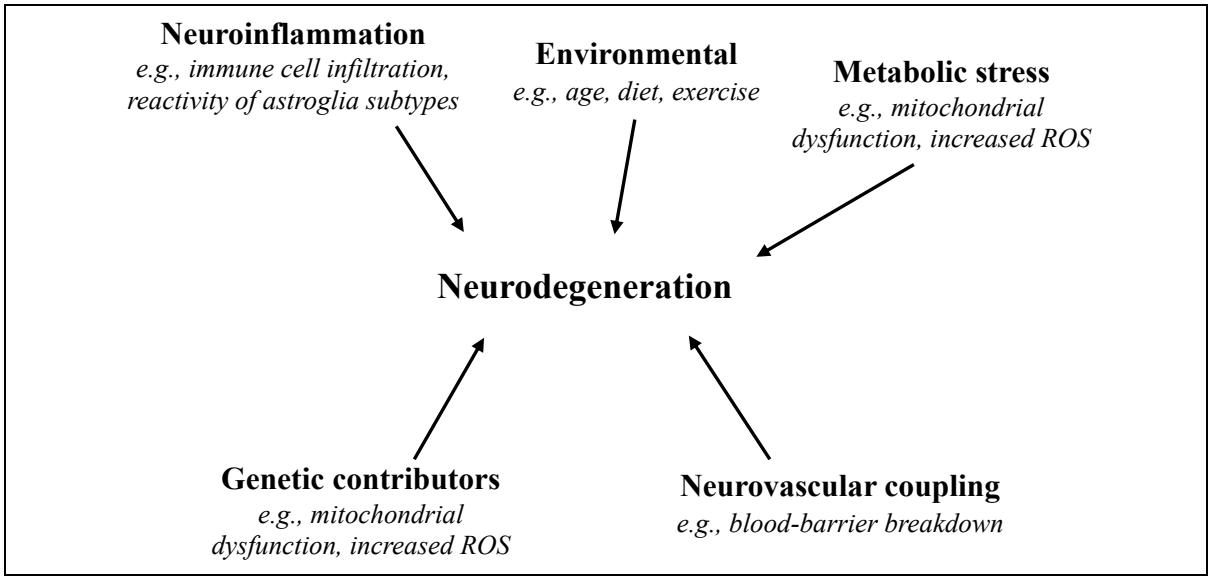


Figure 1.6. Pathophysiology of neurodegeneration.

Note: Adapted from Wareham et al., 2022.

Nutrition plays a significant role in the prevention or development of neurodegenerative disease, specifically diet composition or profile. Both overnutrition and malnutrition lead to the pathogenesis of various neurodevelopmental complications. In late adulthood, deprived dietary habits such as a deficiency or imbalance of certain nutrients (vitamin B, omega-3 acids) have been linked to dementia and Alzheimer's disease (Ajibawo-Aganbi et al., 2020). In animal models, the balance of omega-3 and omega-6 acids, predominantly docosahexaenoic acid (DHA) and arachidonic acid (AA) respectively, have been shown to influence brain phospholipid fatty acid composition (Bianchi et al., 2021). Lipids within the brain assist in signalling within and between cells (Martínez & Mougan, 1998).

1.4 Pathophysiology of chronic disease

1.4.1 Chronic inflammation

When the body is injured, the immune system reacts by releasing types of white blood cells named macrophages to encircle, protect, and heal the affected area. This response mechanism is typically defined as inflammation. Inflammation is associated with the secretion of soluble mediators (proteins) such as cytokines, chemokines, and acute-phase proteins (Gusev & Zhuravleva, 2022). There are two types of inflammation: acute inflammation, characterised by short-term injury such as redness, swelling or pain around tissues and joints (Varela et al.,

2018), and chronic inflammation, where unresolved acute-phase inflammation (persistent inflammation, prolonged exposure to stimulation) leads to the development of tissue damage and many non-communicable diseases (Phillips et al., 2019). Acute inflammation is an essential phenomena indispensable for tissue repair and healing and handling of infection and is not particularly serious by itself. Acute inflammation can be caused by a multitude of simple triggers or traumas such as a small cut, bruise, laceration, allergic reaction, infection (bacteria, virus), burn, frostbite or chemical irritant (Hannoodee & Nasuriddin, 2020). On the other hand, chronic inflammation is low-grade inflammation that persists for long periods of time.

Chronic inflammation is associated with diseases within MetS including atherosclerosis (Libby, 2021), diabetes (Rohm et al., 2022), cancer (Coussens & Werb, 2002), arthritis (Shrivastava & Pandey, 2013) and bowel diseases like Crohn's disease (Tibble et al., 2000) and ulcerative colitis (Yao et al., 2019). The cytokines (produced by cells such as macrophages and monocytes) and chemokines (chemotactic cytokines), regulate the process of inflammation through stimulation; either accelerating (pro-inflammatory) or decelerating (anti-inflammatory) the inflammatory reaction or response. Interleukin-one alpha (IL-1α), one beta (IL-1β), six (IL-6), and tumour necrosis factor alpha (TNF-α) are the major pro-inflammatory cytokines responsible for early responses of inflammation (Kany et al., 2019). There are many pro-inflammatory genes within acute and chronic inflammation, some sharing an overlap (Figure 1.7).

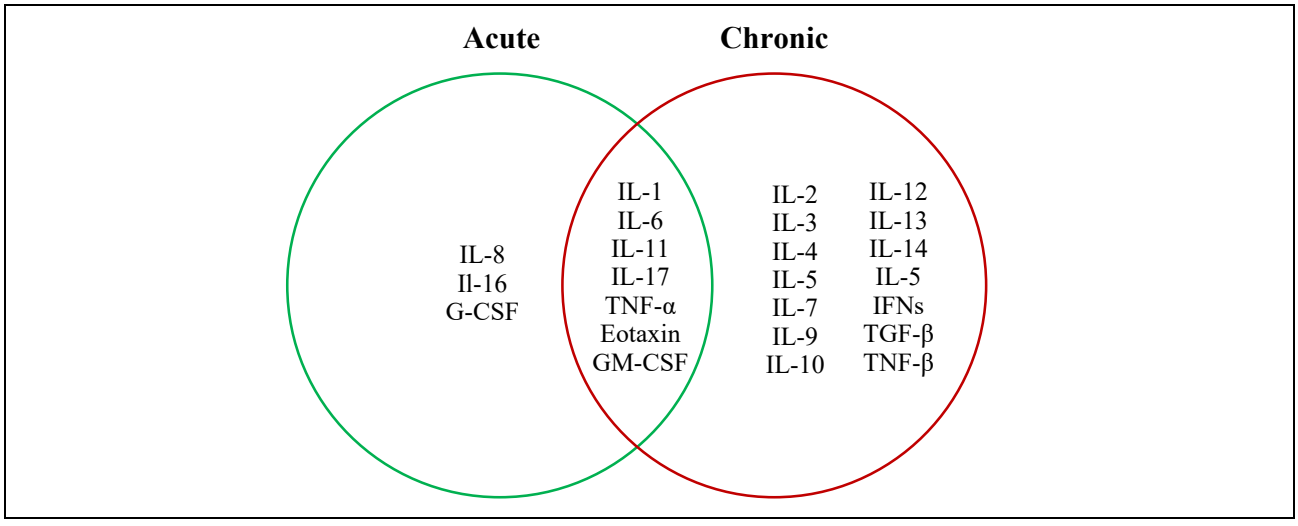


Figure 1.7. Pro-inflammatory genes in acute and chronic inflammation.

Note: Adapted from Feghali & Wright, 1997.

Anti-inflammatory cytokines regulate the pro-inflammatory cytokine human immune response by maintaining a balance between both pro-inflammatory and anti-inflammatory cytokines. This balance is vital for maintaining optimal health, as an imbalance can lead to prolonged inflammation, tissue damage and increased risks of disease. Interleukin- four (IL-4), ten (IL-10), and thirteen (IL-13) are the prominent anti-inflammatory cytokines (Marie et al., 1996). IL-10 is a particularly effective anti-inflammatory, inhibiting TNF-α, IL-6 and IL-1 expression by activated macrophages (Zhang & An, 2007). Other key identifier(s) of inflammation includes blood tests. Key plasma (serum) indicators of inflammation include 1: C-reactive protein (CRP) levels, CRP is secreted by the liver when inflammation occurs, more specifically in response to increased inflammatory cytokine production (Eklund, 2009). 2: Erythrocyte sedimentation rate (ESR), an analysis of the sedimentation (fall) rate of red blood cells within a test tube (El Maataoui et al., 2015). Ranges of ESR are dependent on both sex/gender and age, in millimetres per hour (mm/hr) (Siemons et al., 2014). 3: Lastly, plasma (liquid fragment of the blood) viscosity (PV) (Késmárky et al., 2008). Viscosity (thickness) of the plasma is affected by proteins within the blood, a typical response to inflammation. Plasma viscosity correlates with CRP levels in some instances, such as ulcerative colitis (Lobo et al., 1992), and is typically increased in patients with metabolic syndrome (Skretteberg et al., 2010). Normal (healthy) levels of each of these biomarkers are presented in Table 1.5.

Table 1.5. Identifiable biomarkers of inflammation.

Note: Adapted from El Maataoui et al., 2015; Kushner & Antonelli, 2015; Tishkowski & Gupta, 2020.

Biomarker	Healthy (normal) levels/range
C-reactive protein (CRP)	<10mg/L
Erythrocyte sedimentation rate (ESR)	Male <50 years old: ≤15 mm/hr Female <50 years old: ≤ 20 mm/hr Male >50 years old: ≤20 mm/hr Female >50 years old: ≤30 mm/hr Child: ≤10 mm/hr
Plasma viscosity	1.3-1.7mPas (millipascal second).

Metabolic inflammation is a chronic state of low-grade inflammation, associated with increased risks of MetS-related diseases; type 2 diabetes, MASLD and cardiovascular disease (Charles-Messance et al., 2020). Obesity associated cytokine signalling encompasses cytokine production by macrophages, leading to an increase of circulating free fatty acids (Lauterbach

& Wunderlich, 2017). In type 2 diabetes, pro-inflammatory cytokines are secreted as a response to β-cell damage (Dludla et al., 2023). Expression of cytokines TNF-α, IL-1 and IL-6 are linked to tumour progression (Zhao et al., 2021). Atherogenesis, inflammation and atherosclerotic progression within the artery walls; strongly associated with cardiovascular disease, is accelerated by cytokines. In MASLD, pathogenesis is promoted by pro-inflammatory cytokines TNF-α, IL-1 and IL-6 (Rafaqat et al., 2024).

Lipids play an essential role in resolving the inflammatory response, omega-3 fatty acids typically reduce inflammation, whereas omega-6 fatty acids can be pro-inflammatory (DiNicolantonio & O'Keefe 2021). It is widely accepted that both saturated fats and sugars are the key drivers in the development of metabolic inflammation. Obesity-induced elevated circulating palmitic acid levels promotes inflammation in peripheral tissues through the activation of pro-inflammatory toll-like receptor (TLR) signalling pathways. Post TLR2 and TLR4 activation, signals are transmitted through the MyD88 to IRAK pathway, as well as the PI3K to PKD/Akt pathways, resulting in the activation of NFκB (Korbecki & Bajdak-Rusinek, 2019).

Central to the mammalian cellular inflammatory response lies the nuclear factor kappa-light-chain-enhancer of activated β-cells, or NF-κB, system/pathway. NF-κB is an inducible transcription factor that both directly and indirectly regulates inflammatory gene expression, acting as a critical mediator in maintaining homeostasis (Figure 1.8a). Activation of NF-κB occurs through a multitude of stimuli in addition to cytokine production, including infection, stress, as well as viral or bacterial antigens (Prescott et al., 2021). The inflammatory response, beginning with IκBα (NF-kappa-B inhibitor alpha), employs various therapeutic processes targeted at NF-κB, including the inhibition of κB kinase (IKK) activity, inhibition of protease activity, inhibition of nuclear translocation, inhibition of DNA binding, and ultimately transcription inhibition (Figure 1.8b). The inhibitor of IKKs complex is paramount in the phosphorylation of inhibitor I-κB, regulating NF-κB activation through a negative feedback loop (Hinz & Scheidereit, 2014). The IKK complex consists of two kinases: IKKα and IKKβ, and a subunit: NF-kappa-B essential modulator (NEMO)/IKKγ (Israël, 2010). Dysregulation of NF-κB activation is instrumental in the pathogenesis of various chronic diseases, including certain cancers (Lawrence, 2009).

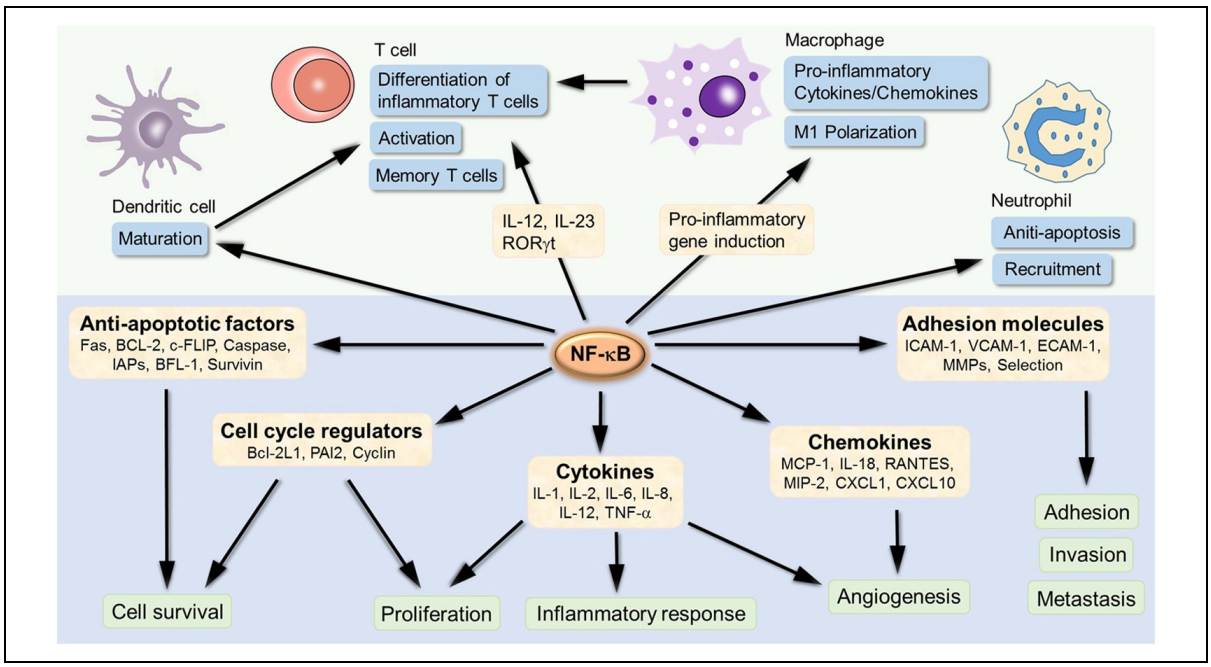


Figure 1.8a. The NF-κB system and associated molecular and cellular signalling pathways.

Note: From Liu et al., 2017.

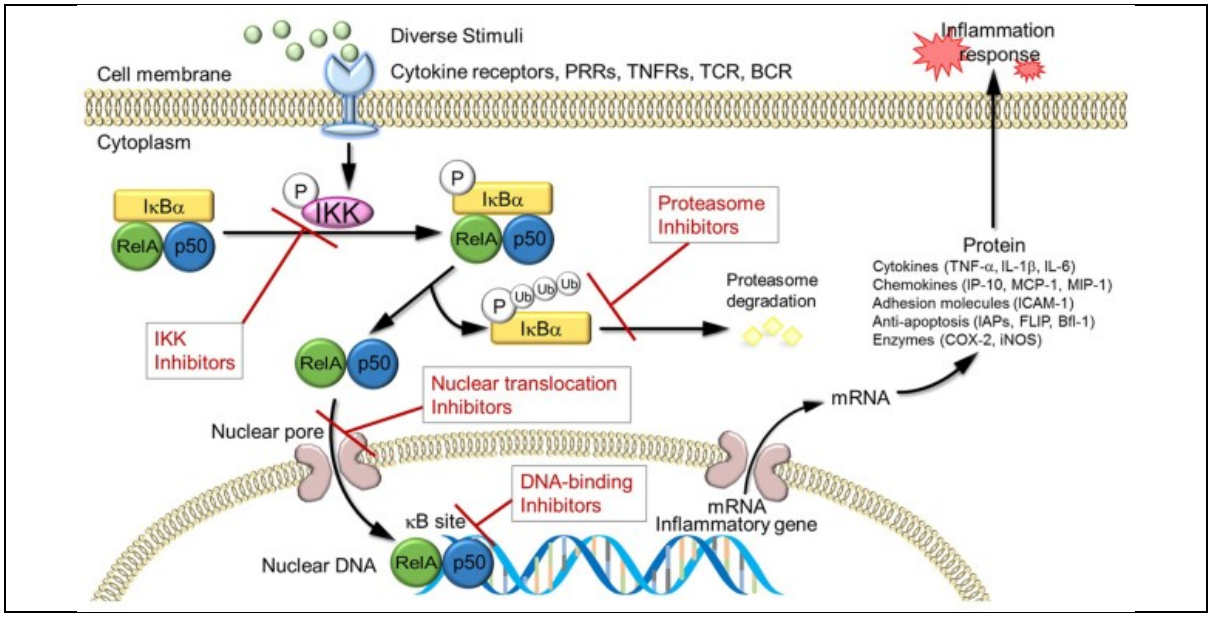


Figure 1.8b. NF-κB targeted therapeutics in inflammatory disease.

Note: From Liu et al., 2017.

1.4.2 Oxidative stress

Cellular oxidative stress is a normal physiological phenomenon. However, an imbalance in the relationship between oxidative forces such as free radicals and the antioxidant capability of bodily protective mechanisms, specifically when the former exceeds the latter (Yoshikawa &

Naito, 2002), can play a role in the pathogenesis of various chronic diseases. Oxidative stress is induced by various factors including antioxidants such as vitamin C, vitamin E, carotenoids, xanthine, curcumin, catechin and glutathione. (El-Demerdash et al., 2018); in addition to xenobiotics and heavy metals (Fryzova et al., 2018). Induced reactive oxygen species (ROS) causes an increase in peroxidation (damage by free radicals), in addition to protein and DNA damage, leading to the development of many diseases (Checa & Aran, 2020). Cellular anti-oxidation defence/balance systems assist in the preservation of oxidative homeostasis, neutralising excess reactive oxygen species (ROS). These defensive systems are typically one of two types; enzymatic and non-enzymatic (Kozlov et al., 2024). An important cellular defence system in oxidative-stress-mediated damage prevention is the Keap1-Nrf2 pathway, regulated by the interactions between NF-E2-related factor 2 (Nrf2) and Kelch-like ECH-associated protein 1 (Keap1) (Baird & Yamamoto, 2020). Within cytoplasm, Keap1 negatively regulates Nrf2 activity in basal (homeostatic) conditions, whereas conditions where stress occurs - Nrf2 will detach from Keap1, translocating to the nucleus (Figure 1.9). Initially, free radical production leads to cellular sensor Nrf2:INrf2(Keap1) interaction, which helps detoxify reactive oxygen species (ROS) and electrophiles, in addition to preventing free radical generation (Kaspar et al., 2009; Zhang et al., 2015).

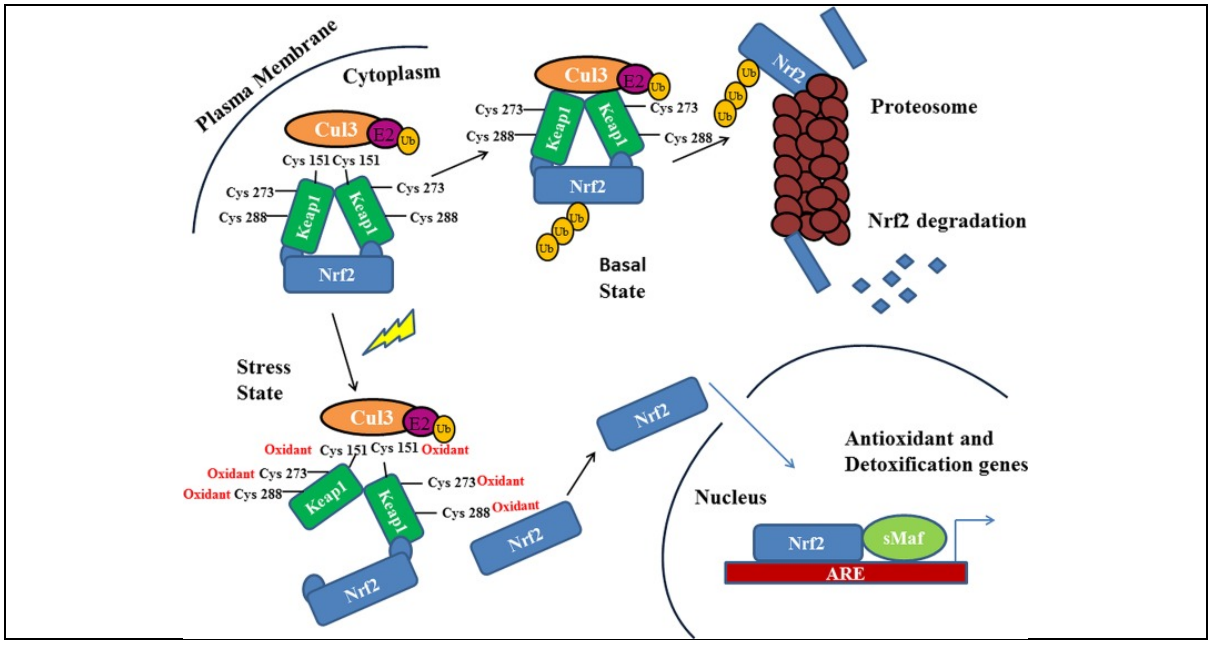


Figure 1.9. The Nrf2 regulation pathway.

Note: From Deshmukh et al., 2016.

Failure in the elimination of oxidants (free radicals), leads to the aforementioned imbalance and begins an accumulative effect on cells and cellular macromolecules; leading to cell damage, cell death (apoptosis), DNA damage and genome instability (mutations). These cellular effects can accelerate aging, as well as induce host chronic inflammation (Pizzino et al., 2017; Yu & Xiao, 2021). Oxidative stress induces inflammation through toll-like receptor 10 (TLR10) expression (Sindhu et al., 2018). Activation of the inflammatory pathway through transcription factor NF-κB activation, alongside excess ROS production, are common mechanisms in the chronic disease, atherosclerosis (Figure 1.10).

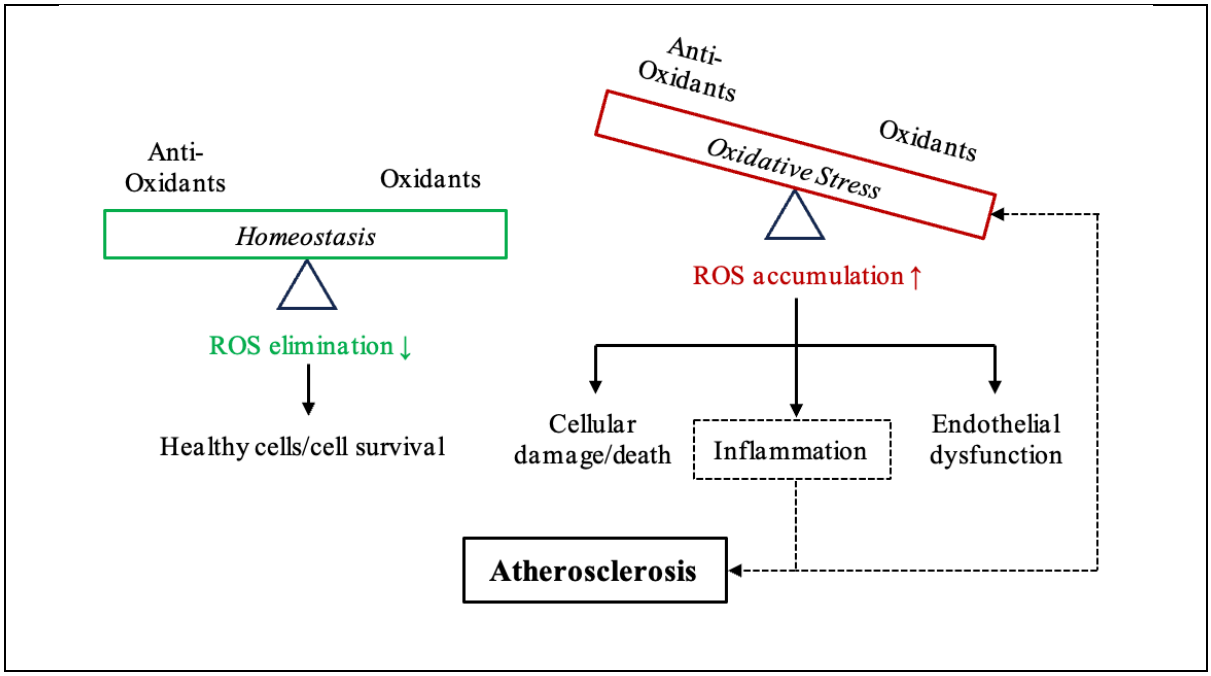


Figure 1.10. Homeostasis and imbalance outcomes within oxidative stress.

Note: Adapted from Kattoor et al., 2017.

Oxidative stress homeostasis is influenced greatly by dietary intervention. Increased lipid (i.e., saturated fatty acids, trans-fatty acids) and carbohydrate (i.e., sucrose, fructose) consumption (Western diet) increases oxidative stress, with post-prandial effects strongly linked to ROS accumulation and dysregulation. Specific dietary foods and nutrients can mediate regulation, inhibit inflammation, and lower the risks atherosclerosis. The Mediterranean diet (MD), high in vegetable oils, seafood, fruits, vegetables, beans, nuts and wholegrains, in addition to the active avoidance in consumption of processed foods, trans fats and refined sugars (Davis et al., 2015), has been found to ameliorate oxidative stress overloads due to an abundance of plant-based foods rich in antioxidants (Tuncay & Ergoren, 2020; Jiang et al., 2021).

1.4.3 Glucose homeostasis

Blood glucose plays an important role as a source of metabolic energy for the cell and storage of energy (Qaid & Abdelrahman, 2016). Following digestion of carbohydrates, within the gastrointestinal system, sugar/glucose is absorbed in the small intestine and eventually released into the bloodstream for the transportation to individual cells. (Patricia & Dhamoon, 2019). The homeostatic range of blood glucose in a healthy adult is between 4.0mmol/L (72 mg/dL) and 6.0 mmol/L (108 mg/dL) (Mathew & Tadi, 2020). Following the absorption of dietary sugar, a temporary spike in the blood is expected (Sinar & Charles, 1983). A typical profile of the blood glucose following consumption of a carbohydrate rich food in a healthy individual is shown in Figure 1.11.

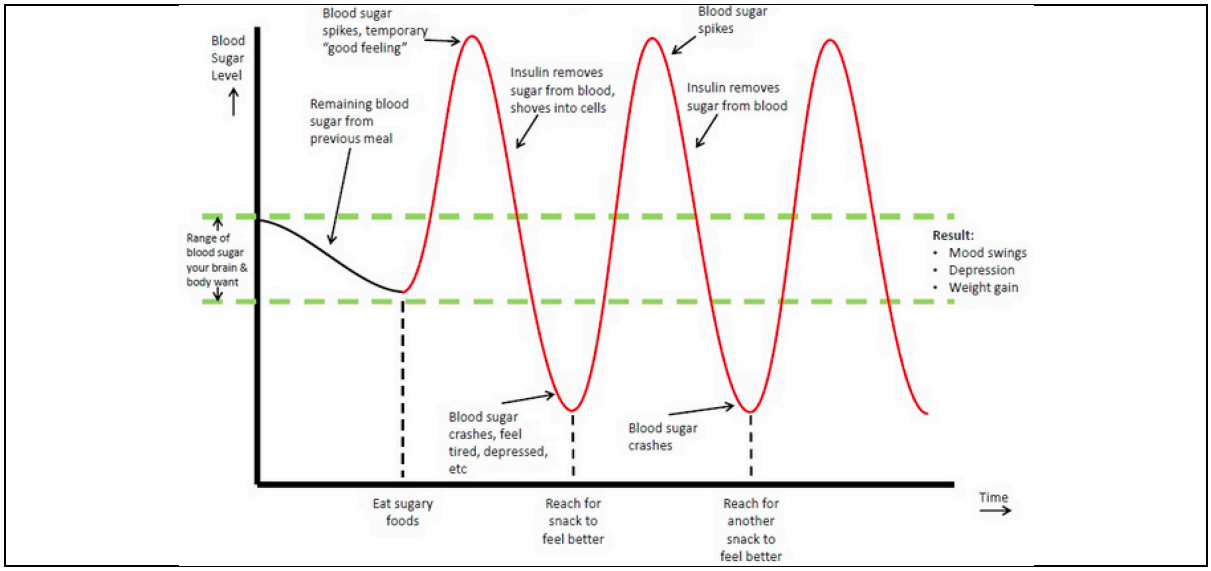


Figure 1.11. Post-prandial effects of carbohydrates on blood glucose.

Note: From Faruqui, 2017.

An elevated level of circulating blood glucose is sensed by the pancreatic beta-cells that secrete insulin, a vital hormone required for the triggering the cellular mechanism ultimately leading to the entry, metabolism, and storage of glucose derived metabolites within the cell. Hence, there is a positive association of blood glucose level with that of circulating level of insulin. The level of glucose in blood is tightly regulated because a prolong high blood glucose (hyperglycaemia) cause damage to vascular tissues (Ruderman et al., 1992) including damaging organs like kidneys. The glycaemic control system is comprised of the interplay of insulin and glucagon, two important hormones produced by the pancreas. Low blood glucose status triggers secretion of glucagon that induces break down of glycogen in the liver and

skeletal muscle and release of free glucose for metabolism. On the other hand, higher concentrations of blood glucose triggers secretion of insulin that results in entry and utilization of glucose in metabolism (Qaid & Abdelrahman, 2016). The inability of the glycaemic control system to regulate blood glucose results in either too low level of blood glucose (hypoglycaemia) or high level of blood glucose (hyperglycaemia) (Mukhtar et al., 2020) which are the hallmark of type-2-diabetes. One of the underlying causes behind the failure of the glycaemic control is insulin resistance.

Dietary fat, or high-fat diets containing increased saturated fat intake (particularly arachidonic acid and palmitic acid) can adversely affect insulin sensitivity, resulting in insulin resistance (von Frankenberg et al., 2017). Fatty acid induced insulin resistance can affect various areas of the body, including the hypothalamus, liver, adipose tissue, skeletal muscle, pancreas, and gastrointestinal tract (Figure 1.12).

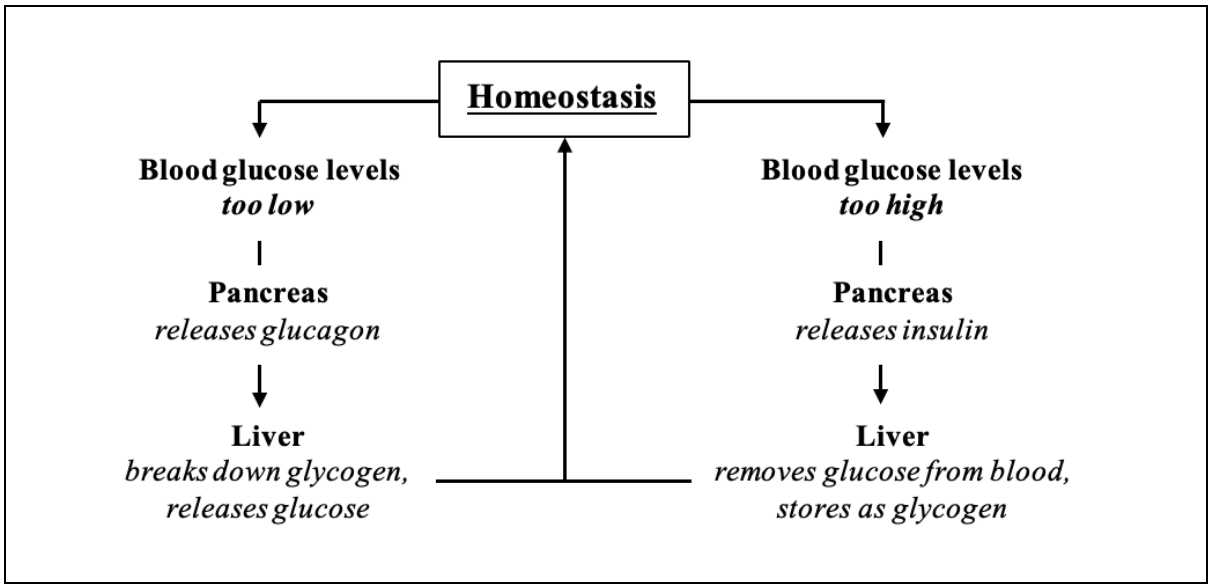


Figure 1.12. Blood glucose homeostasis feedback within the liver.

Note: Adapted from Röder et al., 2016.

Palmitic acid induces insulin resistance within macrophages by activating the TLR4 to NFκB signalling pathway (Amine et al., 2021). Excess consumption of sugars leads to the inefficient utilisation of sugar being converted into energy and instead stored as fat, fostering insulin resistance (Laville & Nazare, 2009). Fatty acid mediated insulin resistance affects the

hypothalamus, liver, adipose tissue, skeletal muscle, pancreas, and gastrointestinal tract (Sears & Perry, 2015).

1.4.4 Dyslipidaemia

Dietary lipids provide sources of fatty acids, storage of energy and sources of metabolic fuel for the body (Alves-Bezerra & Cohen, 2017). Following the absorption of dietary fats, lipids are absorbed into the small intestine through vessels named lacteals, then transported to the blood stream via the lymphatic system. Lipids are typically considered one of four types: fatty acids, glycolipids, phospholipids, and sphingolipids. Another type of lipid, cholesterol, is a chemical compound both found and created homogenously, as well as absorbed (in small amounts) from animal livers, or foods such as meat, poultry, and dairy products; with its' main directive being cell, hormone, and vitamin production (Craig et al., 2018). There are two key types of cholesterol: low-density lipoprotein cholesterol (LDL-C), oft-reported as atherogenic, aids in cholesterol transportation; and high-density lipoprotein (HDL-C), believed to possess cardioprotective properties, which returns excess cholesterol to the liver. Optimal levels of cholesterol are as follows - total cholesterol (TC): <11.1 mmol/L (<200 mg/dL); LDL-C: <5.6 mmol/L (<100 mg/dL); HDL-C >2.2 mmol/L (>40 mg/dL) (Ma & Shieh, 2006). Cholesterol and triglycerides pass through the bloodstream, assisted by these lipoproteins (particles of fats and protein) (Olson, 1998).

Lipid dysregulation (dyslipidaemia) can be defined as an imbalance of lipids, a disorder in which irregular cholesterol levels or an abundance of triglycerides are present (Berberich & Hegele, 2022). Dyslipidaemia is strongly associated with atherosclerotic cardiovascular disease (ASCVD) or atherosclerosis (Nelson, 2013), as fat or cholesterol abundance can lead to build-up or blockage in artery walls. Blockages within coronary arteries make it difficult for the body to maintain blood flow towards the heart. Other conditions or disorders immediately relating to dyslipidaemia include: hyperlipidaemia (high lipid levels), increasing risks of cardiovascular disease and stroke (Alloubani et al., 2021); hypercholesterolemia (excess levels of LDL-C), increasing risks of myocardial infarction and CVD (Mortensen & Nordestgaard, 2020); hypertriglyceridemia (triglyceride abundance), increasing risks of pancreatitis (Yang & McNabb-Baltar, 2020).

Dietary fatty acids, particularly saturated fats, and trans fats, as well as animal-origin foods have strong negative effects on serum lipids (Siri-Tarino et al., 2010). In addition, the vegetable oil: palm oil, has been shown to increase LDL-C significantly higher than its lower-in-saturated-fat counterparts (Sun et al., 2015). Palmitic acid has been found to have deleterious effects on lipid metabolism; however, it is unclear whether or not this stems from dysregulated de novo lipogenesis (DNL), or dietary palmitic acid itself (Murru et al., 2022). De novo lipogenesis is a metabolic pathway that utilises acetyl coenzyme A **(**acetyl-CoA) to synthesize saturated fatty acids, in addition to other molecules such as sugars or amino acids. Excessive accumulation of lipids (lipotoxicity), predominantly found in obese individuals, particularly in the form of saturated fatty acids, are deadly to cells (Schaffer, 2016). Excess palmitic acid plays a major role in lipotoxicity: treatment of surplus palmitate within non-adipose cells reportedly increases saturated glycerolipids, as well as promotes the over-production of lipid metabolism enzymes glycerol-3-phosphate acyltransferase 3 (*GPAT3)* and *GPAT4* - leading to an upregulation of endoplasmic reticulum stress, cellular damage and ultimately, apoptosis (cell death) (Figure 1.13).

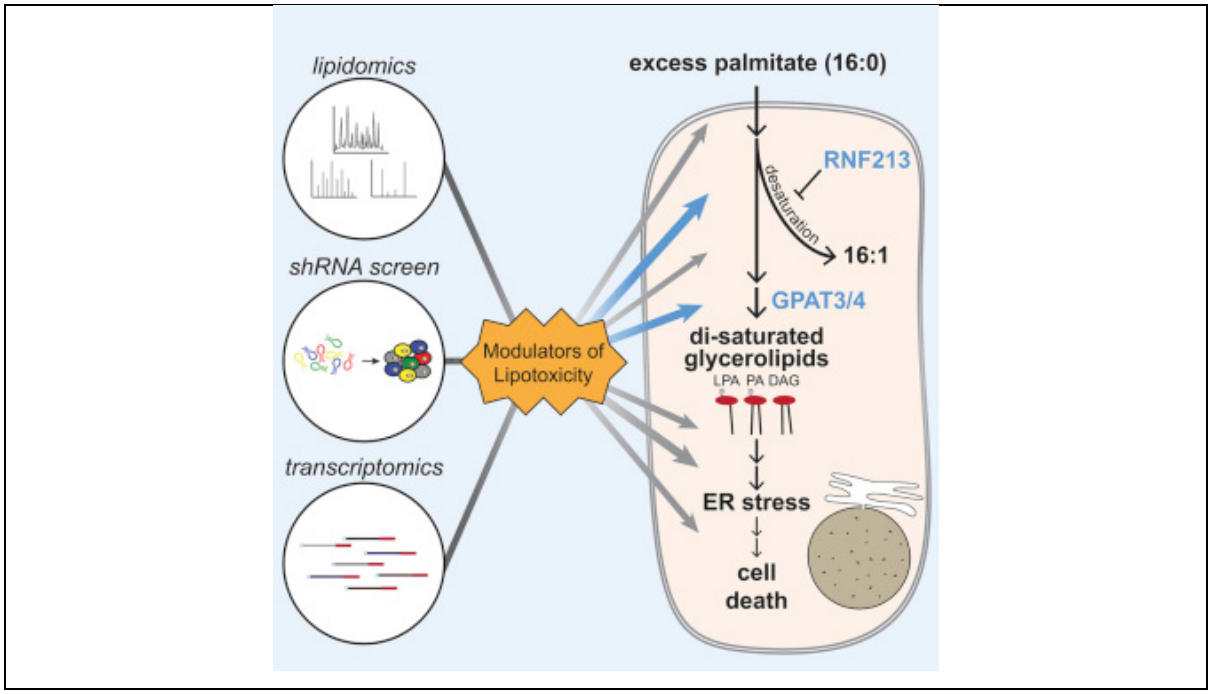


Figure 1.13. Excess palmitic acid (palmitate) role in lipotoxicity.

Note: From Piccolis et al., 2019.

1.4.5 Gut microbiology

Growing evidence over the past two decades has indicated that the human gut microbiome, and intestinal microbiota, greatly effect host metabolic health (Fan & Pederson, 2021). From around the start of the millennium, studies began to suggest this strong correlation, specifically the composition of the microbiome (diversity of microbiota) and the many influences of pre- and pro-biotics (Macfarlane & Cummings, 1999; Steer et al., 2000). Most microorganisms within the gut have a symbiotic and mutually beneficial relationship with the host, communicating and co-operating with various organs and tissues within the body, including the immune system, aiding in the strengthening of gut integrity and protection against harmful pathogens (Thursby & Juge, 2017), as well as the deterrence or prevention of the development of disease (Zheng et al., 2020).

Each host has a unique arrangement of intestinal microorganisms residing in their gastrointestinal (GI) tract (Gilbert et al., 2018). Early life (infant) microbiota profile reflects future health of the host (Isolauri et al., 2012). These microorganisms, known as gut microbiota or gut flora, include a multitude of types of bacteria, archaea, fungi (mycobiota) and viruses (Hugon et al., 2017). The dominant bacterial phyla are Firmicutes (Bacillota) and Bacteroidetes (Bacteroidota); accounting for 90% of gut microbiota (Magne et al., 2020), as well as Actinobacteria (actinomycetota), Proteobacteria (pseudomonadota), Fusobacteria (fusobacteriota), and Verrucomicrobiota. Trillions of microbiota(s) are situated within the gut microbiome, specifically in the stomach, duodenum, jejunum, ileum, and colon (Cresci & Bawden, 2015). Diet is one of the three (in addition to aging, antibiotics) principal factors that influence microbiota (Hasan & Yang, 2019). A high fibre diet has a strong correlation with a healthy microbiome, whereas a high-fat, low-fibre diet (Western diet) leads to dysbiosis (Figure 1.14).

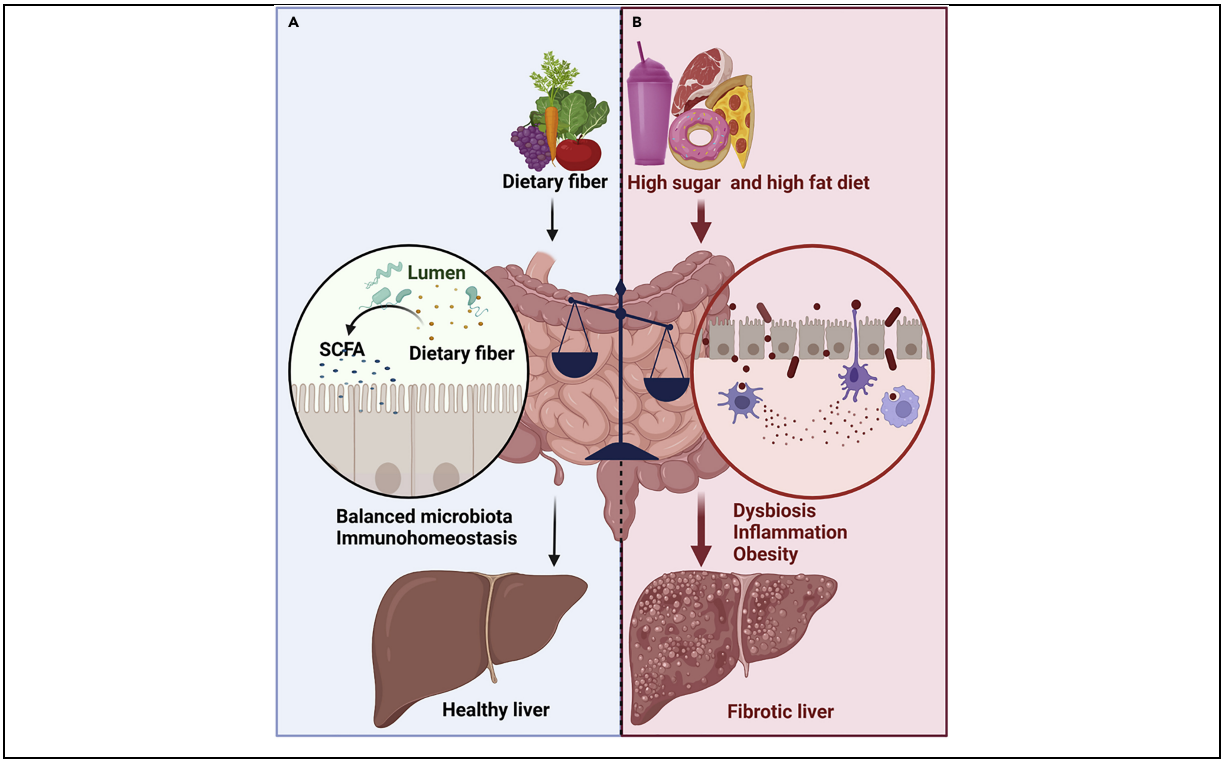


Figure 1.14. Adverse effects of dietary imbalances (high-fat, high-sugar) on gut health.

Note: From Kang et al., 2023.

Dysbiosis, an imbalance in bacterial composition within the gastrointestinal tract (GI), can induce the pathogenicity of several common metabolic disorders i.e., obesity, non-alcoholic fatty liver disease and type 2 diabetes (Chang & Lin, 2016). Loss of diversity or an imbalance, is the microbiome in a less-rich and less-diverse state, leaving the gut open to external threats (Iacob & Iacob, 2019). Many factors can affect this disproportion, including genetics, medications, host health (e.g., inflammation) or mental health (psychological or physical stress, anxiety), dietary changes (e.g., increased levels of alcohol), or poor nutrition such as overfeeding or malnourishment (Myers & Hawrelak, 2004). The Firmicutes/Bacteroidetes (F/B) ratio has a strong influence on maintaining normal, healthy, intestinal homeostasis (Stojanov et al., 2020). There are several risk factors involved in the reduction of diversity of gut microbiota (Table 1.6).

Table 1.6. Risk factors of acute and chronic microbial inflammation.

Note: Adapted from Nagpal et al., 2014.

Cause	Effect	Implication(s)
Prudent diet & lifestyle Pre- & Probiotics	**Gut microbial homeostasis** ↑ *beneficial/commensal* ↓ *harmful/opportunistic*	↓ Gut permeability ↓ Toxaemia/Sepsis ↓ Proinflammation ↑ Insulin sensitivity ↑ Gut/metabolic/cardiovascular health
High-fat/high-sugar diets Over-nutrition Sedentary lifestyle	**Gut microbial dysbiosis** ↑ *harmful/opportunistic* ↓ *beneficial/commensal*	↑ Gut permeability ↑ Endotoxemia, septicaemia ↑ Systemic inflammation ↑ Insulin resistance ↑ Adiposity, diabetes, MetS, CVD ↑ NAFLD/NASH, IBD/IBS
MetS = metabolic syndrome. CVD = cardiovascular disease. NAFLD/NASH = non-alcoholic fatty liver disease/steatohepatitis. IBD/IBS = irritable bowel disease/syndrome.		

Gut dysbiosis is used as a biomarker in many disorders, including type 2 diabetes, inflammatory bowel disease (IBD) colorectal cancer, celiac disease (Mosca, et al., 2016), chronic fatigue syndrome (Giloteaux, 2016), polycystic ovary syndrome (Lindheim, et al., 2017), as well as insulin resistance, hypercholesteremia and intestinal inflammation (Le Chatelier et al., 2013). Overweight, or obese, individuals showcase lower diversity and increased risks of dysbiosis (Singh et al., 2023). Chronic inflammation of the gastrointestinal (GI) tract, also known as inflammatory bowel disease (IBD), refers to two conditions: Crohn's disease and ulcerative colitis (Manichanh et al., 2012). A microbial imbalance, or archaea abundance has been found to be present in patients with inflammatory bowel disease (Alam et al., 2020) and colorectal cancer (Coker et al., 2020). Toll-like receptor 2 (TLR2) and toll-like receptor 4 (TLR4) are upregulated during intestinal inflammation, suggesting they play a role in the prevention of inflammation and maintaining gut homeostasis (Hausmann et al., 2002). TLR2 and TLR4 have been reported to sense gram-negative bacteria induced inflammatory responses (Frolova et al., 2008), such as the detection of lipopolysaccharide (LPS) by TLR4 (Soares et al., 2010). Dysbiosis typically coincides with increased intestinal permeability, leading to hyperpermeability, also known as leaky gut syndrome (Stewart et al., 2017). The primary cell surface of the GI tract is the mucous layer, the integrity of this layer assists with the safe passage of, as well as the absorption of, water and nutrients (from food) into the bloodstream (Kiela & Ghishan, 2016). Whilst gut microbiota has been reported to be an

essential factor in the pathogenesis of metabolic syndrome (Wang et al., 2020); several recent studies suggest that the role of gut microbiota in disease is still very poorly, or at least not fully yet, understood (Bennedsen et al., 2022; Dissanayaka et al., 2024; Liu et al., 2021; Rinninella et al., 2019; Sidhu et al., 2023).

Lipid-microbiota interaction can influence metabolic health in various ways. Low intake of saturated fatty acids contributes to an increase in microbial diversity (Schoeler et al., 2023). Highly processed foods have been found to compromise the lining of the gut barrier (Mansour et al., 2021). When the epithelial barrier integrity is compromised, this leads to an increased risk of exposure to inflammation and carcinogenesis, primarily colorectal cancer (Akbar et al., 2022). Plant-based fats and animal-based fats are believed to influence different types of microorganisms. Plant-origin fats upregulate beneficial microbiota and downregulate harmful microbiota, whereas the animal-origin fats show opposing effects (Figure 1.15).

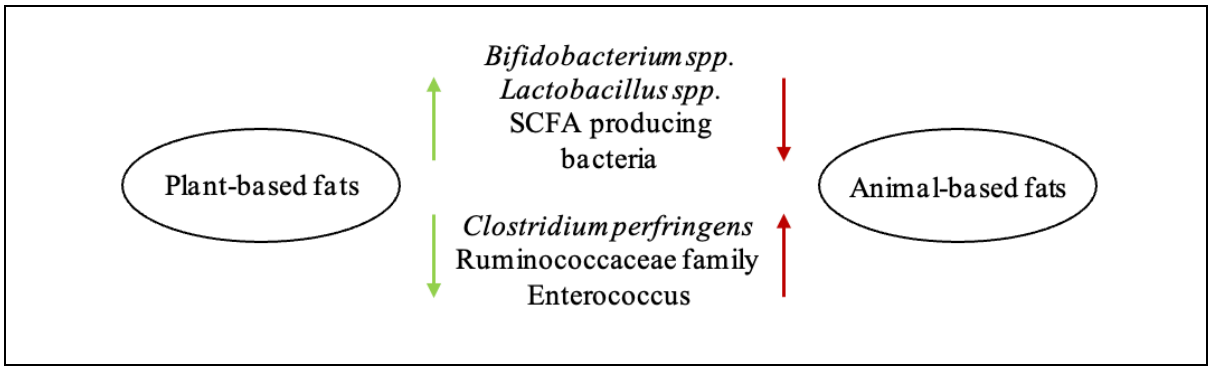


Figure 1.15. Effects of plant-based versus animal-based fats on gut microbiota.
Note: Adapted from Muralidharan et al., 2019.

1.5 Role of dietary habits in health

To be healthy, a diet should have a mix of vital nutrients such as carbs, fats, proteins, minerals, vitamins, and water, along with other beneficial compounds like phytochemicals (Traka et al., 2020). Dietary habits refer to the habitual preferences and choices regarding nutrient intake and specific food groups (Hu, 2002). Some common dietary habits include omnivores who eat both animal and plant foods, pescatarians who mostly consume fish and seafood, flexitarians who limit animal-based products, vegetarians who avoid meat but include dairy and eggs, and vegans who stick to plant-based foods. Some dietary habits are effective in achieving some

specific health outcome. For instance, Atkins diet or ketogenic diet (a low-carbohydrate- high-fat) can reduce the body fat content as well as lowering of blood pressure (Filippou et al., 2020).

Dietary patterns based on the geography, such as the Mediterranean diets have characteristic consumption of some food ingredients such as the olive oils (García-Montero et al., 2021). The Mediterranean diet also characterised by a high consumption of fruits, vegetables, legumes, and wholegrains as well as fish, seafood and vegetable oils (Willet, 2006). Health benefits of the Mediterranean diet are contributed by the higher intake of monounsaturated fatty acids and polyunsaturated fatty acids and limited presence of saturated fatty acids and trans fats. Higher intake of vegetables, fruits, nuts and legumes and a lower intake of animal-based products are believed to contribute to the lower incidence of chronic diseases (Martín-Peláez et al., 2020). In contrast, the western dietary habit heavily relies on the consumption of processed and ultra-processed foods, foods from animal origins (meat and dairy) and refined grans with limited consumption of fruits, vegetables, and dietary fibre (Azzam, 2021). The growing epidemics of chronic diseases in the western world is blamed due to a higher consumption of ultra-processed foods and a sedentary lifestyle (Malesza et al., 2021).

1.5.1 Lipids in health and disease

Over the last 100 years; dietary fat, an important source of energy for the human body, has changed considerably with regards to quality and quantity (Gifford, 2002). Lipids, naturally occurring compounds intrinsic to food sources as well as synthesized by the body, are an important tool of energy metabolism (Beenakkers et al., 1981), as well as metabolic health and disease (Yoon et al., 2021). Types of lipids include fats and oils (triglycerides), cholesterol, phospholipids, as well as waxes and steroids (Asokapandian et al., 2021). In excess of 95% of lipids in the diet are that of triacylglycerols (triglycerides) (Ros, 2000). Fatty acids are characteristically hydrocarbon chains which act as major components of triacylglycerols, the main contributors to dietary fat in humans (Calder, 2015). There are many different types of fatty acids (FAs), classified precisely by their unique bond structure; usually, unbranched and even numbered with 4 to 28 carbon atoms (Chemical Entities of Biological Interest, 2021), the most common ranging from 12-22 (Rustan & Drevon, 2005). The two most predominant types of fatty acids, saturated fatty acids, and unsaturated fatty acids, each have a distinctive arrangement (Figure 1.16).

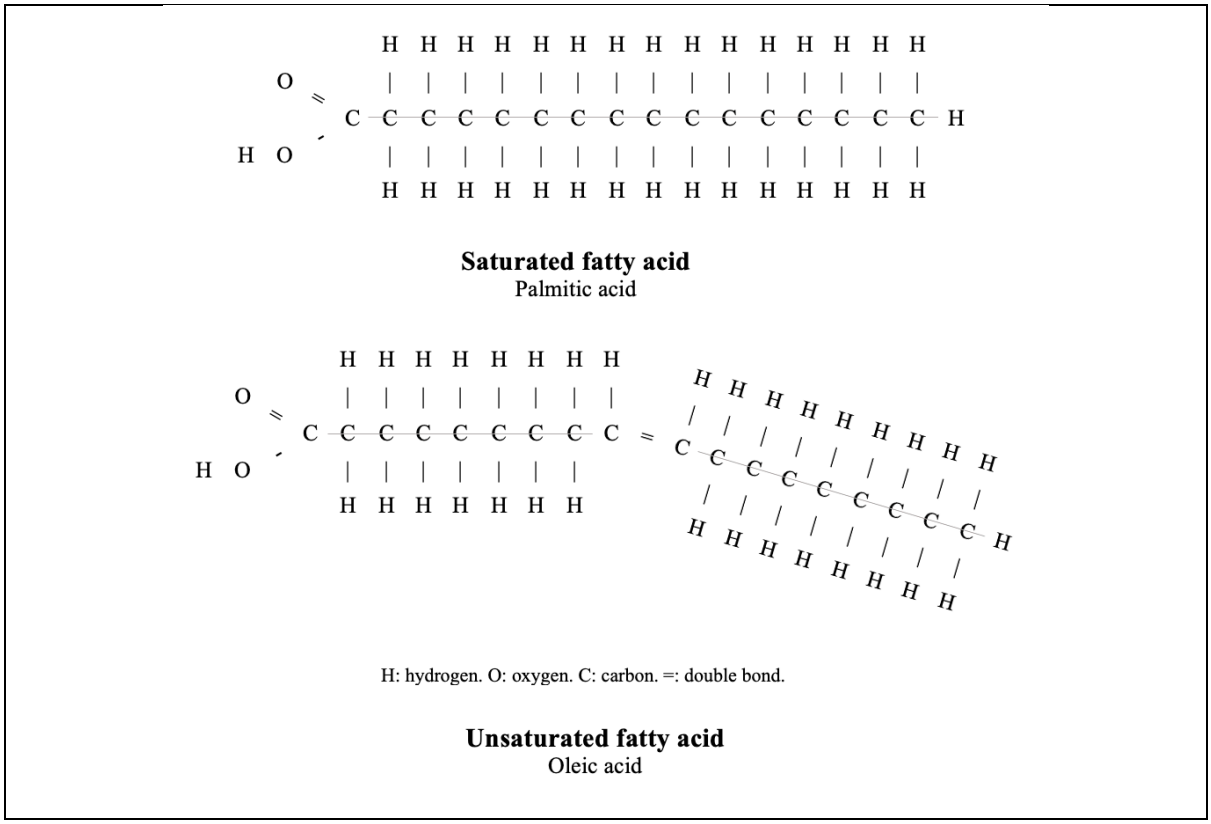


Figure 1.16. Chemical structure of example saturated and unsaturated fatty acids.

Animal-derived fatty acids from sources such as red meat, cheese, or ice cream, are generally saturated (German & Dillard, 2004), whereas fish- and plant-derived fatty acids from sources such as salmon, edible oils, or nuts, are usually unsaturated (Lunn & Theobald, 2006). Interestingly, plant-based alternatives (to meat and dairy) share nutritional similarities to their animal-based equivalents (Appendix 1.1). Once consumed; fatty acids, as structural components of fats (or triglycerides), are broken down during digestion, subsequently absorbed (via the small intestine) into the blood, cells, and tissues of the human body (Ratnayake & Galli, 2009). The most prevalent fatty acids found in both animal and plant origin food items are the 16-carbon chain and 18-carbon chain fatty acids, known as palmitic (hexadecenoic) acid and stearic (octadecanoic) acid (Ouellette & Rawn, 2018). PA (C16:0) molecular formula is $C_{16}H_{32}O_2$ (PubChem, 2023). These particular acids are defined as saturated fatty acids (SFAs), denoting that they are 'saturated with' (containing the maximum number of) hydrogen atoms, due to the presence of zero carbon-to-carbon double bonds (Astrup et al., 2020). An SFA-rich diet increases LDL cholesterol levels (Siri-Tarino et al., 2010), increases inflammation (Rocha et al., 2016) and risks of cardiovascular disease (Hooper et al., 2020). The most abundant saturated fatty acid in foods is palmitic acid, predominantly found in animal-based products

such as meat and dairy, however it is also found in highly prevalent plant-based sources such as vegetable oils (e.g., palm oil, olive oil), dark chocolate and various non-dairy spreads (i.e., margarine) (Unhapipatpong et al., 2021). The Scientific Advisory Committee on Nutrition (SACN) advocates replacing saturated fat with unsaturated fats to reduce risks of chronic disease, including heart disease and stroke (Public Health England, 2019).

Unsaturated fatty acids are defined when there is at least one double bond present within the fatty acid chain, broken down further into monounsaturated fatty acids or MUFAs (one double bond) and polyunsaturated fatty acids or PUFAs (two or more double bonds) (Roche, 1999). A MUFA-rich diet increases fat oxidation (Polley et al., 2018), as well as insulin sensitivity (Paniagua et al., 2007). A PUFA-rich diet (including essential and semi-essential fatty acids) reduces blood triglyceride levels, increases HDL cholesterol, and lowers LDL cholesterol (Kaviani et al., 2019). Fatty acids can be synthesized endogenously, typically from glucose or from other fatty acids (Wakil, 1961).

'Essential' fatty acids (often referred to as EFAs) are defined explicitly as exceptions to this rule; unable to be synthesized in the human body, these fatty acids must be attained through dietary consumption (Innis, 1991). α-linoleic acid (ALA), an omega-3 (ω-3) fatty acid and linoleic acid (18:2), an omega-6 (ω-6) fatty acid are considered to be the two only truly 'essential' fatty acids. However, 'semi-essential' (limited production within the human body) fatty acids (Lehner et al., 2021) such as eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) are believed to be regarded with as much significance (Stark et al., 2016). Vegetable oils are rich sources of alpha-linolenic acid (ALA) whereas eicosapentaenoic acid (EPA) and docosahexaenoic acids (DHA) are abundantly found in fish and fish oils (Kris-Etherton et al., 2000). Another important dietary fat is trans-unsaturated fatty acids, or trans fatty acids (TFAs), a both naturally-occurring (ruminants) and artificially produced (refined and processed) unsaturated fat (Dhaka et al., 2011). TFAs typically develop within processed food products through industrial processing of vegetable oils, named hydrogenation - however, trace amounts do occur naturally in meat and dairy (Ascherio & Willet, 1997). Consumption of TFAs is believed to harmful for human health and can significantly increase the risk of cardiovascular disease (Remig et al., 2020). TFAs are typically found in snack foods such as cakes, cookies, popcorn, dough, as well as processed or fried foods (fries, doughnuts, fried chicken) and margarine or shortening (Dhaka et al., 2011). In addition to being processed, fatty acids can

also be affected by cooking and storage methods, such as moisture loss when cooking or succumbing to rancidity (Gruffat, 2021).

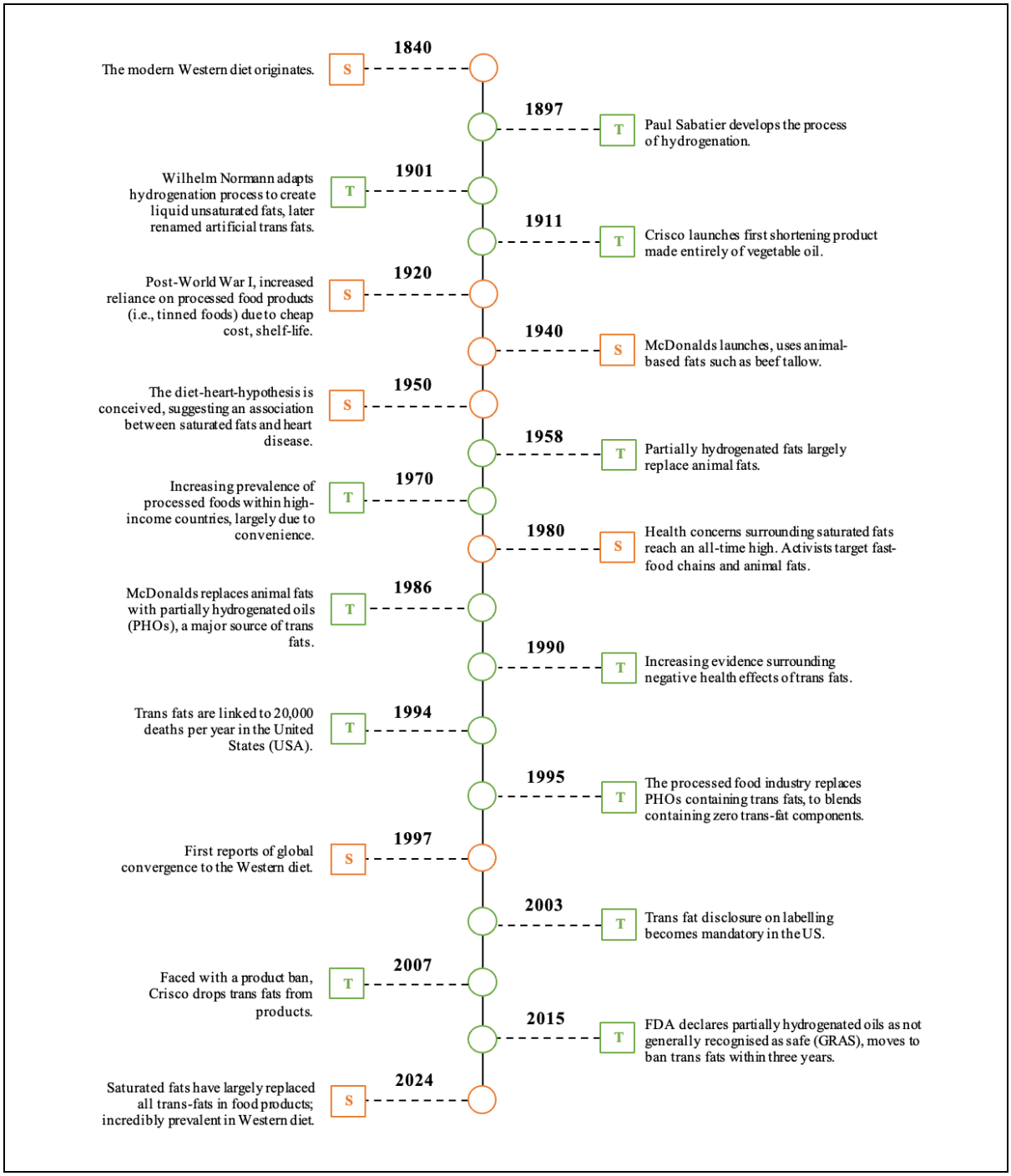
1.5.2 Lipids in food processing and preservation

Use of lipids in food processing and preservation have evolved over the last two centuries (a timeline of important events is displayed in Figure 1.17). At the turn of the 20th century, soybean oil was routinely used to create hydrogenated trans-fat-based food items such as margarine and vegetable shortening. Trans-fat replaced butterfat or buttermilk, beef-tallow, and lard (Prodöhl, 2023). From 1940 onward, 'speedee drive-ins', 'fast food' or 'quick serve' restaurants, led by brands such as McDonalds, Kentucky Fried Chicken (KFC), Burger King and Taco Bell, were at their pinnacle in popularity. These fast-food eateries were popular due to convenience, speed, low cost and foods with better consumer acceptability and hyper-palatability. These restaurants used to fry their foods in 100% fat predominantly containing animal-based saturated fat and beef-tallow (Quinton, 1990). The UK followed this trend shortly after, with its' first KFC restaurant opening in Preston, Lancashire in 1965 (Jones et al., 2022).

During the 1960s, processed vegetable fats became increasingly more popular gradually replacing the use of animal fats, due to health concerns and recommendations over lower saturated fat intake (Teegala et al., 2009). Alongside this, ultra-processed foods were becoming incredibly prevalent in most diets through the 1970's (Baker et al., 2020); perhaps in part due to the rise in home use of microwave ovens and the convenience of a pre-cooked packaged meal. By the 1980s, health concerns (particularly cardiovascular disease) surrounding saturated fats grew to an all-time high, leading to both UK and US dietary guidelines/recommendations of limiting saturated fat diet intake to ≤10%, consistent since 1983, and 1990, respectively (Astrup et al., 2020; Walker, 1983). Additionally, activism groups advocated for the replacement of trans fats with vegetable oils (Schleifer, 2012). Fast-food restaurant chains responded by replacing animal-based saturated fats with partially hydrogenated vegetable-based oils, high in trans-fats, yet generally perceived to be benign or innocuous at the time (Amico et al., 2021).

As of 2025, vegetable oils (although generally trans-fat free) are still largely used in industrial processing of food items such as jams, butters, cookies, cakes, crisps, as well as 'healthier' (less animal-fat) versions of animal-based products or meats (Murphy, 2024). Nevertheless,

due to the abundance of reports and scientific research in the 1990s reporting adverse effects of trans-fats: increased cardiovascular risk (Dwyer, 1995), increased risks of myocardial infarction (Ascherio et al., 1994), hypercholesterolaemia, atherogenesis, obesity, insulin resistance (Mann, 1994) and coronary heart disease (Willett et a., 1993); the use of trans-fats in the processed food industry were restricted. On account of these new findings and public outcry, the processed food industry sought a replacement in their products, ideally a cheap-to-produce, lucrative, non-hydrogenated, zero-cholesterol, trans-fat-free fat source, with a prolonged shelf-life.



Note: s = saturated fats, t = trans fats.

Figure 1.17. Timeline of important milestones in dietary fat evolution.

1.5.3 Entry of Palm oil into the food chain

In the early 19th century, oil derived from the palm fruit (*Elaeis guineensis*) was imported into Britain from Africa. Palm oil was perceived as a cheaper alternative to animal-based oils and was used in the production of inedible products such as soaps and candles, as well as industrial

lubricants (Lynn, 1992). It wasn't until the turn of the century that 'Palm-nut oil', the Chambers' Encyclopaedia definition as of 1865 (Chambers et al., 1888), became a mainstay commodity in certain edible products. As a cheaper alternative to butter, palm oil was used in foods such as margarine, giving it a light-yellow colour due it's carotene content (van Stuyvenberg, 1969). Because of the World War I (1914-1918), the Great Influenza (1918-1919) and the Great Depression (1929-1939); the West suffered severe poverty, food shortages and malnourishment, at the same time good advancement in food processing and preservation (Welch & Mitchell, 2000).

Following the Second Industrial Revolution (1870-1914), the cultivation of oil palm started in Southeast Asia (Indonesia, Malaysia) (Giacomin, 2018). Post-World War II (1939-1945), independence, refinement technology advancements and contemporary transport methods allowed palm oil production to continue its growth for the next few decades. In 1975, palm oil overtook both groundnut oil and cottonseed oil in worldwide vegetable oil production (also above coconut oil, olive oil, linseed oil, palm kernel oil), staying at the top and consistently growing to this day. More recently, palm oil has exceeded the production of soybean oil, rapeseed oil and sunflower oil (Food and Agriculture Organization of the United Nations, 2025). By the mid-1990s, palm oil was sought as the replacement for partially hydrogenated oils and trans-fats, leading to an explosion in popularity. Over the past twenty years, palm oil production has quadrupled in Indonesia alone (Kraus et al., 2021). Total worldwide production of palm oil for 2023/24 reached over 78 million metric tonnes, with Indonesia and Malaysia still leading by a large margin (Table 1.7). Palm oil, together with rapeseed oil and sunflower oil account for approximately 76% of oil consumed in the United Kingdom (Tennant & Gosling, 2015).

Table 1.7. Global percentage and figures share of palm oil production for 2023/24.

Note: Adapted from USDA, 2024.

Market	Global Production (%)	Total Production 2024/25 (Metric Tonnes)
Indonesia	**58%**	**46,000,000**
Malaysia	**25%**	**19,400,000**
Thailand	4%	3,300,000
Colombia	2%	1,900,000

Nigeria	2%	1,500,000
Guatemala	1%	990,000
Papua New Guinea	1%	830,000
Cote d'Ivoire	0.76%	600,000
Honduras	0.76%	600,000
Brazil	0.75%	595,000

Use of palm oils in food/food processing and preservation has several distinct advantages that contribute to its widespread use in the recent times. Palm oil has a melting point of 35°C, meaning it is semi-solid at room temperature, its high saturated fat content reduces susceptibility to oxidation and increases resistance to rancidity. Palm oil is also stable at high temperatures, meaning it can be used in frying without spoiling. From the crop husbandry perspective, palm trees are evergreen and perennial and that gives fruit round the year. Perhaps most importantly it has a very low cost of production, being much less than any other vegetable or animal fat, leading to no economic-based reason for a replacement; high efficiency per land mass (provides significantly more oil/yield per amount of land used). One hectare of land produces 1.9 - 4.8 tonnes of palm oil which is significantly higher than any vegetable oil crop (Figure 1.18). Palm oil contributes 36% of worldwide oil exports while uses only 8.6% of total land (used to produce all vegetable oils); in comparison to second-place soybean oil, needing 39% of total land to provide 25.5% of production (Our World in Data, 2024).

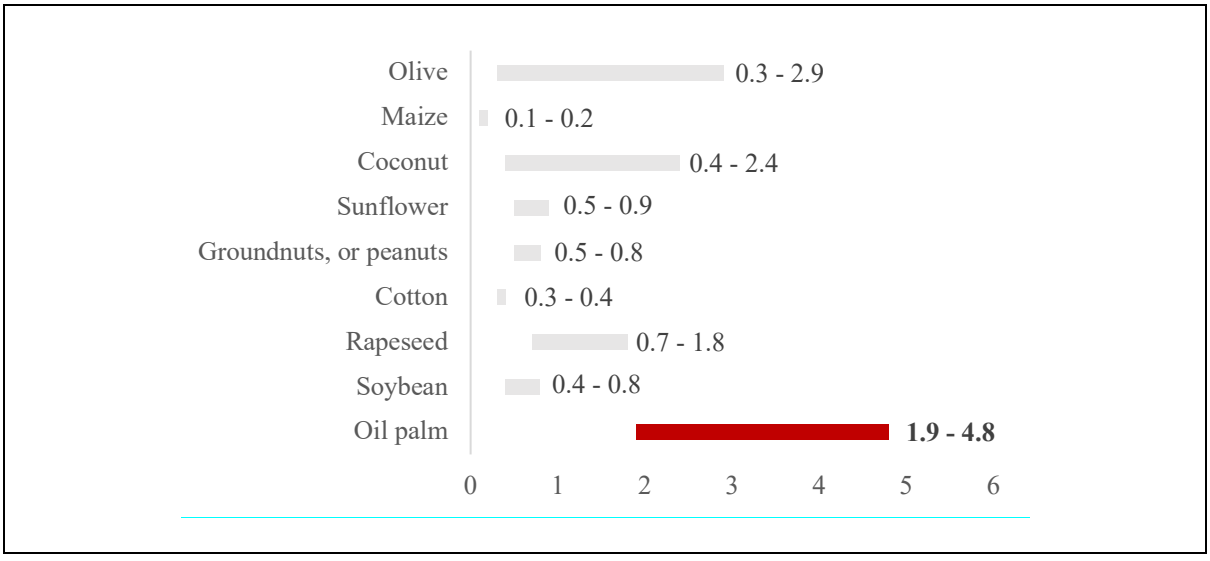


Figure 1.18. The range of yield (t/ha per year) of various edible oils.

Note: Adapted from Meijaard et al., 2020.

Palm oil comes in various forms that is based on the type of uses. Of those, red or 'crude' palm oil, made from the pulp of the palm fruit, is the most natural state which is one of the richest natural sources of vitamin E, and vitamin A (carotenoids) (Oguntibeju et al., 2009). Red palm oil is often used as a cooking oil in African and Southeast Asian countries (Rice & Burns, 2010). During industrial processing, crude palm oil is refined, bleached, and deodorised, and generally colourless, odourless, and tasteless, to use in a multitude of food products (Kim et al., 1997; Aryana et al., 2003). Unfortunately, this process (high heat, oxidation) removes/destroys many of the beneficial nutrients such as vitamin E (tocopherols, tocotrienols) found in crude/red palm oil (Gharby, 2022). In addition, certain bleaching methods can reportedly reduce carotene content by 97% (Almeida et al., 2024). Further refinement of palm oil such as fractionation, a process in which the solid and liquid phases are separated in order to maximise characteristics, functionality, and monetary viability, is also common. The liquid fraction, palm olein, is used in cooking, frying or salad oils (Ismail, 2005); whereas the solid fraction, palm stearin, is typically used as a shortening in bakery products or margarine (Subroto & Nurannisa, 2020). Both palm olein and palm stearin have their own fatty acid profile, containing approximately 40%, and 54% palmitic acid, respectively (Pande et al., 2012).

Palm kernel oil, derived from the seed of the palm fruit, is used in both edible and non-edible products such as commercial cooking oil, confectionary products, as well as making soaps (Young, 1983). Palm kernel olein is a refined, bleached, and deodorised fraction of palm kernel oil; high lauric acid content (approximately 48%) allows for a sturdy liquid surface, used in products such as ice cream coating (Liew et al., 2001). Palm kernel stearin and hydrogenated palm kernel oil are often blended together to create cocoa butter substitutes (Hassim & Dian, 2017). These various stages and outcomes of palm oil processing all stem from the oil palm tree which is presented in Figure 1.19.

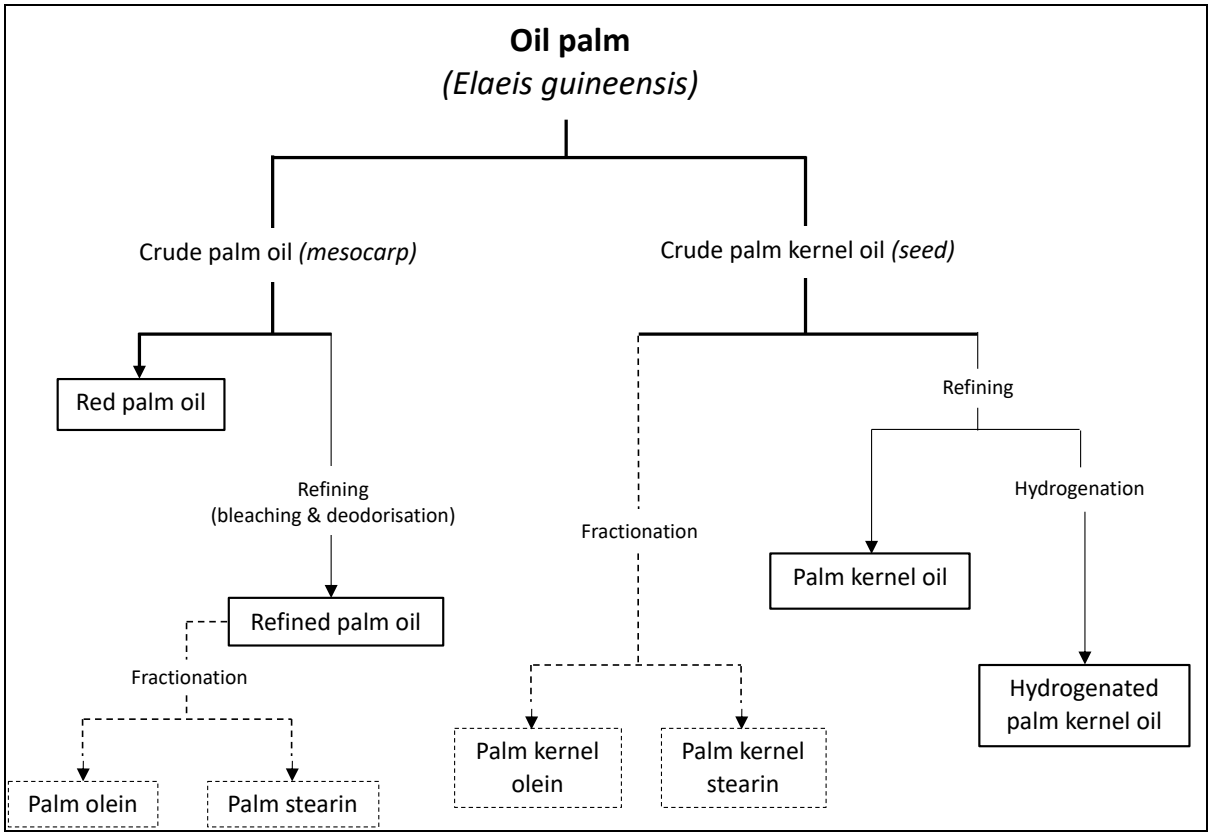


Figure 1.19. The creation of palm oil derivatives for human use.

Note: Adapted from Yılmaz & Ağagündüz, 2022.

Palm oil is prevalent in a range of edible and non-edible foods used for human consumption, with around 50% of all products containing some derivatives of palm oil (Yue et al., 2020). Despite this, the consumption of palm oil particularly in the UK is not well-documented. The data available are mostly represented the extent of import and market potential of palm oil with limited information on the consumption at the individual level and the trend of palm oil use in the processed food over the last few decades. Perhaps the most accurate investigation to date within the UK, reported the average dietary intake of palmitic acid (rather than palm oil) is 13.5 ± 5.7g per day; with the main sources being cereals and cereal products (mostly desserts, cakes and pastries), milk and products (including cheese), and meat products, followed by fat spreads and vegetables (Figure 1.20). The leading four food categories are likely high in palmitic acid due to their individual food items being either processed food products containing palm oil, or meat products such as beef or pork, which both contain around 25% palmitic acid content (Brody, 1998; Wood et al., 2004). It is important to note that palm oil contains palmitic acid as the major fatty acid (~44% palmitic acid content), non-palm oil derived sources such as animal origin foods also contain high content of palm oil. Hence, the estimated palmitic acid

consumption by the British household represents consumption of palmitic acid from either or both sources.

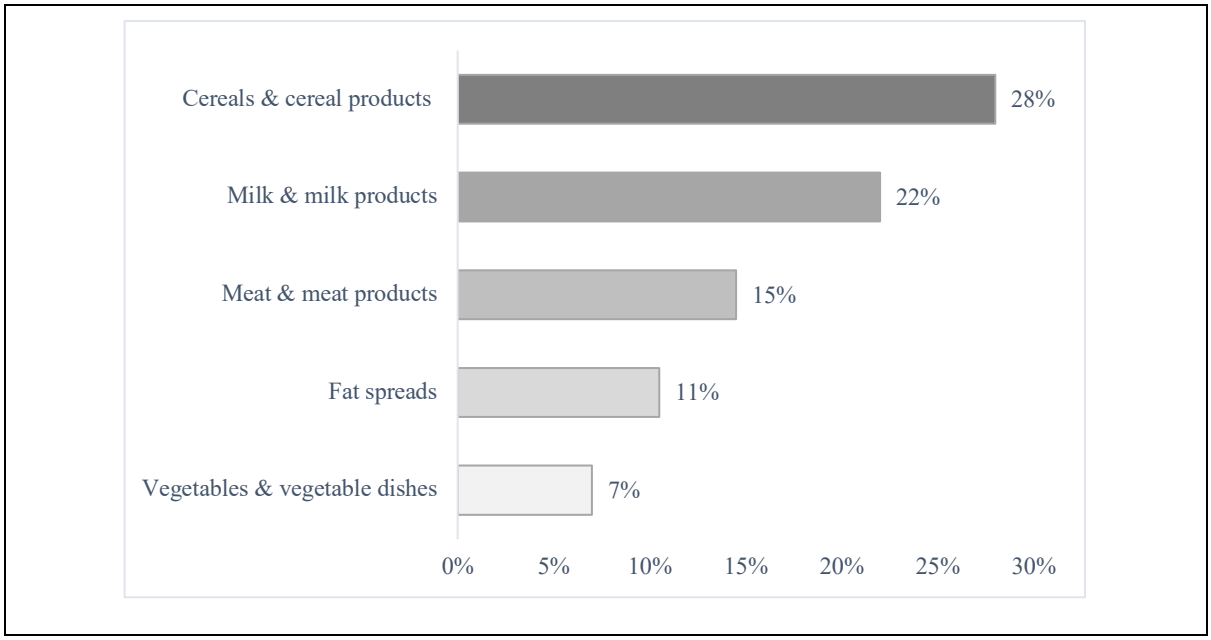


Figure 1.20. Content of palmitic acid (16:0) in common food groups consumed in the UK.
Note: Adapted from Kelly et al., 2022.

Compared to other edible oils, palm oil is highly economical, provides superior stability, organoleptic quality, and extended shelf-life of the processed food products (Singh, 2014) all of which contributed to its growing prevalence in ultra-processed foods. Industrialised processed foods in general are not a public health concern, however ultra-processed foods, typically refined from whole foods such as hydrogenated oils, fats, flours and starches - fabricate food products consisting of little-to-no nutritional value (Monteiro et al., 2013), in addition to much higher levels of sugar, salt, fat and artificial 'flavour-enhancing' additives (Luiten et al., 2016). These products include many prevalent food items such as crisps, cakes, chocolate, cereals, biscuits, and bread. Animal-derived products that are also 'ultra-processed' include yoghurts, ice cream, bacon, sausages, chicken nuggets and canned meats. Ultra-processed foods have been rapidly increasing in high and middle-income countries since the 1980's (Elizabeth et al., 2020; Marrón-Ponce et al., 2018; Monteiro et al., 2010). In the United Kingdom, the consumption of palm oil is predominantly contributed by the use of ultra-processed foods because palm oil is not extensively used as a cooking oil in the British household. In the UK, 56.8% of the daily energy comes from the consumption of ultra-

processed foods (Rauber et al., 2019). Recent studies report that greater exposure to ultra-processed foods was associated with a higher risk of adverse health effects, including certain cancers, cardiometabolic disorders, common mental disorders, and incidences of mortality (Lane et al., 2024; Wang et al., 2022).

1.5.4 Palm oil, palmitic acid, and metabolic health

Palmitic acid (PA), a long-chain saturated fatty acid, is a highly abundant within most diets and constitutes 20-30% of total fatty acids in the human body (Carta et al., 2017). Palmitic acid is predominantly found in cell membrane phospholipids and adipose triacylglycerols. Palmitic acid is sourced from the diet, as well as produced endogenously via synthesis within the liver and adipose tissue (Annevelink et al., 2023). Malaysian palm oil contains approximately 50% saturated fatty acids and approximately 50% unsaturated fatty acids. Palmitic acid, at approximately 44% of total fatty acids, is the major fatty acid present in palm oil (Table 1.8). Other dietary sources of palmitic acid include edible oils from vegetables and seeds, as well as meat and dairy (animal lipid) food items (Table 1.8).

Table 1.8. Typical fatty acid composition of Malaysian palm oil.

Note: Adapted from Kifli, 1981.

Systematic name	Common name	FA chain length	Mean (%)	Range observed
Dodecanoic acid	Lauric acid	12:0	0.1	0.0 - 0.4
Tetradecanoic acid	Myristic acid	14:0	1.0	0.6 - 1.7
Hexadecanoic acid	**Palmitic acid**	**16:0**	**43.5**	**41.1 - 47.0**
Octadecanoic acid	Stearic acid	18:0	4.4	3.7 - 5.6
9-Octadecenoic acid	Oleic acid	18:1	39.9	38.2 - 41.8
9,12-Octadecadienoic acid	Linoleic acid	18:2	10.3	6.6 - 11.9
9,12,15-Octadecatrienoic acid	α-Linolenic acid	18:3	0.3	0.0 - 0.8

Table 1.9. Palmitic acid content of common dietary fat sources.

Source	[a]PA range observed (%)	Author, year
Palm Oil	**39.2 - 45.8**	Kifli, 1981
Olive oil	7.5 - 20.1	Boskou et al., 2006
Sunflower oil	5.0 - 7.6	Rosa et al., 2009
Soybean oil	10.4 - 10.5	Bobo et al., 2021
Sesame oil	8.2 - 8.4	

[1]Rapeseed oil	3.0 - 4.2	
Flaxseed oil	5.0 - 5.3	
[2]Beef	23.2 - 26.8	Enser et al., 1996
[2]Pork	21.7 - 24.7	
[2]Lamb	20.7 - 23.8	
[3]Milk	22.0 - 24.0	Djordjevic et al., 2019
[4]Cheese	28.7 - 30.7	Paszczyk & Łuczyńska, 2020
[5]Butter	36.9 - 74.7	Anwar et al., 2006
[5]Margarine	24.1 - 38.5	
Lard	22.7 - 26.5	Nizar et al., 2013.

[1]Two types of Rapeseed/Canola oil.

[2]Sourced from animal muscle.

[3]Three sources: cow, sheep, and goat.

[4]Cheddar (cow) cheese.

[5]Various sample values merged.

[a]PA: Palmitic acid. In some cases, range values estimated based on standard deviation(s).

Palm kernel oil is extracted from the kernel (seed) of the fruit, while palm oil is derived from the mesocarp (flesh). Interestingly, palm kernel oil possesses a markedly different fatty acid composition to that of palm oil. Although containing higher saturated fat than palm oil (approximately 82%), palm kernel oil contains predominantly lauric acid, with low contents of palmitic acid (Table 1.10).

Table 1.10. Full fatty acid composition of palm kernel oil.

Note: Adapted from Edem, 2002.

Fatty acid	**Fatty acid content (%)**
Caproic acid (6:0)	0.2
Caprylic acid (8.0)	3.3
Capric acid (10:0)	3.5
Lauric acid (12:0)	**47.8**
Myristic acid (14:0)	16.3
Palmitic acid (16:0)	**8.5**
Stearic acid (18:0)	2.4
Oleic acid (18:1)	15.4
Linoleic acid (18:2)	2.4
Linolenic acid (18:3)	-
Arachidic acid (20:0)	0.1

Several studies have indicated that palmitic acid causes inflammation, insulin resistance, and oxidative stress (Saraswathi et al., 2021). An overabundance of long-chain saturated fatty acids (LCSFAs), including palmitic acid, is widely known to promote the low-grade chronic inflammatory disease, obesity (Shin, 2022), as well as dyslipidaemia and gut dysbiosis (Akieda-Asai et al., 2019; Alnahdi et al., 2019; Li et al., 2019). In obese individuals, blood concentration of palmitic acid is elevated, leading to inflammatory responses (Korbecki & Bajdak-Rusinek., 2019) and insulin resistance (Liu et al., 2015). There is convincing, contemporary evidence that palmitic acid has strong associations with cardiovascular disease (He et al., 2025). However, palmitic acid's links to cardiovascular disease could be mostly due it being a saturated fat, rather than the individual effects of the acid itself, promoting the uptake of LDL-C and dyslipidaemia (Briggs et al., 2017). Although typically occurring in the presence of a high-carbohydrate diet, high-fat diets can also stimulate de novo lipogenesis (Strable & Ntambi, 2010), a process occurring in the liver and adipose tissue where excess carbohydrates, lipids and proteins are converted into fatty acids for energy storage (Ameer et al., 2014). The initial outcome of de novo lipogenesis is palmitic acid (Lee et al., 2020). Therefore, over-abundance of fat within the diet may, expectedly, lead to an over-accumulation of palmitic acid; inducing insulin resistance, hepatic inflammation and dyslipidaemia (Khan et al., 2023). Studies have reported that PUFAs downregulate de novo lipogenesis induced inflammation, as well as limit excessive fat (lipid) storage, suggesting a PA/PUFA ratio is advocated (Crescenzo et al., 2017).

Palmitic acid reportedly induces systemic inflammation (adipose tissue, liver, muscle, kidney, hypothalamus); acting as an agonist to toll-like receptors 2 (TLR2) and 4 (TLR4) (Huang et al., 2012; Lee et al., 2001; Lee et al., 2011; Sindhu et al., 2016; Wang et al., 2012[a]; Wang et al., 2012[b]), providing stimulation of proinflammatory cytokine expression and promoting NFκB/KLF7 signalling pathway activation (Suganami et al., 2007; Qiu et al., 2022). Pro-inflammatory cytokines, tumour necrosis factor alpha (TNF-α), IL-1β and IL-6 are widely reported to induce insulin resistance (Moller, 2000; Andersen & Pederson, 2008; Tilg & Moschen, 2008; Wieser et al., 2013). Insulin resistance, or reduced insulin sensitivity/decreased insulin secretion, reportedly induced by the SFA palmitic acid (Lankinen et al., 2015), can be prevented by the MUFA oleic acid; suggesting that a ratio between the two acids is of great importance to diabetes risk (Palomer et al., 2018). Palmitic acid exerts inflammatory effects by inducing expression of C-reactive protein (elevated levels associated

with cardiovascular disease) (Sproston & Ashworth, 2018), nitric oxide synthase (large amounts are produced at sites of inflammation) (Evans, 1995), and tumour necrosis factor-α (TNFα), elevated in arthritis, diabetes, Crohn's diseases and some cancers (De et al., 2002; Wu et al., 2014).

The 'lipid hypothesis', which proposes that elevated plasma cholesterol, or hypercholesterolemia (specifically LDL-C) is the chief marker in atherosclerosis, coronary heart disease and cardiovascular disease (Ference et al., 2017), has been a well-accepted theorem for over 100 years (Steinberg, 2013). Palmitic acid accumulation in liver cells (hepatocytes) has been found to induce hepatic toxicity (Edem, 2002), in addition to induce production of IL-8, contributing to inflammation and injury (Joshi-Barve et al., 2007). Palmitic acid accumulation has also been reported to have associations with certain cancers, particularly breast and prostate. Significant associations between palmitic acid levels and risks of breast cancer were reported via meta-analysis (Saadation-Elahi et al., 2004). Studies in the last two decades further substantiate these findings, reporting significant associations between levels of PA and breast cancer risk (Bassett et al., 2013; Chajès et al., 2020).

Increased levels, or excessive intracellular accumulation of palmitic acid, reportedly causes lipotoxicity, increased production of reactive oxygen species (ROS) endoplasmic reticulum (ER) stress, in addition to activation the neuroinflammation signalling pathway (Fatima et al., 2019; Vesga-Jiménez et al., 2022; Zhou & Chang, 2023). Palmitic acid has been reported to have deleterious effects on the gut microbiome: including chronic inflammation (Ghezzal et al., 2020), and damage to intestinal epithelial barrier integrity and permeability (Gori et al., 2020). Palmitic acid induces the initiation of intestinal inflammatory cytokine production; however, the underlying mechanisms are not well understood (Fang et al., 2022). Palmitic acid also plays a role in conditions such as irritable bowel syndrome (IBS) (Ma et al., 2019) Crohn's disease and ulcerative colitis (Wei et al., 2024). However, these effects appear largely due to high saturated fatty acid content (de Wit et al., 2012).

Palmitic acid was found to be highly pro-inflammatory in nature due to its ability to activate the NFκB pathway and has been widely used in the laboratory experiments as an inducer of inflammatory response. Concurrent with TLR4 activation, palmitate cell uptake by fatty acid transporter CD36 induces inflammatory chemokine (IL-6, TNF-α, IL-1β) production (Figure 1.21a). Upregulation of the expression of interleukin cytokines at the transcriptional level occurs while activating the inhibitor of nuclear factor kappa B (IκBα), a negative regulator of

the NfkB (Sergi et al., 2021). Palmitate-induced upregulation of pro-inflammatory cytokines activates the innate immune response; contributing to the pathogenesis of various chronic diseases (Figure 1.21b).

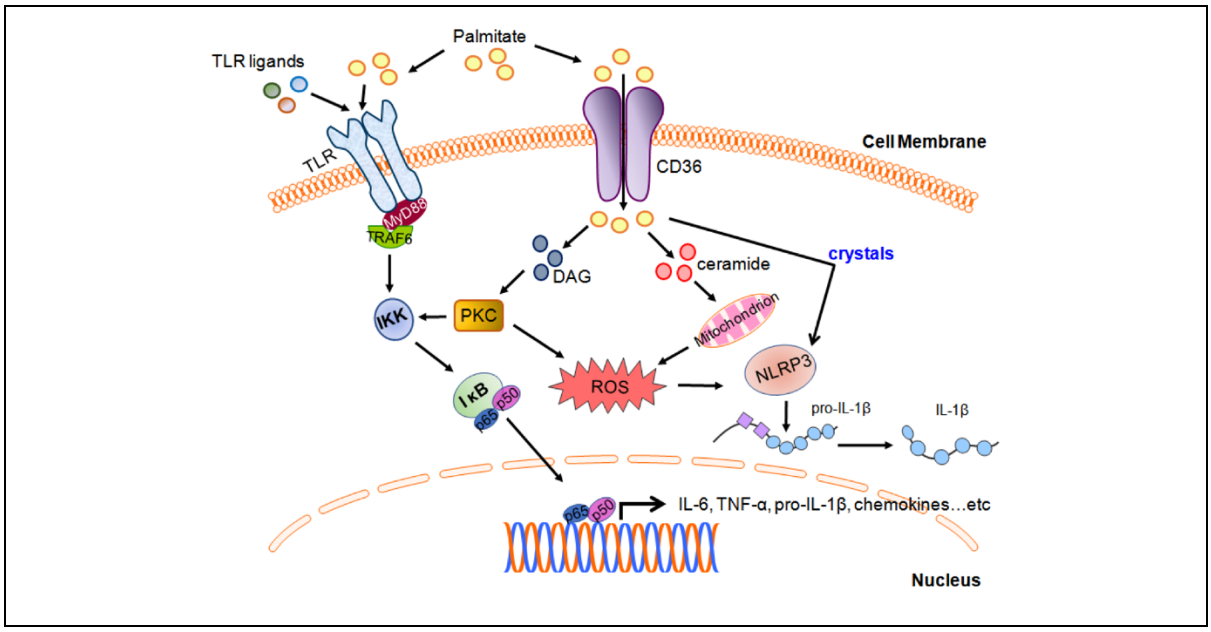


Figure 1.21a. Palmitate-mediated pathways in the immune response.

Note: From Tzeng et al., 2019.

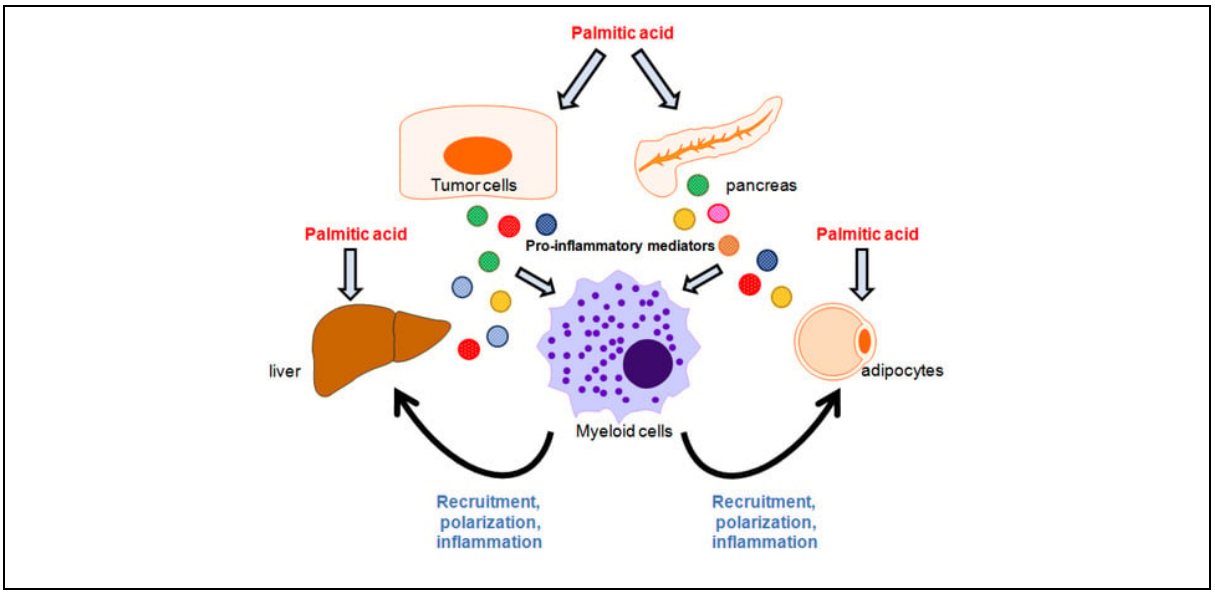


Figure 1.21b. Innate immune response amid palmitic-induced inflammation.

Note: From Tzeng et al., 2019.

Similarly, palmitic acid has well-known role as an inducer of oxidative stress through affecting the Kelch-like ECH-associated protein 1 (KEAP1) - transcription factor NF-E2 p45-related factor 2 (NRF2) pathway which is otherwise essential for the protection of cells from oxidative stress (Baird & Dinkova-Kostova, 2011). Palmitic acid reportedly induces KEAP1 degradation and downregulation, activating the NRF2 target gene transcription, although intensity appears to be adapter protein p62 dependant (Park et al., 2015).

Although there has been convincing evidence over the years, recent findings suggest associations between palm oil, palmitic acid and cardiovascular disease or certain cancers to be beneficial, or at the least, unclear (Fattore & Fanelli, 2013; Ismail et al., 2018; Odia et al., 2015). Palmitic acid has been investigated for its potential therapeutic applications in cancer: reportedly inducing the death of breast cancer cells (Zafaryab et al., 2019). Various anti-tumour activities of palmitic acid were also reported, proposing that palmitic acid significantly suppressed prostate cancer cell growth, through suppression of inflammatory pathway(s) (Zhu et al., 2021). Although it has been proposed that there are therapeutic effects of palmitic acid on cancer cells, further exploration is believed to be necessary (Wang et al., 2023).

There is limited evidence to suggest that dietary palmitic acid (alone) from different sources have contradictory or even dissimilar effects on metabolic health. However, factors such as distinct fatty acid profile (of its source), or triacylglycerol structure, should be considered as more than probable to influence the effects of palmitic acid in numerous ways. Palm oil for example, whilst containing around 44% palmitic acid; also contains approximately 40% oleic acid (MUFA), found to have therapeutic effects on inflammation, immunity, and cardiovascular disease risk factors (Karacor & Cam, 2015). Therefore, upon consumption of palm oil, it is likely that the harmful bioactivity of palmitic acid is attenuated by the oleic acid. The counteractive effect of the palmitic acid present in palm oil can also be mediated by the presence of stearic acid, another common fatty acid present in palm oil (4.3%) that has been reported to present no detrimental effects on cardiovascular health (Crupkin & Zambelli et al., 2008). This is the case for many consumable food ingredients where synergistic and counter effective interactions are common phenomena. This phenomenon further justifies rationale behind consumption of whole versus the ultra-processed foods/food ingredients.

Stereospecific numbering (sn) is used to identify the three fatty acids contained within a triglyceride, respective of their position on the glycerol backbone. Both palmitic acid and

stearic acid are typically esterified at the sn-1 and sn-3 positions of the glycerol backbone, differing from the other key fatty acids such as oleic, lauric and myristic (Figure 1.22). Palmitic acid in foods is rarely found at the middle (sn-2) position of the glycerol backbone. While present at the sn-2 position in butter and lard (due to cow's milk), this stereospecificity is not present amongst all common vegetable oils (Table 1.11).

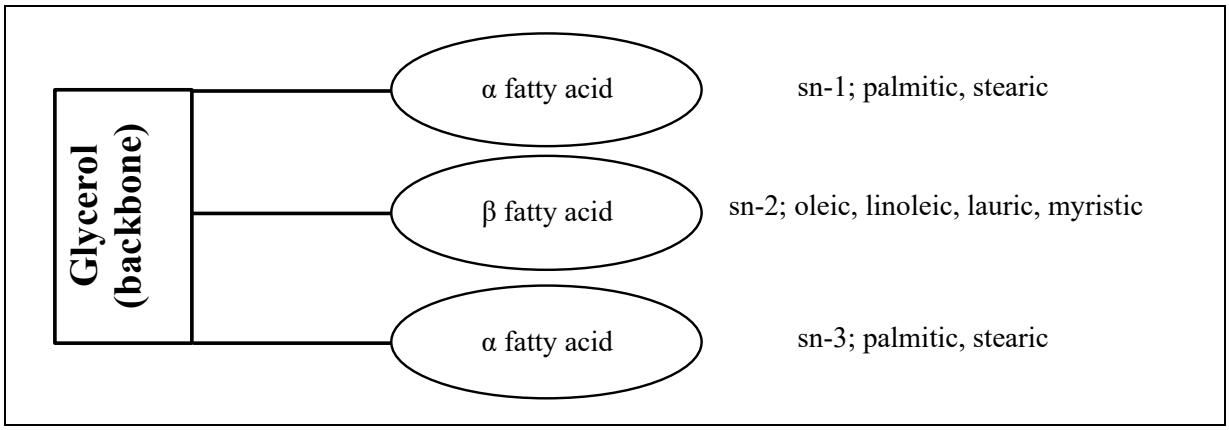


Figure 1.22. Sn positioning within palm oil triacylglycerols.

Note: Adapted from Yılmaz & Ağagündüz, 2022.

Table 1.11. Stereospecificity of dominant triacylglycerol species in edible oils and natural fats.

Note: Adapted from Karupaiah & Sundram, 2007.

Vegetable oil	**Triacylglycerol (TAG) species**		
Palm oil	**POP**	**POO**	**POL**
Almond oil	OOO	OLO	OLL
Canola oil	OOO	LOO	OOLn
Cocoa butter	POS	SOS	POP
Coconut oil	DDD	CDD	CDM
Corn oil	LLL	LOL	LLP
Cottonseed oil	PLL	POL	LLL
Olive oil	OOO	OOP	OLO
Palm kernel oil	DDD	MOD	ODO
Peanut oil	OOL	POL	OLL
Safflower oil	LLL	LLO	LLP
Soybean oil	LLL	LLO	LLP

	Sunflower oil	LLL	OLL	LOO
	Walnut oil	LLL	OLL	PLL
	Animal fat	**Triacylglycerol (TAG) species**		
	Butter	P**P**B	P**P**C	POP
	Egg fat	POO	PLO	POS
	Lard	S**P**O	O**P**L	O**P**O
	Tallow	POO	POP	POS
	P = palmitic; O = oleic; S = stearic; D = dodecanoic or lauric; C = capric; M = myristic; L = linoleic; Ln = linolenic; B = butyric.			

Palmitic acid present in human milk, however, does have this unique arrangement (Innis, 1994). Breast milk contains 18-24% of the total fatty acid as palmitic acid depending on the ethnicity and dietary backgrounds (Yuhas et al., 2006). Around 70% of palmitic acid is esterified to the 'middle' position of triacylglycerols, referred to as sn-2 palmitic acid or beta/β-palmitate (Giuffrida et al., 2018), with some variations again caused by the ethnicity and diet (Lopez-Lopez et al., 2002). Fatty acids at the sn-1 and sn-3 positions typically detach from the triacylglycerol, resulting in the sn-2 fatty acid becoming the predominant attached substance. Therefore, the sn-2/β-palmitic acid in breast milk post-consumption is subsequently absorbed as a monoglyceride (MAG), rather than a free fatty acid, providing several favourable effects to infants, including modulation and proliferation of beneficial gut microbiota via fast absorption (Shoji et al., 2024), improved fatty acid (including 16:0) and mineral (calcium) absorption (Bar-Yoseph et al., 2013). Such an increase in β-palmitic acid absorption alters gut microbiota through a difference in the flow of substrates and decreases gut inflammation (Miles & Calder, 2017). Improved calcium absorption is due to palmitic acid remaining attached and avoiding coupling with the calcium (Tomarelli et al., 1968).

Sn-2/β-palmitic acid is generally added to infant formula to achieve similar effects to that of maternal breast milk (Havlicekova et al., 2015), produced using an enzymatic process (beta oxidation), bonding palmitic acid to the middle position of the glycerol backbone (Carnielli et al., 1996) to mimic natural breast milk. This process is named interesterification (IE), in which the position of fatty acids is manipulated or rearranged within the triglyceride, to form new triacylglycerol molecules. Examples of these structured triglycerides include palmitic-linoleic-oleic (PLO), linoleic-palmitic-oleic (LPO) and oleic-linoleic-palmitic (OLP) molecules, each

including palmitic acid in a different stereospecific numbering (SN) position on the glycerol backbone (Figure 1.24).

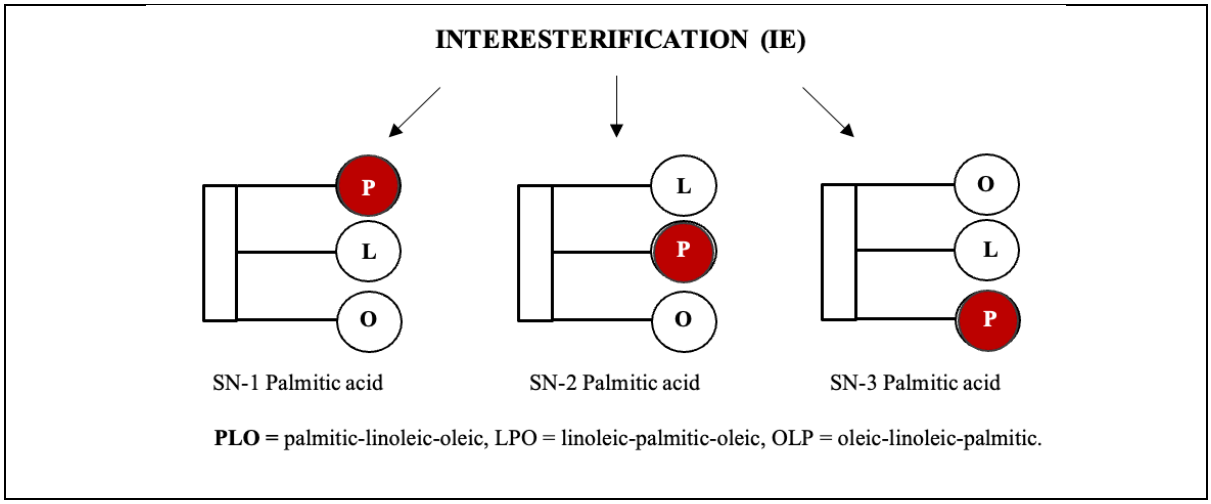


Figure 1.23. Changes in palmitic acid stereospecific numbering post-interesterification.

Note: Adapted from Karupaiah & Sundram, 2007.

1.6 Research Gaps/Limitations

The limitations and knowledge gaps observed within the Review of Literature raise many questions:

1. **What is the general consensus on palm oil and palmitic acid's health effects within contemporary available literature?**
2. **Are consumers (United Kingdom) aware of palm oil and palmitic acid?**
    a. Do they understand their prevalence, their health effects?
3. **How prevalent (exactly) are palm oil, palmitic acid in commonly consumed foods?**
    a. Which food groups contain higher content(s) of palmitic acid?
    b. Which dietary habit is most vulnerable to chronic palmitic acid consumption?
4. **Does palm oil and palmitic acid intake level have significant differences/effects on individual risk factors for chronic disease?**
    a. What should be the suggested (if any), dietary recommendations (i.e., recommended daily intake) of palm oil or palmitic acid?
    b. Are consumption levels of palm oil or palmitic acid different per dietary habit (i.e., plant-based/vegan versus animal-based diets)?
    c. Does the source (animal, plant) of palm oil or palmitic acid influence physiological processes, incidences of disease?
5. **What are the wider health implications?**
    a. Future research and clinical recommendations?

1.7 Conclusions

The prevalence of palm oil and palmitic acid within commonplace food items, together with ambiguous determinant health effects, in addition to the ever-growing consumption of ultra-processed foods, creates enough evidence to suggest the inevitability of a future metabolic health epidemic. This review of literature provides well-defined criteria to investigate palm oil and palmitic acid, particularly their significance and implications in a multitude of chronic disorders and disease across various dietary habits, with a focus on plant-based/vegan diets.

Chapter 2. Effects of palm oil, palmitic acid, and their derivatives on metabolic health: a systematic review

2.1 Chapter Overview

Palm oil (PO) contains approximately 50% saturated fatty acids (44% palmitic acid) and represents one of the most widely utilised vegetable oils in the world. Growing evidence suggests that palmitic acid may exacerbate metabolic inflammation associated with a range of chronic diseases. The widespread use of palm oil in the western diet; particularly its presence in processed food products, suggests that palmitic acid might be consumed at a potentially alarming rate. This review aims to evaluate the evidence on the role of palm oil and palmitic acid (16:0) on metabolic health and the gut microbiome. This systematic review was conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) framework. A comprehensive literature search was performed using the following databases: Scopus, Web of Science, Ovid and PubMed and Google Scholar. The search terms were limited to the title, abstract and keywords of the available literature and consisted of the following: “palmitic acid”, “palm oil”, “palmitate”, “palm kernel oil”, “palm stearin”, “palm olein”, “retinyl palmitate”, “retinol palmitate”, “vitamin a palmitate”, “dietary palm” and “dietary palmitic” - alongside - “metabolic health”, “gut health”, “gut microbiome” and “gut microbiota”. Studies were excluded based on various criteria: duplicated studies, studies considered unrelated, review papers. Quality assessment of studies was performed using various risk of bias tools (Cochrane RoB2, ROBINS-I, SYRCLE, OHAT). Risk of bias was noted for several studies, most interestingly due to conflicts of financial interest, or close relationships with various palm oil corporations or institutes, including the Palm Oil Research Institute of Malaysia, the Malaysian Palm Oil Council, and the Malaysian Palm Oil Board. A total of 132 studies are included in the final review. Based on this review of the available literature, the results conclude that the majority of studies provide evidence to suggest that palm oil, or more specifically palmitic acid intake, contributes significantly to various chronic diseases associated with metabolic syndrome, such as type 2 diabetes, cardiovascular disease(s), stroke, and nutrition-related cancers.

2.2 Introduction

Approximately 1749 kilotons (kt) of vegetable oils are consumed yearly in the United Kingdom (FEDIOL, 2024). Oils originating from vegetables/plants are generally perceived to be healthier than oils originating from animal sources, due to their lower saturated fatty acid content. Common edible vegetable oils used in UK households contain varying levels of saturated fat content, such as olive oil (~17%), coconut oil (~86%), sesame oil (~16%), sunflower oil (~11%) and rapeseed oil (~7%) (Chapter 4 of this thesis). These oils contain relatively low contents of saturated fatty acids, when compared to cooking alternatives such as animal-origin fats, including lard (~41%), beef tallow (~56%), butter (~68%) or hard margarine (~80%) (Kenneth, 2024; Zhou et al., 2024). In recent years, vegetable oils with a high saturated fat content such as coconut oil (~86%) and palm oil (~50%) have become prevalent in western countries. Palm oil (*Elias guineensis*) is one of the most widely used vegetable oils consisting of 57% of the global vegetable oil exports (Mba et al., 2015). In 2023-24, Indonesia and Malaysia account for 57% (44 million metric tonnes) and 26% (19.7 million metric tonnes) of global palm oil exports respectively (USDA Foreign Agricultural Service, 2024). Global consumption of palm oil has doubled over the past 15 years, with a per capita (individual) annual consumption of around 8 kg (Europarl, 2018). The widespread popularity of palm oil is most likely attributable to its lower price, oxidative stability, and extended shelf-life (Almeida, 2017). Palm oil has natural resilience against deterioration, due to its abundance of both carotenoids and tocopherols. This exceptional oxidative stability helps prevent processed food from becoming rancid (Falade et al., 2015; Tan et al., 2016). Red, or 'crude' palm oil, is a term to describe palm oil at its most natural, minimally processed form. Red palm oil contains one of the largest natural sources of vitamin E - the largest source amongst vegetable oils (Chow, 2007). Red palm oil also contains phenolic compounds and is a natural source of tocotrienol; typically considered to have overall positive effects on human health (Ibrahim et al., 2020; Mukherjee & Mitra, 2009; Zainal et al., 2019). Red palm oil is most typically used as a cooking oil in tropical regions of Africa and Asia, whereas in the UK refined palm oil is predominantly used as a substitute for animal-based saturated oil(s) in a wide range of processed food products.

Palm oil contains approximately 50% saturated fatty acids with 44% as palmitic acid (C16:0), 5% stearic acid (C18:0), both long chain fatty acids (LCFAs) and trace amounts (around 1%) of myristic acid (C14:0), a medium chain fatty acid (MCFA) (Montoya et al., 2014). Palmitic acid (C16:0), or *hexadecanoic* acid (not to be confused with *hexedecenioc*, or palmitoleic acid;

C16:1) is one of the most abundant saturated fatty acids of plant and animal origin widely found in the Western diet (Gunstone & Harwood, 2007). It is generally known that dietary saturated fats adversely affect human health; specifically, the lipid profile, including LDL cholesterol that exacerbate the risk of developing diseases within the metabolic syndrome (MetS) spectrum (Harrison et al., 2020). Hence, it is reasonable to suspect that widespread use of palm oil may exacerbate metabolic inflammation and insulin resistance associated with a range of chronic or noncommunicable diseases (NCDs) (Amine at al., 2021; Straczkowski et al., 2004).

Dietary palm oil may also affect gut microbiology and thus contribute to MetS (Ghezzal et al., 2020). The gut microbiota plays a pivotal role in regulating metabolic, endocrine, and immune functions (Silva et al., 2020). Gut microorganisms, particularly those affecting the inflammatory immune system within the epithelial layer, play a role in a wide range of human diseases (Scher et al., 2015). Although dietary polysaccharides are crucial in maintaining a gut microbial ecology that influences metabolic health, dietary fats/lipids have been shown to alter the composition of the human gut microbiome (Spady et al., 1993; Stamler et al., 2000). Quality of fats is also important; for example, saturated fats negatively affect the composition of the gut microbiome (Menni et al., 2015), leading to dysbiosis associated with many intestinal and extra-intestinal disorders (Carding et al., 2015) including metabolic dysfunction-associated steatotic liver disease (MASLD) (Safari & Gérard, 2019).

The fat present in the modern and westernized diet mainly originates from edible oils; derived from vegetables or crops such as seeds, nuts, fruits, grains, and legumes. Vegetable oils are in the vast majority of packaged and processed food items (Hawkes, 2010). Typically considered healthy fats due to their relatively low saturated fat and higher monounsaturated and polyunsaturated fat content (Trevisan et al., 1990). Monounsaturated fatty acids can increase HDL-C levels, whilst lowering LDL-C, reducing risks of MetS and CVD, respectively (Gillingham et al., 2011). Polyunsaturated fats containing essential fatty acids such omega-3 and omega-6 are believed to have differing effects, the former assisting in reduction of inflammation whereas the latter inclines to promote it (Khanapure et al., 2007).

However, not all studies within the literature report these negative effects. Palmitic acid has been shown as an important saturated fatty acid required for cell membrane integrity and transportation of lipids (German, 2011), with no incremental risk of CVD (Odia et al., 2015).

There are also many derivatives of palm oil and palmitic acid, such as palm olein, palm stearin, crude/red palm oil, palmitate, and sn-2 palmitate/β-palmitic acid.

The many derivatives of palm oil and palmitic acid, alongside conflicting, or unclear reports of their various health effects within the literature, could conceivably lead to misconceptions. More specifically, confusion within public health knowledge concerning a definitive answer. Additionally, the perceived unfavourable health effects of palm oil and palmitic acid may be a consequence of their intrinsic abundance within Westernised diets, or simply palm oil's natural high saturated fat content, rather than health issues directly from palm oil itself. It is also difficult to discern whether outcomes (favourable or unfavourable), occur as acute or chronic effects, and the extent of their role within metabolic health status and chronic disease development. Lastly, palm oil or palmitic acid may be influenced by various other factors, such as supplementary dietary or endogenous vitamins/nutrients, as well as an individual(s) pre-disposition to disorder or disease. Therefore, this systematic review of available studies was conducted with the following aims and objectives:

1. To provide a comprehensive overview of the available evidence within the literature on palm oil, palmitic acid, and their derivatives' various metabolic health effects, as well as an exploration of health outcomes within the gut microbiome.
2. To discuss the various implications of these health effects and explore recommendations for clinical applications and areas of study.

2.3 Methodology

2.3.1 Validity statement

This review was conducted in accordance with the guidelines set by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) framework (Page et al., 2021; Appendices 2.1, 2.2), and addresses the identification, screening, data collection, and appraisal of relevant research studies. The protocol was registered online (REF: CRD42022298007) in accordance with the National Institute for Health and Care Research, or PROSPERO (University of York, 2024).

2.3.2 Literature sources and search criteria

A literature search was performed in the most widely used online databases including Scopus, Web of Science, Ovid, PubMed, and Google Scholar. A total of two searches were conducted, at different time points: November 2022, and January 2024. All searches included article, conference paper, editorial, letter, note, business article, review, systematic review, meta-analysis, and short survey document and book chapters, both published and unpublished (grey literature) from the earliest available dated publication to 2023 (year-end). While the search mainly focused on mapping the existing literature on the effects of dietary palm oil/palmitic acid on human health, studies conducted on animals, as well as mechanistic studies in cell culture were also included. The majority of animal studies were that of a rodent model, the preferred method for trials investigating effects similar to that of human disease (Bryda, 2013). The search terms were limited to the title, abstract and keywords of the available literature and consisted of the following: “palm”, “palm oil”, “oil palm”, “palmitic acid”, “palmitate”, “16:0”, “palm kernel oil”, “palm stearin”, “palm olein”, “palmitate”, “retinyl palmitate”, “retinol palmitate”, “vitamin a palmitate”, “dietary palm”, “dietary palmitic” - alongside - “metabolic health”, “chronic”, “disease”, “gut health”, “microbiome” and “microbiota”. In order to identify additional relevant studies potentially missed within the initial searches, reference lists of identified sources were subsequently analysed.

2.3.3 Data extraction

All relevant available information (including title, abstract, keywords, author, year of publication, document type and journal) was exported and compiled within a Microsoft Excel spreadsheet.

2.3.4 Eligibility assessment

All literature included within the preliminary data extraction were subject to a thorough eligibility assessment. The article title, abstract and keywords were evaluated to ensure their appropriateness and relevance to palm oil, palmitic acid or their derivatives’ effects on metabolic health and/or related subjects. During this process: dismissal of unrelated papers, filtration of duplicate records and inaccessible full-text articles were identified and subsequently removed. Any paper possessing ambiguous relevance (not explicitly apparent from the title, abstract or keywords alone), were passed through to the next screening stage. All review papers were also dismissed at this stage. To ensure the quality of the eligibility

assessment, the author analysed the content of the full texts to determine their validity and relevance. A total of 213 studies passed the eligibility assessment phase.

2.3.5 Risk of bias assessment

Appraisal of the available evidence risks of bias was performed using various assessments, depending predominantly on study type and population. For human intervention studies: randomised controlled trials were assessed using the revised Cochrane risk of bias tool (RoB 2), non-randomised trials were assessed using the Risk of Bias in Non-randomised Studies - of Interventions (ROBINS-I). Both highly acclaimed methods in assessing risk of bias in randomised, and non-randomised intervention studies, respectively (Sterne et al., 2019; Sterne et al., 2016). For animal trials: studies were assessed using the Systematic Review Centre for Laboratory animal Experimentation (SYRCLE) risk of bias tool (a modified version of the RoB2 tool) (Hoojimans et al., 2014). For in vitro trials: studies were assessed using the United States National Toxicology Program Office of Health Assessment and Translation risk of bias tool (OHAT). Various domains of bias were assessed, depending on the tool used (Table 2.1) Risk(s) of bias across these domains were interpreted subjectively by the author of this review, using a scale of various ratings (again, tool-dependant), including: 'High', 'Some concerns', 'Low' and 'No information' (RoB2), 'Serious', 'Moderate', 'Low' and 'No information' (ROBINS-I), 'High', 'Unclear' and 'Low' (SYRCLE), 'Definitely high', 'Probably high', 'Probably low' and 'Definitely low' (OHAT). Overall risk of bias (per study) was then determined based on totality these ratings. Microsoft Excel was used to perform all risk of bias assessments. Lastly, visualisation (traffic-light plot) of assessment outcomes were generated using the 'Risk-of-bias VISualization' (robvis) tool (McGuinness et al., 2020).

Table 2.1. Domain(s) of biases evaluated within each risk assessment tool.

Biases analysed throughout assessment of randomised human trials (RoB2)	**Biases analysed throughout assessment of non-randomised human trials (ROBINS-I)**
D1: Bias arising from the randomisation process. D2: Bias in deviations from intended intervention. D3: Bias due to missing outcome data. D4: Bias in measurement of the outcome. D5: Bias in selection of the reported result.	D1: Bias due to confounding. D2: Bias due to selection of participants. D3: Bias in classification of interventions. D4: Bias in deviations from intended intervention. D5: Bias due to missing data. D6: Bias in measurement of outcomes. D7: Bias in selection of the reported results.
Biases analysed throughout assessment of animal trials (SYRCLE)	**Biases analysed throughout assessment of in vitro trials (NTP)**
D1: Bias due to sequence generation. D2: Bias due to baseline characteristics. D3: Bias due to allocation concealment. D4: Biases in random housing. D5: Bias in blinding of caregivers. D6: Bias in random outcome assessment. D7: Bias in blinding of outcome assessment. D8: Bias due to incomplete outcome data. D9: Bias in selection of the reported results. D10: Other biases.	D1: Bias arising from the selection process. D2: Bias due to confounding. D3: Bias in blinding during study. D4: Bias in measurement of the outcome. D5: Bias due to incomplete outcome data. D6: Bias in selection of the reported results. D7: Other biases.

2.3.6 Sorting of literature

For the organisation of literature, studies were divided into three categories; studies reported on humans (infants, adults), animal trials, and in vitro trials. Relevant reviewed source(s) information was extracted and presented as a summary via table format. Study characteristics include intervention, population (subjects, animal model or cell type), study type, treatment, and reference. The outcomes/effects of palm oil, palmitic acid or their derivatives within each study were also presented, using: ↑ or ↓ to indicate favourable effects, ↑ or ↓ to indicate unfavourable effects, or — to indicate neutral, or insignificant effect(s). Many of the studies reported multiple outcomes, therefore two or more distinctive effects (favourable, neutral,

and/or unfavourable) could be reported per study. As some (6) studies included multiple experiments, these studies were shared across their relevant categories (i.e., animal, in vitro).

2.3.7 Meta-analysis

A meta-analysis was initially planned for this review. However, it was subsequently deemed unsuitable. In line with section 9.1.4 of the Cochrane Handbook for Systematic Reviews of Interventions (Cochrane, 2024): heterogeneity in study design, including intervention, populations (human, animal, in vitro), measurement tools used, as well as reported health outcomes - would more than likely cause accuracy, legitimacy, and credibility concerns.

2.4 Results

2.4.1 Studies included

The search strategy identified a total of 618 studies, before duplicates (179) removed. Records (439) were subsequently screened for eligibility, based on their title and abstract, leading to a removal of 223 papers due to their relevance, or study type (review). A total of 216 records were sought for retrieval, with 3 unavailable to obtain. The screening process then assessed records (213) for quality and validity, through full-text analysis and risk of bias assessment. A total of 81 studies were removed at this stage. There is a total of 132 studies included within the review, dated from 1985 to 2021 (year-end). The highest number of studies originate from China (22), USA (21), and Malaysia (17). A visualisation of included studies' countries of origin is available to view in Figure 2.1. The PRISMA flow diagram (Figure 2.2) shows the full literature inclusion and exclusion information within each stage.

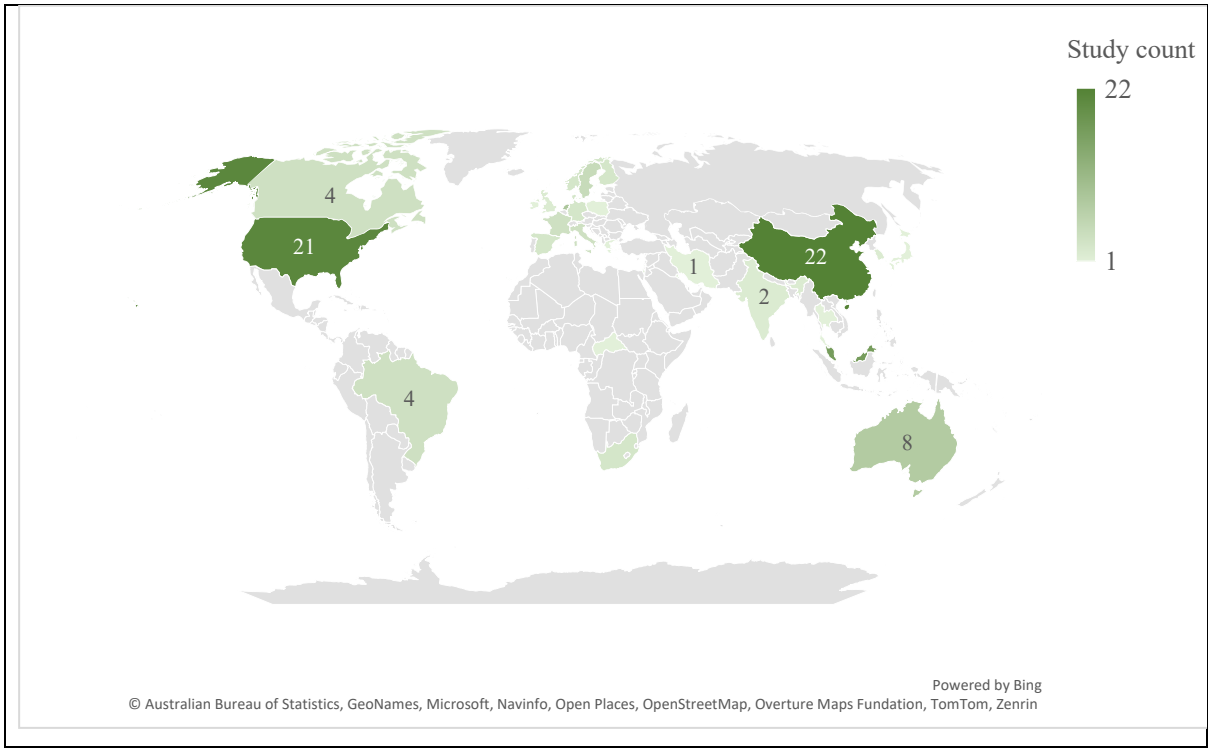


Figure 2.1. Included studies' various countries of origin, by number of studies.

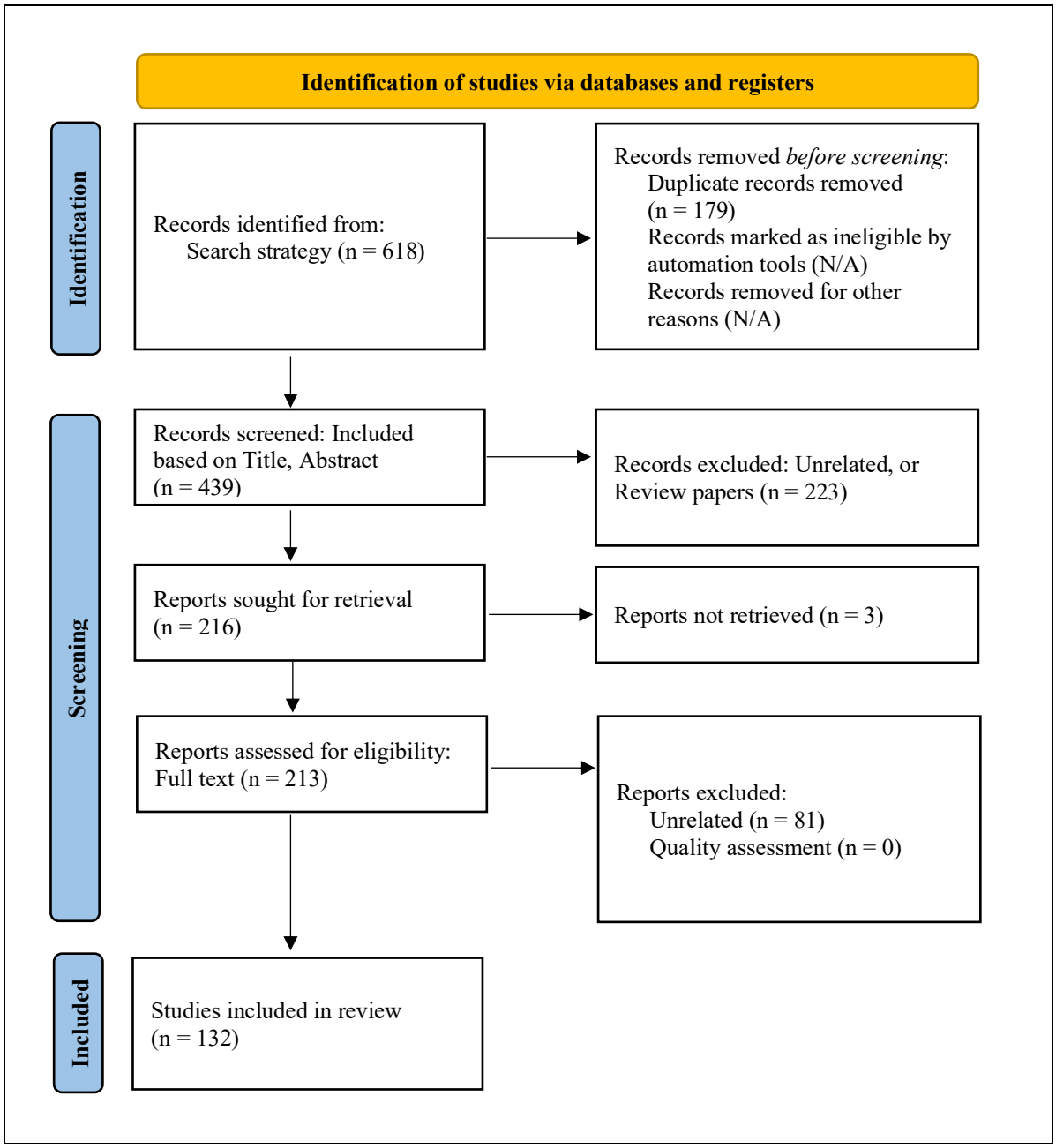


Figure 2.2. The PRISMA flow diagram applied to extract literature on the effects of palm oil, palmitic acid and their derivates on metabolic health.

2.4.2 Presence of bias

A total of 132 studies were assessed for risk of bias. As a total of 6 studies contained two relevant experiments (animal, in vitro), these studies were assessed in both of their respective category and by their respective risk of bias tool. Overall risk of bias for the majority of studies (72) was considered 'Low', indicating that study outcomes were more than likely not

significantly impacted by any element of study design, protocol, or methodology. A total of 30 studies were considered (overall) as 'High' or 'Serious' risks of bias. Interestingly, the majority of these studies originate from Malaysia, the second highest in palm oil production worldwide. More specifically their author(s)' direct affiliation(s) with funding or sponsorship from organisations such as the Palm Oil Research Institute of Malaysia, the Malaysian Palm Oil Council, the Malaysian Palm Bureau, and the Malaysian Palm Oil Board. These conflicts of interest led to concerns in the domains of selection of participants and interventions, measurement tools used, as well as the reporting (including omissions) of outcomes. In human studies, other notable risks of bias included non-randomisation, blinding (such as the application of single blinding, participant only), as well as small sample size. For the animal studies, risks of bias were apparent in the random housing (lack thereof) of animal subjects, incomplete outcome data (failure to report population number), as well as blinding (of caregivers, outcome assessment). For in vitro studies, biases were identified predominantly in the interventions (cell lines) used, such as the selection of unhealthy cells (not representative of effects in healthy cells, potential confounding), as well as non-randomisation, and blinding.

Although several outcomes of included studies should be interpreted with caution, they provide a comprehensive understanding of the available evidence, as well as highlight certain (potential) literature practices as well as study design limitations. Therefore, all studies assessed for risks of bias were included in the review. Risk of bias presence for all included studies is presented in Figures 2.3, 2.4a, 2.4b, 2.5, 2.6a, 2.6b, 2.7.

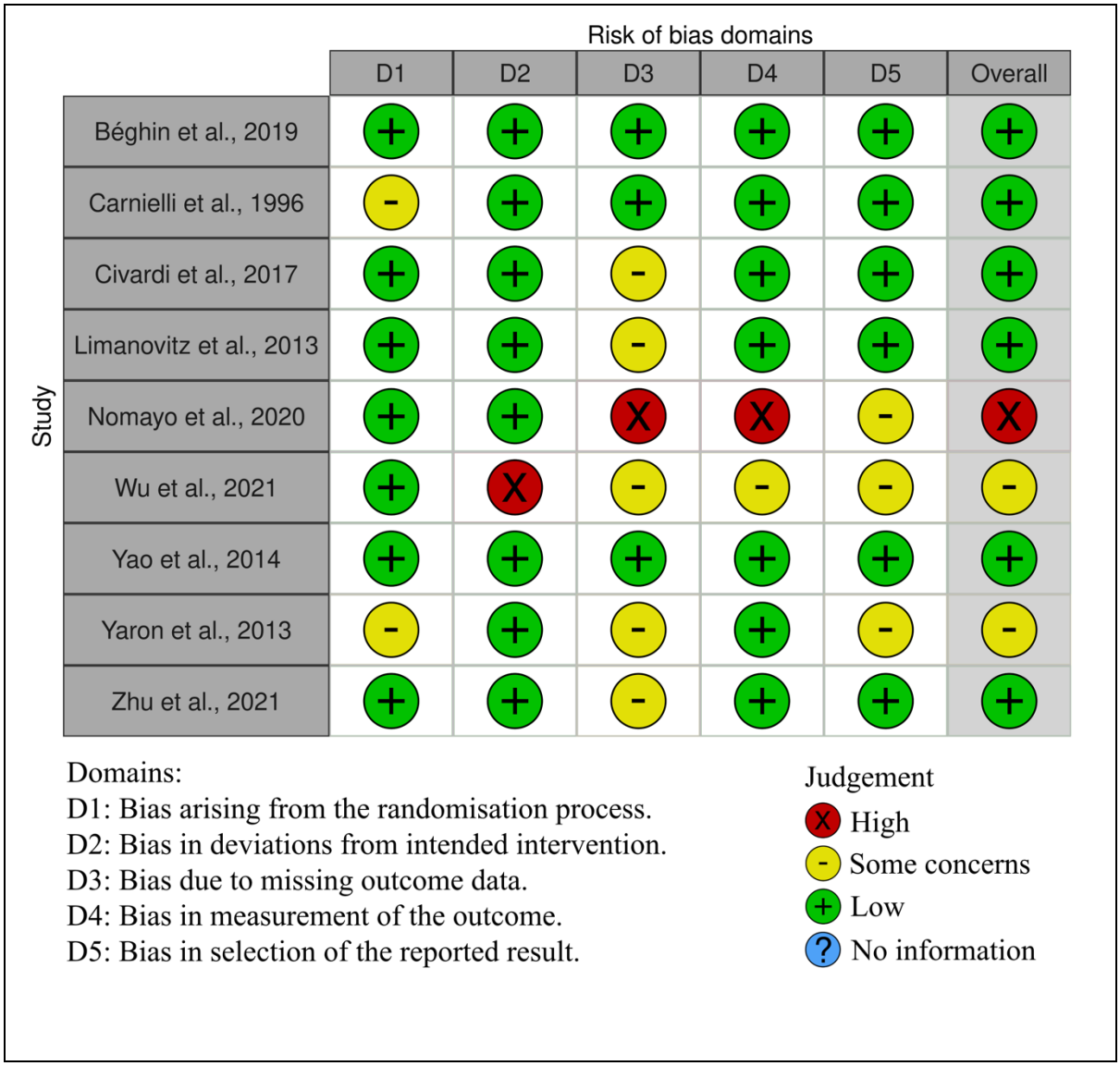


Figure 2.3. Risk of bias assessment of randomised human infant intervention trials (RoB2).

Figure 2.4a Risk of bias assessment of human adult intervention trials (RoB2).

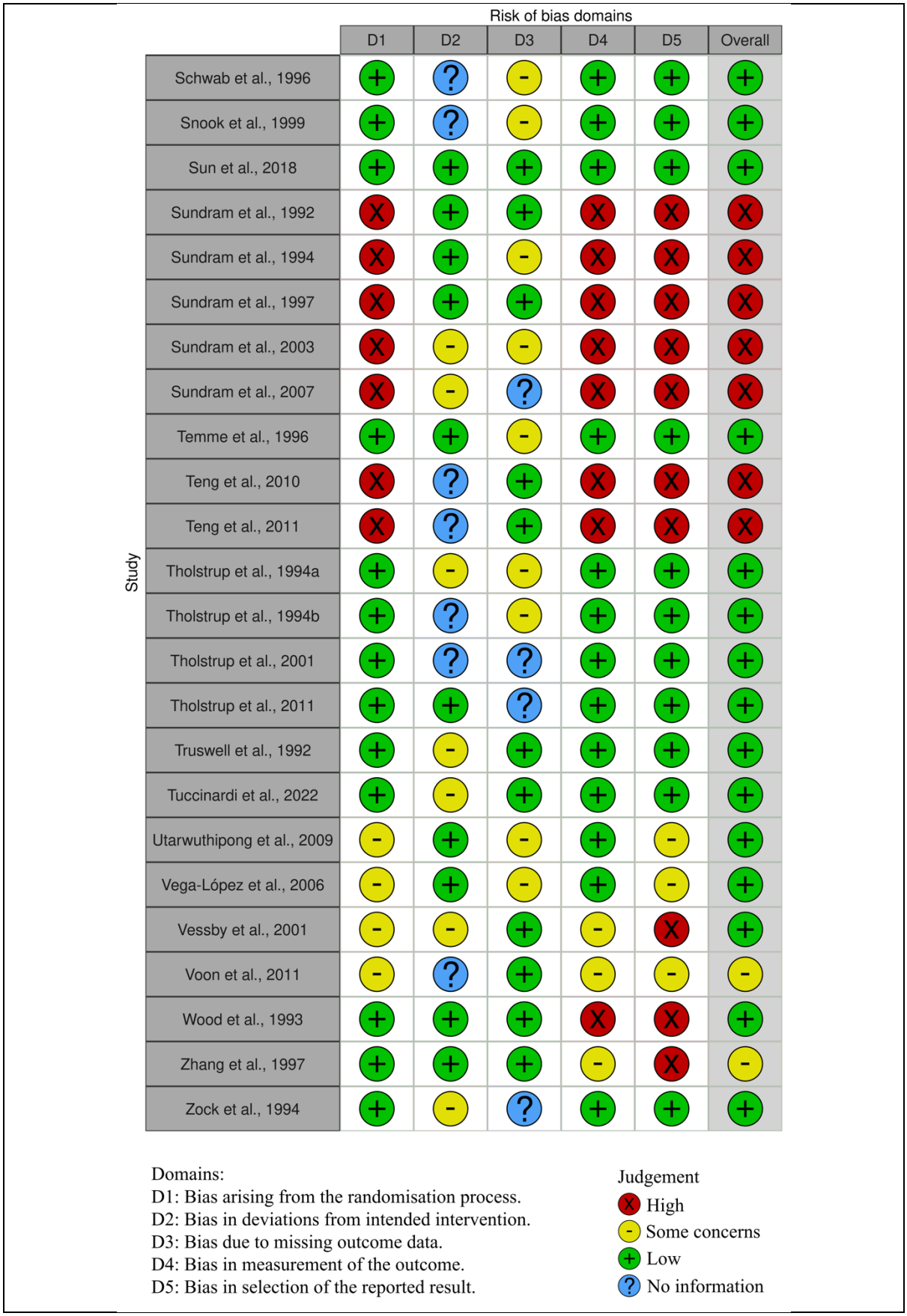


Figure 2.4b Risk of bias assessment of human adult intervention trials (RoB2).

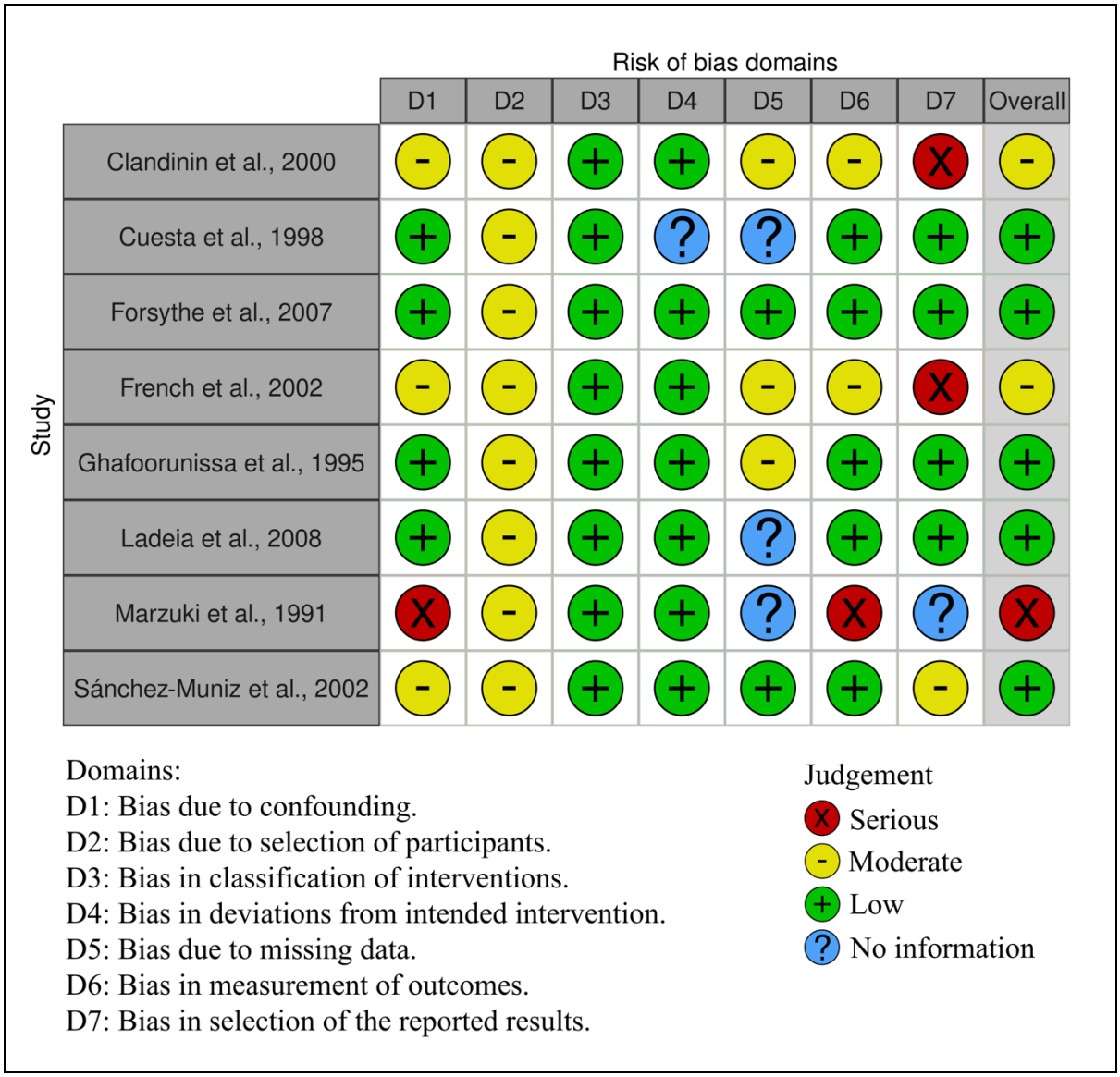


Figure 2.5. Risk of bias assessment of non-randomised human adult intervention trials (ROBINS-I).

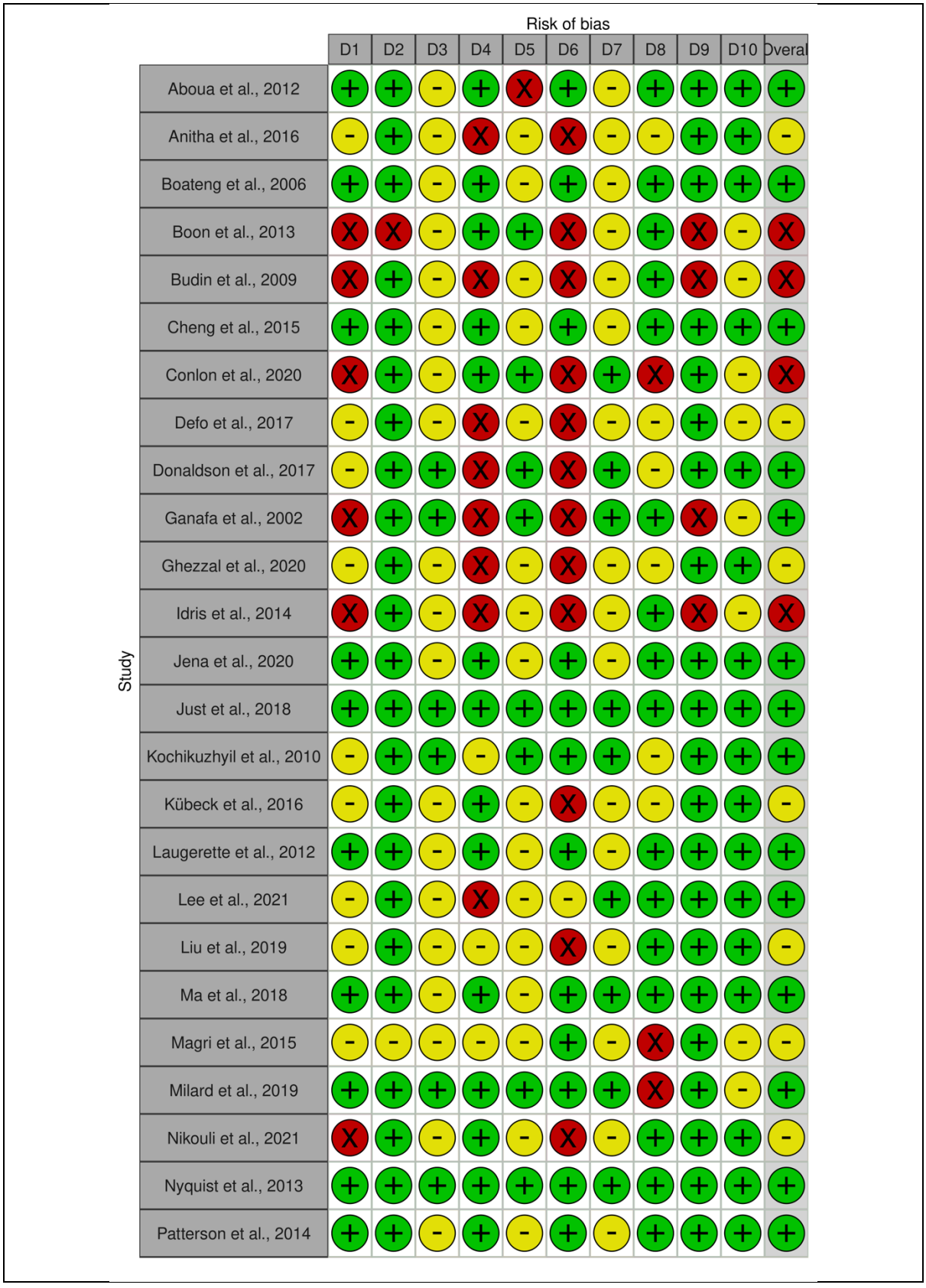

Risk of bias
D1
D2
D3
D4
D5
D6
D7
D8
D9
D10
Overal
Study
Aboua et al., 2012
Anitha et al., 2016
Boateng et al., 2006
Boon et al., 2013
Budin et al., 2009
Cheng et al., 2015
Conlon et al., 2020
Defo et al., 2017
Donaldson et al., 2017
Ganafa et al., 2002
Ghezzal et al., 2020
Idris et al., 2014
Jena et al., 2020
Just et al., 2018
Kochikuzhyil et al., 2010
Kübeck et al., 2016
Laugerette et al., 2012
Lee et al., 2021
Liu et al., 2019
Ma et al., 2018
Magri et al., 2015
Milard et al., 2019
Nikouli et al., 2021
Nyquist et al., 2013
Patterson et al., 2014

Figure 2.6a. Risk of bias assessment of animal trials (SYRCLE).

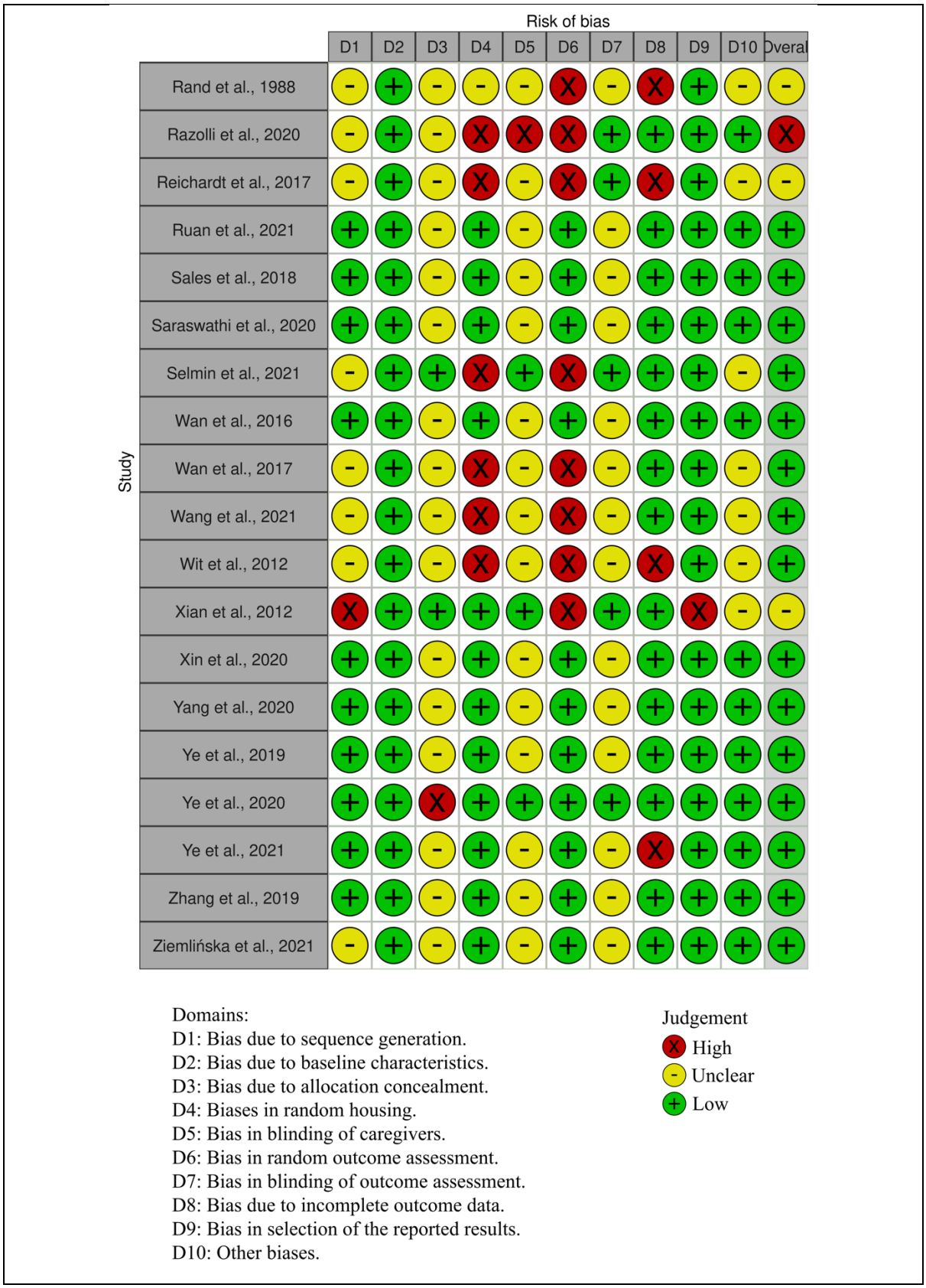


Figure 2.6b. Risk of bias assessment of animal trials (SYRCLE).

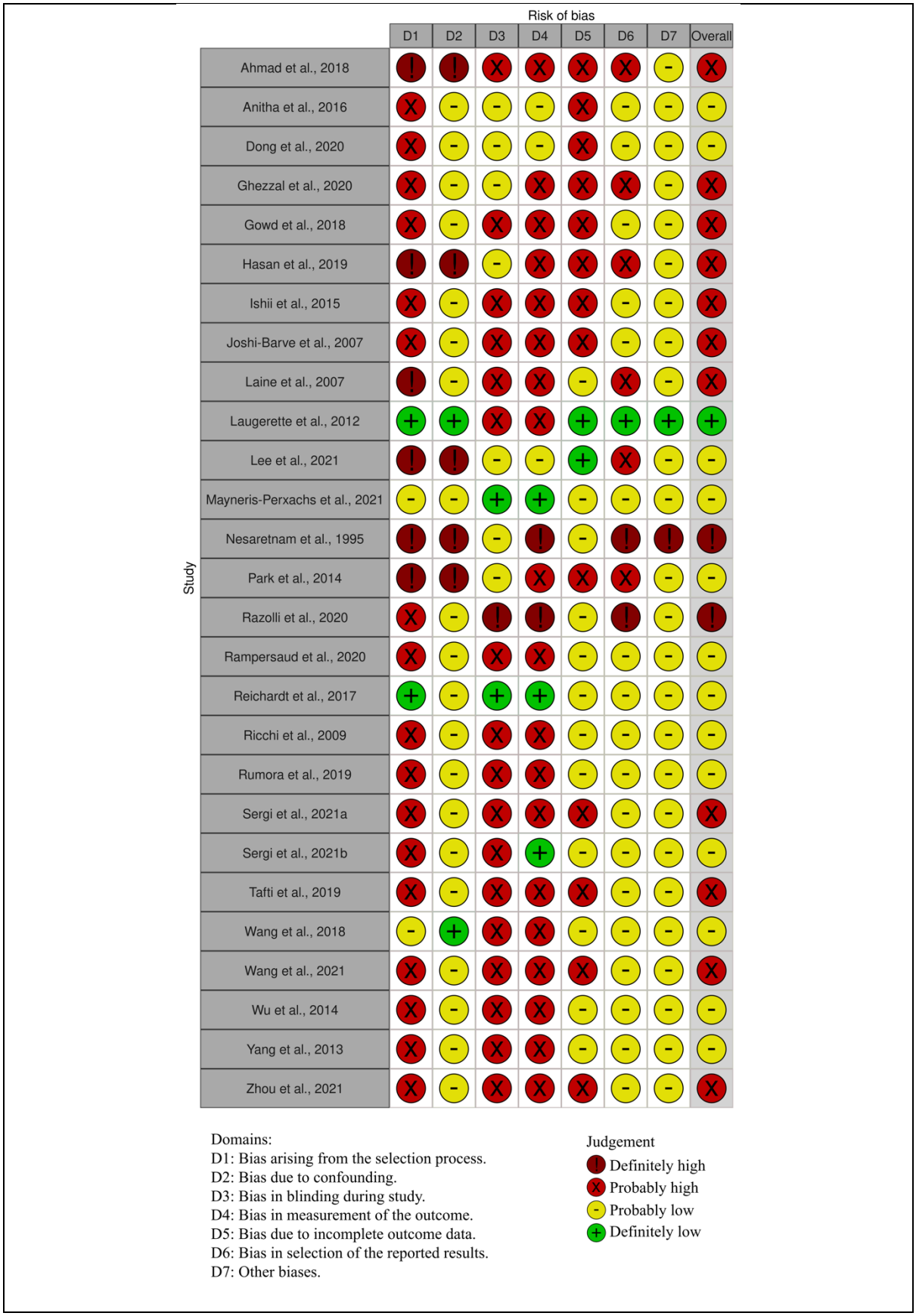
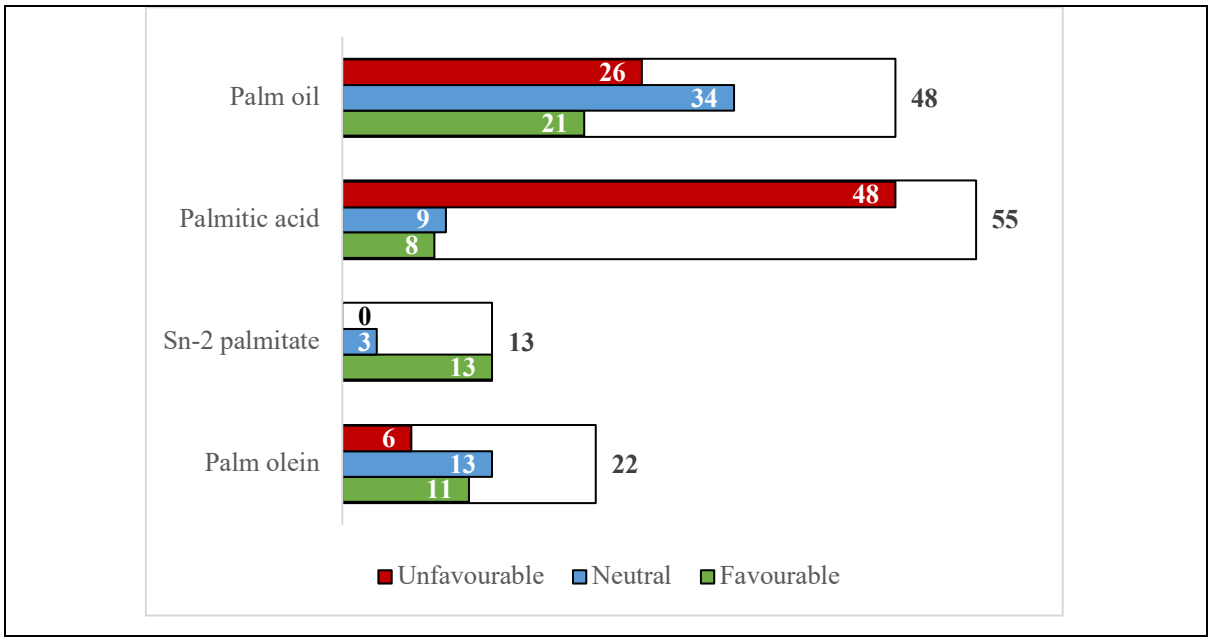


Figure 2.7. Risk of bias assessment of in vitro trials (OHAT).

2.5 Summary of Findings

Based on the reviewed literature, a total of 57% studies reported unfavourable health outcomes of palm oil, palmitic acid, and their derivatives. A total of 33% studies reported favourable health effects, a total of 26% studies reported neutral health effects (see Figure 2.8 for percentages clarification). Included studies investigated various interventions: palm oil (including red/crude palm oil, other variations), palmitic acid/palmitate, sn-2 palmitate/β-palmitic acid, and palm olein. For palm oil, 54% of studies reported unfavourable health outcomes. A total of 71% of studies reported neutral health outcomes, and 42% of studies reported favourable health outcomes. For palmitic acid/palmitate, 87% of studies reported unfavourable health outcomes. A total of 18% of studies reported neutral health outcomes, and 14% studies reported favourable health outcomes. For sn-2 palmitic acid/β-palmitic acid, 100% of studies reported unfavourable health outcomes. A total of 23% of studies reported neutral health outcomes, with 0% of studies reporting unfavourable health outcomes. For palm olein, 27% of studies reported unfavourable health outcomes. A total of 59% of studies reported favourable health outcomes, a total of 50% of studies reported neutral health outcomes. A visualisation of health outcomes for each intervention can be found in Figure 2.8.



Note: Percentages exceed 100%, or outcome total count(s) exceed total study count(s), as several studies reported multiple outcomes. For example, while 48 studies investigating palmitic acid reported unfavourable effects, some of these studies also reported neutral, or favourable effects, thus outcome total (65) exceeds total study count (55). A maximum of 3 outcomes were counted per study, 1 per outcome-type (unfavourable, neutral, favourable).

Figure 2.8. Total number of observed health outcomes of palm oil-related interventions.

Full terms for any abbreviations used throughout the characteristic's tables are presented in Table 2.2. A glossary of interventions used within all study types is presented in Table 2.3.

Palm oil, palmitic acid, and their derivatives' health effects in human (infant) health

A total of 9 studies investigated the effects of palm oil, palmitic acid, and/or their derivatives in human infants. The included studies examined ≥1,424 human infants, ranging from ≥1 days to ≤1 year old. Interventions include sn-palmitate/β-palmitic acid (8 studies) and 1,3-olein-2-palmitin/1,3-dioleate-2-palmitate-glycerol (OPO) (1). All of the included studies (9) reported favourable health outcomes in human infants. A total of 2 studies also reported neutral health outcomes. The details of the studies on human (infant) are presented in Table 2.4.

Palm oil, palmitic acid, and their derivatives' health effects in human (adult) health

A total of 58 studies investigated the effects of palm oil, palmitic acid, and/or their derivatives in human adults. The included studies examined ≥2,222 human adults, ranging from 18 to 75 years old. Interventions include palm oil (18 studies), palmitic acid/palmitate (18), sn-2 palmitate (2), palm olein (21; 1 of which, also investigated palm stearin). The majority of studies (30) reported unfavourable health outcomes in human adults. A total of 25 studies reported neutral health outcomes, while 24 studies reported favourable health outcomes. Study characteristics for human adult trials can be found in Table 2.5.

Palm oil, palmitic acid, and their derivatives' health effects in animal trials

A total of 44 studies investigated the effects of palm oil, palmitic acid, and/or their derivatives in animal trials. The included studies examined ≥1,863 animals, with the majority of trials being of rodent (mice, rats) model (35). Interventions include palm oil (30 studies), palmitic acid/palmitate (10), sn-2 palmitate (2), and a palm olein (1). The majority of included studies (25) reported unfavourable health outcomes in animal trials. A total of 11 studies reported favourable health outcomes, while a total of 7 studies reported neutral health outcomes. Study characteristics for animal trials can be found in Table 2.6.

Palm oil, palmitic acid, and their derivatives' health effects in vitro

A total of 27 studies investigated the effects of palm oil, palmitic acid, and/or their derivatives in in vitro trials, examined in various human (21 studies) or animal (5) cell types/lines. Interventions include palm oil (1 study), palmitic acid/palmitate (26). The majority of included studies (24) reported unfavourable health outcomes in vitro. A total of 3 studies reported

favourable health outcomes, while a total of 1 study reported neutral health outcomes. Study characteristics for in vitro trials can be found in Table 2.7.

Table 2.2. Full terms for any abbreviations used within the characteristic's tables.

Abbreviation	Full term
AP-1	Activator Protein-1
CCL2	C-C motif chemokine ligand 2
CD14	Cluster of differentiation 14
COX-2	Cyclooxygenase-2
CPE	Carboxypeptidase E
CRP	C-reactive protein
EDN1	Endothelin-1
Fas	Fatty acid synthase
FOXO1	Forkhead box O1
GLUT4	Glucose transporter type 4
GOT2	Glutamic-oxaloacetic transaminase 2
IFN-γ	Interferon-γ
IL-6	Interleukin-6
IL-8/CXCL8	Interleukin-8
IL1-β	Interleukin-1beta
IP-10	Interferon-gamma inducible protein 10
JAK2	Janus kinase 2
JNK	c-Jun N-terminal kinase
Lp(a)	Lipoprotein(a)
LPS	Lipopolysaccharide
MAS	Malate-aspartate shuttle
MDHD2	Malate dehydrogenase 2
MIG/CXCL9	Monokine Induced by Gamma Interferon
MyD88	Myeloid differentiation primary response 88
NF-kB	Nuclear Factor kappa-light-chain-enhancer of activated B cells
NLRP3	NOD-, LRR- and pyrin domain-containing protein 3
PC1/3	Proprotein convertase 1
PC2	Proprotein Convertase 2
PCSK1	Prohormone convertase 1/3
PKB/Akt	Protein Kinase B
PPARα	Peroxisome proliferator-activated receptor alpha
PRCP	Prolylcarboxypeptidase

SERPINE1/PAI1	Plasminogen activator inhibitor-1	
SREBP-1c	Sterol Regulatory Element-Binding Protein 1c	
STAT3	Signal Transducer and Activator of Transcription 3	
tHcy	Plasma total homocysteine	
TNF-α	Tumor necrosis factor-alpha	
TXA_2	Thromboxane A2	

Table 2.3. Glossary of included interventions.

Term(s)	Definition
1,3-dioleoyl-2-palmitoylglycerol (OPO)	A triglyceride typically found in infant formula and human breast milk. Consisting of two oleic acid molecules and one palmitic acid molecule (at the sn-2 position).
Calcium palmitate	A salt/soap consisting of calcium and palmitic acid, primarily involved with absorption/malabsorption within the gut. Naturally occurring in dairy products, meats, some edible oils, as well as breast milk.
Carotenoids (β-carotene)	Anti-oxidant sources of vitamin A. Crude palm oil is the highest natural source of carotenoids, of which is predominantly β-carotene.
Crude (red) palm oil	Palm oil in its most natural state; unrefined yet sterilised, pressed and impurities removed. Typically used as a cooking oil. High in vitamin A (β-carotene) and vitamin E (tocopherols, tocotrienols) content.
Elaeis guineensis	African oil palm tree, grown and harvested for palm oil. Heavily used in the commercial production of palm oil.
Elaeis oleifera	American oil palm tree, (very) rarely used to produce commercial palm oil.
Hybrid palm oil	A mixture containing both Elaeis guineensis and Elaeis oleifera.

Oil palm phenolics	Also known as phenols, formed during the palm oil production/refining process. Possess anti-oxidant and anti-inflammatory properties.
Oil palm preparation (OPP)	Formed during the palm oil production/refining process. High polyphenol content.
Palm kernel oil	Obtained from the kernel (seed) of the palm fruit, ~8.5% palmitic acid.
Palm oil	Obtained from the mesocarp (flesh) of the palm fruit, ~44% palmitic acid. Often used as a simplified term for both crude (red), or refined/processed versions.
Palm oil (rapeseed oil blend)	Approximately 4-5x more oleic acid content, 3-4x less palmitic acid content than palm oil alone. Typically used as a cooking oil.
Palm olein	Liquid fraction of palm oil produced after processing/fractionation, ~37% palmitic acid, ~45% oleic acid.
Palm stearin	Solid fraction of palm oil produced after processing/fractionation, ~46% palmitic acid, 36% oleic acid.
Palm vitamin E	Extracted from crude (red) palm oil. A potent anti-oxidant.

Palmitate	The salt and ester of palmitic acid, or the form in which palmitic acid exists in the body. Often referred to within the literature as an alternative term for palmitic acid or utilised within in vitro studies.
Palmitic acid	Hexadecanoic acid. Long-chain fatty acid C16:0. The most abundant saturated fatty acid in the diet.
Palmitoleic acid	Hexadecenoic acid. Palmitic acid synthesised endogenously, (de-saturated) into palmitoleic acid (monounsaturated). Found in trace amounts in most Western diets.
Refined palm oil	Typically refined, bleached, and deodorised (RBD) palm oil. Palm oil as it appears in most foods, specifically processed and ultra-processed foods.
sn-2 Palmitate/β-palmitic acid	Palmitic acid present at the central, or middle, position of the glycerol backbone of triglycerides. Rarely found naturally (palmitic acid is typically found in the sn-1/sn-3 positions).
Super olein	A further processed liquid fraction of palm oil produced after subsequent processing/fractionation of palm olein.
Tocopherols, tocotrienols	Anti-oxidant and anti-inflammatory source of vitamin E. While tocopherols are common in many vegetable oils, the highest tocotrienol content (of any edible oil) is found in crude (red) palm oil.
Tocotrienol-rich fraction (TRF) of palm oil	The extracted vitamin E fraction of crude (red) palm oil, particularly tocotrienol-rich.

Table 2.4. Characteristics of studies investigating palm oil, palmitic acid, and their derivatives' health effects in human infants.

Author(s)	Intervention	Subjects	Study type	Treatment(s)	Outcomes	Implications
Wu et al., 2021	Sn-2 palmitate	Infants (7-14 days old) (n= 199)	Randomised controlled trial	Control (breast feeding) high sn-2 (46.3%) palmitate, low sn-2 (10.3%) palmitate, for 24 weeks	Higher dosage: ↑ Fine motor skills ↑ Faecal Bifidobacteria	↑ Neurodevelopment ↑ Eubiosis
Béghin et al., 2019	Sn-2 palmitate	Healthy term infants (1-7 days old) (n=488)	Randomised controlled trial	Control formula (22% palmitate; 16% sn-2), formula 1 (21.7% palmitate; 43% sn-2), formula 2 (19.8% palmitate; 51% sn-2), for 12 months	Higher dosage: ↑ Improved stool consistency ↑ Bone mineral content	↑ Digestion ↓ Constipation ↓ Risk of fracture
Yao et al., 2014	Sn-2 palmitate	Infants (7-14 days old), (n= 300)	Randomised controlled trial	Standard formula, sn-2 PA, sn2+3g/L oligofructose (OF), sn-2+5g/L oligofructose, palmitate (21.3%), palmitate (22.8%), palmitate (23.0%), palmitate (23.2%), for 8 weeks	↓ Stool soaps ↑ Softer stools ↑ Bifidobacteria	↓ Malabsorption ↑ Eubiosis
Carnielli et al., 1996	Sn-2 palmitate	Healthy term infants, total n= 27	Randomised controlled trial	Beta (24% palmitic acid, 66% sn-2), intermediate (24% palmitic acid, 39% sn-2), regular (20% palmitic acid; 13% sn-2), for 5 weeks	↑ Fat, calcium intestinal absorption	↑ Digestion ↓ Malabsorption
Nomayo et al., 2020	β-palmitic acid	Infants (<10 days old), total n= 94	Randomised controlled trial	Control (breast feeding), β-palmitic acid 10%, 20-25%, for 12 weeks	↑ Faecal Bifidobacteria — Gastrointestinal infections — Weight gain	↑ Eubiosis — Metabolic homeostasis

Civardi et al., 2017	β-palmitic acid	Infants (<11.1 days old), total n= 110	Randomised controlled trial	Standard formula, enriched formula (60% PA, 39% sn-2 PA), for 135 days	↑ Faecal Bifidobacteria — No significant differences in gastrointestinal effects	↑ Eubiosis — Metabolic health
Yaron et al., 2013	β-palmitic acid	Infants (<7 days old), total n= 32	Randomised controlled trial	High β-palmitic acid (44% sn-2), low β-palmitic acid (14% sn-2), for 6 weeks	↑ Increased Lactobacillus counts in faecal stools ↑ Increased Bifidobacteria counts in faecal stools	↑ Eubiosis
Limanovitz et al., 2013	β-palmitate	Healthy term infants (<14 days old) total n= 66	Randomised controlled trial	Control (breast feeding), high β-palmitate formula (22% palmitate, sn-2 44%), low β-palmitate formula (19% palmitate, sn-2 14%), for 12 weeks	↑ Improved bone strength positively correlates with β-palmitate	↓ Risk of fracture
Zhu et al., 2021	1,3-olein-2-palmitin/1,3-dioleate-2-palmitate-glycerol (OPO)	Infants (<1 months old), total n= 108	Randomised controlled trial	Standard formula, OPO (sn-2 PA) supplemented (4%) formula, for 4 months	↑ Intestinal microbial diversity ↑ Enhydrobacteria and Akkermansia	↑ Eubiosis

Table 2.5. Characteristics of studies investigating palm oil, palmitic acid, and their derivatives' health effects in human adults.

Author(s)	Intervention	Subjects	Study type	Treatment(s)	Outcomes	Implications
Rosqvist et al., 2014	Palm oil	Healthy men and women (20-38 years old), total n= 41	Randomised trial	Palm oil-based muffins (high palmitic acid, PA), sunflower oil-based muffins (high linoleic acid), (51% of energy from fats) for 7 weeks	↑ Increased liver fat accumulation	↑ Metabolic dysfunction-associated steatotic liver disease (MASLD)
Engelberts et al., 1993	Palm oil	Healthy males (19-45 y/o), total n= 38	Randomised controlled crossover trial	High-fat, high-fat + palm oil, for 12 (6 x 2) weeks	— IL-6, IL-8 ↓ TNFα ↓ Proneness of the peripheral blood cells to produce TNFα was reduced	↓ Non-inflammatory
Utarwuthipong et al., 2009	Palm oil	Hypercholesterolaemic women (44-67 years old), total n= 16	Clinical trial	Palm oil (38.6% PA), soybean oil (10.9% PA), rice bran oil (19.7% PA), rice bran/palm (3:1) mixture (24.4% PA), for 40-48 weeks	↑ Low-density lipoprotein cholesterol (LDL-C)	↑ Hyper-cholesterolemia ↑ CVD
Vega-López et al., 2006	Palm oil	Postmenopausal women, ≥50 y (LDL cholesterol ≥3.4mmol/L, or ≥130 mg/dL), total n= 15	Randomised trial	Palm oil (~11% PA), partially hydrogenated soybean oil, soybean oil, canola oil, (~30% of energy from fats), for 210 (35 x 6) days	↑ LDL-C ↑ Apolipoprotein-B — Total/HDL cholesterol ratio	↑ Hyper-cholesterolemia — CVD

Pederson et al., 2005	Palm oil	Adults, total n= 36	Randomised trial	Palm-margarine (33.9-37.2% PA), trans-margarine (12.5-15.8% PA), PUFA-margarine (8.7-11% PA), for 51 (17 x 3) days	— No significant differences in total cholesterol, LDL-cholesterol, and apolipoprotein-B ↑ HDL-cholesterol and apolipoprotein A-I significantly higher	— Cholesterol homeostasis ↓ Hyper-cholesterolemia
Müller et al., 1998	Palm oil	Females (19-42 years old), total n= 37	Clinical trial	Palm-margarine: 80% palm oil, 11% soybean oil, 9% rapeseed oil, trans-margarine: 56% partially hydrogenated soybean oil, 34% refined rapeseed oil, 10% refined soybean oil, PUFA-margarine: coconut oil, palm oil, refined sunflower oil and refined rapeseed oil, for 17 days	↑ Increased total cholesterol ↑ LDL-cholesterol ↑ Apolipoprotein-B	↑ Hyper-cholesterolemia ↑ CVD
Cater et al., 2001	Palm oil	Mildly hypercholesterolemic men, total n= 7	Randomised crossover trial	Palm oil (48.3% PA), behenate oil (3.2% PA), sunflower oil (3.5% PA), for 9 (3 x 3) weeks	↑ Increased total cholesterol, LDL-C	↑ Hyper-cholesterolemia ↑ CVD
Cater et al., 1997	Palm oil	Men (55-75 years old), total n= 9	Randomised crossover	Palm (48.3% PA), sunflower (3.5% PA), medium-chain triacylglycerol diet, for 9 (3 x 3) weeks	↑ Total cholesterol and LDL-C	↑ Hyper-cholesterolemia ↑ CVD

Tholstrup et al., 1994	Palm oil	Men (21-26 years old), total n= 12	Randomised trial	Palm oil (42.9% PA), myristic acid (2.4% PA), for 6 (3 x 2) weeks	↑ Myristic acid increases plasma total cholesterol in the presence of palmitic acid	↑ Hyper-cholesterolemia ↑ CVD
Sundram et al., 1992	Palm oil	Males (19-45 years old), total n= 38	Randomised controlled crossover trial	Control (20.8% PA), palm oil (28.6% PA), for 12 (6 x 2) weeks	— Serum TC and TG concentrations unaffected ↓ LDL: HDL ↓ Apolipoprotein-B: apolipoprotein-A1 ratio reduced	— Cholesterol homeostasis ↓ Hyper-cholesterolemia
Zhang et al., 1997	Palm oil	Experiment a: normocholesterolemic men (18-25 years old), total n= 120 Experiment b: hypercholesterolemic adults (n=31 men, n=20 women; 32-68 years old), total n= 51	Randomised trial	Experiment a: palm oil (35.9% PA), soybean oil (12.4% PA), peanut oil (18% PA), lard (21.6%), for 24 (6 x 4) weeks Experiment b: palm oil (32.7% PA), peanut oil (16%), for 12 (6 x 2) weeks	— Does not raise serum cholesterol	— Cholesterol homeostasis
Sundram et al., 1994	Palm oil	Normocholesterolemic humans, total n= 17	Randomised trial	Palm diet (550g palm stearin, 250g palm oil, 100g palm kernel oil, 100g corn oil) (11.1 ± 0.6% PA), lauric-myristic diet (300g coconut oil, 350g palm kernel	↓ Serum cholesterol concentration ↓ LDL-C concentrations ↓ HDL-C concentrations — Apolipoprotein-A or apolipoprotein-B	↓ Hyper-cholesterolemia ↑ Stroke ↑ CVD — Cholesterol homeostasis

				oil, 350g canola oil) (6.9 ± 0.4% PA), for 8 (4 x 2) weeks		
Mattson et al., 1985	Palm oil	Adults, total n= 20	Randomised trial	SAT: Palm oil (43.4% PA), MONO: high oleic safflower oil (5.1% PA), POLY: high linoleic safflower oil (6.4% PA), 40% of energy from fats, for 4 weeks	↓ HDL-C ↑ LDL-C	↑ Hyper-cholesterolemia ↑ CVD
Tuccinardi et al., 2021	Palm oil	Healthy adults (mean age 24.2 years old), total n= 20	Randomised crossover trial	Chocolate spread enriched with palm oil (100g daily), chocolate spread enriched with extra virgin olive oil (100g daily), for 2 weeks	↑ Circulating harmful sphingolipids ↓ Insulin sensitivity	↑ CVD ↑ Insulin resistance ↑ Hyperglycaemia ↑ Type 2 diabetes
Lucci et al., 2016	Hybrid palm oil (Elaeis oleifera × E. guineensis)	Adults, n= 160	Randomised trial	Hybrid palm oil (27.9 ± 1.2% PA) n=78 25 mL (daily), extra virgin olive oil (12.5 ± 0.8% PA) n=82, 25 mL (daily), for 3 months	↓ Total cholesterol, LDL-C	↑ Lipid profile ↓ Hyper-cholesterolemia
Ladeia et al., 2008	Red palm oil (boiled)	Medical students (16-26 years old), total n= 34	Randomised trial	Boiled red palm oil, 10mL, for 2 weeks	↓ Mild triacylglycerol (triglyceride) reducing effect ↑ Mild LDL-C-increasing effect in males	↓ MASLD ↑ Hyper-cholesterolemia ↑ CVD

Tholstrup et al., 1994	Palm oil	Men, total n= 15	Randomised trial	Palm oil (43.3% PA), palm-kernel oil + high-oleic sunflower oil (6.8% PA), shea butter (3.9% PA), for 9 (3 x 3) weeks	↑ Plasma cholesterol, LDL-C, apolipoprotein-B ↑ HDL-C, apolipoprotein A-I ↑ Factor VII coagulant activity	↑ Hyper-cholesterolemia ↑ CVD
Wood et al., 1993	Palm oil (crude)	Healthy men (30-60 years old), total n= 21	Randomised trial	Crude palm oil (37.3% PA), butter (31.3% PA), margarine (12.5% PA), refined palm oil (42% PA), sunflower oil (7.9% PA), 80% refined palm oil + 20% sunflower oil (29.5% PA), for 30 (6 x 5) weeks	Crude: ↑ HDL-cholesterol and apolipoprotein-A1 levels — Did not elevate serum cholesterol	↓ Hyper-cholesterolemia — Cholesterol homeostasis
Sun et al., 2018	Palm olein	Chinese adults, total n= 120	Randomised trial	Palm olein 48g (daily) (n=60), olive oil 48g (daily) (n=60), for 2 months	— No significant different effect on BMI — Serum total cholesterol — LDL-C + HDL-C — Triacylglycerol — Apolipoprotein-B — Fasting glucose concentration(s) — Insulin concentration(s)	↓ Hyper-cholesterolemia — Cholesterol homeostasis — Glucose homeostasis — Insulin homeostasis — Oxidative homeostasis

Lv et al., 2018	Palm olein	Adults (21.59 ± 0.39 years old), total n= 88	Randomised controlled trial	Palm olein (29.4% PA), soybean oil (11.1% PA), cocoa butter (26.2%), for 16 weeks	— Lipid profile — Insulin resistance — Liver/kidney function (oxidative stress)	— Lipid homeostasis — Insulin homeostasis
Tholstrup et al., 2011	Palm olein	Healthy adult males, 19 - 64 y, total n= 32	Randomised controlled trial	Palm olein, olive oil, lard, for 9 (3 x 3) weeks	↑ Increased plasma cholesterol	↑ Hyper-cholesterolemia ↑ CVD
Voon et al., 2011	Palm olein	36 women, 9 men (avg. age 30 years old), total n= 45	Randomised crossover trial	Palm olein, coconut oil, olive oil, for 5 days	— Postprandial, fasting plasma (tHcy) concentrations — Inflammatory markers TNF-α, IL-1β, IL-6, and IL-8, high-sensitivity, C-reactive protein, and interferon-γ	— Homocysteine homeostasis — Non-inflammatory ↓ CVD ↓ T2D
Teng et al., 2011	Palm olein	Healthy males (21.9 ± 0.7 years old), total n= 10	Randomised crossover trial	Palm plein (20.5% PA), olive oil (7.1% PA), lard (15.2% PA), for 3 days	— Plasma glucose — Insulin — adipocytokines (IL-6, TNF-α, IL-1b, leptin)	— Glucose homeostasis — Insulin homeostasis ↓ Risk of T2D
Karupaiah et al., 2011	Palm olein	Healthy adults (22-38 years old), total n= 20	Randomised trial	Palm olein (POL), Lauric + myristic acid-rich oil (coconut and corn oils) (LM), Stearic acid-rich oil (cocoa butter + corn	↑ HDL-C	↓ CVD

				oil) (STE), POL: PA% 38.82, LM:PA% 8.91, STE: PA% 25.76, for 8 hours		
Sundram et al., 2007	Palm olein	Adults (30 ± 8 years old), total n= 30	Randomised crossover trial	Palm olein (12% PA), partially-hydrogenated soybean oil (6.5% PA), interesterified fat (IE) (12% stearic acid), (~31% of daily energy) for 12 (3 x 4 crossover) weeks	↓ HDL/LDL ratio ↓ Lower fasting blood glucose	↓ CVD ↓ T2D
Sundram et al., 2003	Palm olein	Adult females, total n= 10	Randomised controlled trial	Palm olein, high saturated fat diet (10.6±1.51% PA), control (coconut oil mixture), high trans-fat diet (4.54±0.60% PA), palm oil + coconut oil (90:10) (9.77±1.65% PA), for 30 days	↓ LDL-C levels	↑ Lipid profile
Sánchez-Muniz et al., 2002	Palm olein	Postmenopausal women, total n= 15	Non-randomised trial	Palm olein (14.32 ± 1.27% PA), oleic acid-rich sunflower oil (5.59 ± 0.77% PA), for 28 days	↑ Serum total cholesterol ↑ Significant increase in HDL cholesterol, HDL lipids, HDL proteins, HDL total mass ↑ Increased Apolipoprotein A-II levels ↑ Phospholipids	↑ Hyper-cholesterolemia ↑ CVD ↓ MetS

Cuesta et al., 1998	Palm olein	Adults (63 ± 11years old), total n= 14	Non-randomised trial	Basal oil (8.3 ± 0.2% PA), sunflower oil (4.4 ± 0.3% PA), palm olein (37.7 ± 0.3% PA), for 56 (28 x 2) days	↑ Increased LDL-C ↑ Apolipoprotein-B concentrations	↑ Hyper-cholesterolemia ↑ CVD
Ng et al., 1991	Palm olein	Males (n=61), females (n=22); 20-34 years old), total n= 83	Randomised trial	Refined, bleached, deodorised (RBD) palm olein, RBD coconut oil, RBD corn oil, for 15 weeks	↓ Significant reductions in serum TC, LDL-C and TC/HDL-C	↑ Lipid profile ↓ CVD
Sundram et al., 1997	Palm olein	Adults (n=19 men, n=8 women), total n= 27	Randomised crossover trial	Trans-rich diet (70% hydrogenated soybean oil, 30% soybean oil), Monounsaturated-rich diet (30% rapeseed oil, 25% sunflower seed oil, 45% palm olein), Palmitic-rich diet (100% palm olein), lauric-myristic-rich diet (45% coconut oil, 15% palm kernel oil, 40% corn oil), for 18 weeks	↓ LDL-C ↑ HDL-C	↑ Lipid profile ↓ CVD
Choudhury et al., 1995	Palm olein	Healthy normocholesterolemic men and women (19 to 44 years old), total n= 21	Randomised crossover trial	Palm olein (40.13 ± 0.21% PA), olive oil (10.52 ± 0.24% PA), PO (% PA), OO (% PA); for 60 (30 x 2) days	— Plasma TC and LDL-C concentrations	↓ CVD — Cholesterol homeostasis

Ghafoorunissa et al., 1995	Palm olein	Men (n=12), women (n=12), total n= 24	Non-randomised crossover trial	Refined, bleached, deodorised (RBD) palm olein (32.1 ± 2.9% PA), groundnut oil (30.0 ± 2.2% PA) experiment a: for 16 (8 x 2) weeks, experiment b: for 32 (16 x 2) weeks	— LDL/HDL ratio	↓ Hyper-cholesterolemia
Ng et al., 1992	Palm olein	Adults (n=20 males, n=13 females), 22-41 years old, total n= 33	Randomised controlled trial	Palm olein-rich diet (39.5% PA), for 4 weeks, coconut oil-rich diet (14.5% PA), for 6 weeks, olive oil-rich diet (18.6% PA), for 6 weeks	↑ Beneficial effects on thromboxane/prostacyclin ratio in plasma — Total cholesterol	↓ CVD — Cholesterol homeostasis
Truswell et al., 1992	Palm olein	Adults, total n= 21	Randomised controlled crossover trial	Palm olein crisps (39-43% PA), canola oil crisps (39-43% PA) 53g (men), 35g (women), for 5 weeks	↑ HDL-C	↑ Lipid profile ↓ CVD
Marzuki et al., 1991	Palm olein	Healthy male students (16-17 years old), total n= 110	Non-randomised trial	Palm olein, soybean oil, used as cooking oil, for 5 weeks	— Did not significantly alter plasma total cholesterol, LDL-C, HDL-C concentrations TC: HDL-C ratio (compared with SO) ↑ Apolipoprotein A-I ↑ Apolipoprotein-B	— Cholesterol homeostasis ↓ Hyper-cholesterolemia ↑ CVD

Teng et al., 2010	Palm olein Palm stearin	Adults, total n= 41	Randomised crossover trial	Palm olein diet (8.7 ± 0.1% PA), partially hydrogenated soybean oil diet (8.1 ± 3.1% PA), palm stearin diet (13.9 ± 0.3% PA), for 15 (5 x 3) weeks	Palm olein: ↑ Beneficial effects on blood lipids and inflammatory markers — IL-6, IL-1b, and TNF-a Palm stearin: ↑ Significantly increased total/HDL cholesterol ratio ↑ IL-8	↓ Inflammation ↓ Hyper-cholesterolemia ↑ CVD ↑ Hyper-cholesterolemia ↑ Inflammation
Scholtz et al., 2004	Palm olein Palm olein (red)	Hyperfibrinogenemic Adults (n=37 men, n=22 women), total n= 59	Randomised trial	Palm olein (40.9% PA), red palm olein (36.2% PA), sunflower oil (5.8% PA), for 4 weeks	Red palm olein: — Total serum cholesterol — LDL-C Palm olein: ↑ Total serum cholesterol ↑ LDL-C	↓ Hyper-cholesterolemia ↑ Hyper-cholesterolemia
Filippou et al., 2014	Palm olein Sn-2 palmitate	Healthy Asian men and women (20-50 years old) total n= 41	Randomised controlled trial	Palm olein (9.8% sn-2 PA), interesterified palm olein (45.9% sn-2 PA), high oleic acid sunflower oil diet (0.5% sn-2 PA), for 21 weeks	— Lipid or lipoprotein concentrations — Insulin secretion	↓ CVD — Cholesterol homeostasis — Glucose homeostasis
Sanders et al., 2011	Palm olein Palm olein (interesterified)	Adults (n=25 men, n=25 women), 18-45 years old, total n= 50	Randomised crossover trial	Palm olein (42.5% PA, 9.2% sn-2), high oleic sunflower oil (4.4% PA, 0.6% sn-2), interesterified palm olein (42.5% PA, 39.1% sn-2)	↓ Reduced stool soaps	↑ Digestion ↓ Malabsorption

				lard, (via muffins), lard (29.1%, 70.5% sn-2, for 4 weeks		
Forsythe et al., 2007	Sn-2 palmitate	Adult males, total n= 10	Non-randomised trial	Sn-2 palmitate (7-11% PA), (30-35% energy from fats) for 8 (2 x 4) weeks	↓ Fasting serum total cholesterol (TC), TC/HDL ratio	↑ Lipid profile
Iggman et al., 2014	Palmitic acid	Adults (27 ± 4 years old), total n= 39	Randomised controlled trial	Palm oil-based muffins, sunflower oil-based muffins, 51% energy from fat, for 7 weeks	↑ HDL-C levels ↑ LDL/HDL-C ratio ↑ Apolipoprotein B:AI ratio — No significant differences were observed for other cardiometabolic risk markers	↓ CVD ↑ Hyper-cholesterolemia — Metabolic homeostasis
Bjermo et al., 2012	Palmitic acid	Abdominally obese adults, total n= 61	Randomised controlled trial	SFA diet (baked-on butter scones, butter) (11.24±0.59% PA), PUFA (n-6) diet (sunflower oil, margarine, sunflower seeds) (11.30±0.58% PA), for 10 weeks	↑ TNF-receptor 2 activation ↑ IL-1 receptor activation ↑ Increased liver fat	↑ Inflammation ↑ Oxidative stress ↑ MASLD

Mensink et al., 2008	Palmitic acid	Healthy men and women, total n= 44	Randomised controlled trial	High-palmitic acid, trans-free diet (37.0% PA), high-oleic acid, low trans diet (8.5% PA), 40% energy from fats, for 6 weeks (3 x 2) weeks	↑ TC, LDL-C ↑ HDL-C levels	↑ Hyper-cholesterolemia ↓ CVD
Baer et al., 2004	Palmitic acid	Adult males, total n= 50	Randomised controlled crossover trial	Lauric, myristic and palmitic acid (LMP) diet, carbohydrate (CHO) diet, oleic acid (OL) diet, stearic acid (STE) diet, trans fatty acid (TFA) diet, TFA + STE diet, 39% energy from fats, for 30 (5 x 6) weeks	↑ C-reactive protein ↑ Fibrinogen ↑ IL-6 concentrations	↑ T2D ↑ Autoimmune disease ↑ Inflammation ↑ Blood clots ↑ CVD
French et al., 2002	Palmitic acid	Experiment a: Males (n=3), females (n=3), 25.0 ± 1.7 years old, total n= 6 Experiment b: Normolipidemic females, total n= 10	Non-randomised trial	Experiment a: 8 diets (~10% PA), experiment b: baseline (9.8 ± 1.7% PA), high saturated fat (10.6 ± 1.5), high trans-fat (4.5 ± 0.6% PA), for 8 months	— 16:0 has no effect on serum lipoprotein profiles in the presence of recommended intakes for linoleic acid	— Non-hypercholesterolemic

Kelly et al., 2002	Palmitic acid	Adult males, total n= 9	Randomised crossover trial	Stearic acid diet 13g SA (daily), 17g PA (daily), palmitic acid diet 7g SA (daily), 22g PA daily), 30% of energy from fats, for 6 (3 x 2) weeks	— Plasma lipoprotein concentrations, platelet aggregation or platelet activation status	— Cholesterol homeostasis ↓ Blood clots
Tholstrup et al., 2001	Palmitic acid	Adult males (21-28 years old), total n= 16	Randomised trial	High palmitic acid diet, high palmitic and myristic acid diet, high stearic acid diet, high oleic acid diet. high trans fatty acid diet, high linoleic acid diet, for 6 (1 x 6) days	↑ Concentrations of apolipoprotein-B	↑ Hyper-cholesterolemia ↑ CVD
Vessby et al., 2001	Palmitic acid	Healthy adults, total n= 162	Randomised trial	High saturated diet, high monounsaturated diet, random assignment to fish oil or placebo, for 3 months	↓ Insulin sensitivity — Insulin secretion	↑ T2D
Clandinin et al., 2000	Palmitic acid	Healthy adult males (20-32 years old), total n= 6	Non-randomised trial	Low 16:0/low 18:2 (3.2 ± 0.2% PA), low 16:0/high 18:2 (2.9 ± 0.2% PA), high 16:0/low 18:2 (10.1 ± 0.4% PA), high 16:0/high 18:2 (9.9 ± 0.2% PA), for 84 (21 x 4) days	— 16:0 has no effect on serum lipoprotein profiles in the presence of recommended intakes for linoleic acid	— Cholesterol homeostasis
Clandinin et al., 1999	Palmitic acid	Moderately hyperlipidaemic male and	Randomised trial	Low 16:0 diet (2.9 ± 0.2% PA), high 16:0 diet (9.9 ± 0.2% PA), for 21 days	— 16:0 has no effect on serum lipoprotein profiles in the presence of	— Cholesterol homeostasis

		females, total n= 6			recommended intakes for 18:2n-6	
Snook et al., 1999	Palmitic acid	Healthy premenopausal women, total n= 18	Randomised crossover trial	Baseline (8.4 ± 0.1% PA), palmitic acid (27.8 ± 0.7% PA), myristic acid (6.3 ± 0.2% PA), stearic acid (5.7 ± 0.1% PA), for 15 (5 x 3) weeks	↑ TC, LDL-C	↑ Hyper-cholesterolemia
Temme et al., 1996	Palmitic acid	Adults (20-60 years old), total n= 39	Randomised crossover trial	Palmitic acid (45.1% PA), lauric acid (6.8% PA), oleic acid (12.1% PA), for 18 (6 x 3) weeks	↑ LDL-C	↑ Hyper-cholesterolemia ↑ CVD
Schwab et al., 1996	Palmitic acid	Healthy females (20-31 years old), total n= 12	Randomised crossover trial	Palmitic acid-enriched diet (11.8 ± 0.1% PA), stearic acid-enriched diet (8.5 ± 0.2% PA), 37% of energy from fats, for 8 (4 x 2) weeks	↑ HDL-C, apolipoprotein-A1 concentrations ↑ Total cholesterol ↑ Higher plasma CETP activity	↓ CVD ↑ CVD
Schwab et al., 1995	Palmitic acid	Healthy females (19-34 years old), total n= 15	Randomised crossover trial	Baseline (1.74 ± 0.2% PA), palmitic acid (11.4 ± 0.1% PA), stearic acid (7.2 ± 0.2% PA), for 8 (4 x 2) weeks	— Total cholesterol — Apolipoprotein A-I, apolipoprotein-B, Lp(a), plasma cholesteryl ester	— Cholesterol homeostasis

					transfer protein activity (CETP)	
Zock et al., 1994	Palmitic acid	Healthy Adults (18-62 years old), total n= 59	Randomised trial	Palmitic acid diet (14.9%), myristic acid diet (4.7% PA), oleic acid diet (5% PA), 10% of total energy from fat, for 3 weeks	↑ LDL-C	↑ Hyper-cholesterolemia ↑ CVD
Denke & Grundy, 1992	Palmitic acid	Adult males (44-71 years old), total n= 14	Randomised trial	High palmitic (43.4 ± 0.50% PA), high oleic diet (4.7 ± 0.05% PA), high lauric (2.5 ± 0.05% PA), for 9 (3 x 3) weeks	↑ Total cholesterol, LDL-C	↑ Hyper-cholesterolemia ↑ CVD
Bonanome et al., 1988	Palmitic acid	Adult males, total n= 11	Randomised crossover trial	High palmitic acid (45.1% PA), high in stearic acid (8.2% PA), high in oleic acid (5.5% PA), for 9 (3 x 3) weeks	↑ Plasma total cholesterol, LDL-C	↑ Hyper-cholesterolemia ↑ CVD
Nestel et al., 1994	Palmitic acid	Hypercholesterolemic adult males, total n= 34	Randomised crossover trial	Palmitic acid, palmitoleic acid, oleic acid, through margarine and oils, (3-4% of energy), for 9 (3 x 3) weeks	↑ Total cholesterol, LDL-C (compared to oleic acid)	↑ Hyper-cholesterolemia

Table 2.6. Characteristics of studies investigating palm oil, palmitic acid, and their derivatives' health effects in animal trials.

Author(s)	Intervention	Animal model	Study type	Treatment(s)	Outcomes	Implications
Ziemlińska et al., 2021	Palm oil	C57BL/6 male mice (7 weeks old), total n= 32	Randomised trial	High palm oil diet 45% kcal from fat, soybean oil, (4%), palm oil (19.1%) (n=16), regular diet 10% kcal from fat, soybean oil (4% by weight) (n=16), for 4-12 weeks	↑ Accumulation of C16:0 and C18:1 fatty acid in livers ↑ Accumulation of C16:0 in peritoneal macrophages ↑ TLR4 and the NLRP3 inflammasome activity	↑ MASLD ↑ Hyper-cholesterolemia ↑ Hyperglycaemia ↑ CVD
Milard et al., 2019	Palm oil	C57Bl/6 male mice (8 weeks old), total n= not reported	Randomised trial	21g/100g PO, for 8 weeks	— Showed no signs of inducing inflammation	— Cholesterol homeostasis
Defo et al., 2017	Palm oil	Male Wistar rats, total n= not reported	Non-randomised trial	Palm oil diet (39.5-47.5% PA), standard diet (15% PA), for 16 weeks	↓ Seriously impairs sexual behaviour ↓ Ejaculatory activities and sperm parameters	↑ Neurodegeneration
Kübeck et al., 2016	Palm oil	C57BL/6N male mice (8 weeks old), total n= not reported	Non-randomised trial	Control diet (5% soybean oil), high-fat diet (palm oil, or lard) for 4 weeks	↑ Body mass gain	↑ Obesity
Magri et al., 2015	Palm oil	Pregnant female C57BL/6 mice, male pups, total n= not reported	Non-randomised controlled trial	Control diet (7% soy oil), palm oil diet (5% palm oil + 2% soy oil), interesterified diet (5% interesterified fats: palm kernel	↑ Visceral fat accumulation	↑ Predisposed adult obesity

				oil, palm stearin, soybean oil + 2% soy oil) for 21 days		
Wit et al., 2012	Palm oil	C57BL/6J male mice (9 weeks old), total n=not reported	Non-randomised trial	High-fat palm oil diet (177.5g PO), high-fat olive oil diet, high-fat safflower oil diet, low-fat palm oil diet (20g PO), for 8 weeks	↓ Microbial diversity ↑ Firmicutes-to-Bacteroidetes ratio ↑ Body weight gain ↑ Hepatic lipid accumulation	↑ Dysbiosis ↑ Obesity ↑ T2D
Xian et al., 2012	Palm oil	Male Sprague Dawley rats, (200-280g), total n= 24	Randomised controlled trial	Palm oil pellets (sweet potato fried in palm oil), for 6 months	↑ Tunica intima thickness in aorta ↑ Mononuclear cells infiltration ↑ Thickened subendothelial layer ↑ Disrupted internal elastic lamina	↑ Atherosclerosis ↑ Autoimmune diseases ↑ Colitis
Laugerette et al., 2012	Palm oil	Male C57Bl6/J mice (6 wk, 18 g), total n= 45	Randomised trial	Low-fat chow diet, palm oil-enriched diet, milk fat-enriched diet, rapeseed oil-enriched diet, sunflower oil-enriched diet, for 8 weeks	↑ IL-6 levels in plasma ↑ Expression of IL-1β, TLR4 and CD14 in adipose tissue ↑ Circulating LBP/sCD14 ratio	↑ Inflammation ↑ Insulin resistance ↑ T2D ↑ CVD
Ma et al., 2018	Palm oil	Nile tilapia (juvenile), total n= 480	Randomised trial	Palm oil + linseed oil diet (60% palm oil, 40% linseed oil), Fish oil + linseed oil (60% fish	Encapsulated palm oil: ↑ Lysozyme ↑ Bacillus, Paenibacillus	↑ Eubiosis ↑ Immune response

				oil, 40% linseed oil), both microencapsulated and non-capsulated, for 8 weeks	↓ Pseudoalteromonas, Roseovarius	
Aboua et al., 2012	Red palm oil (supplement)	Male Wistar rats (10-12 weeks old), total n= 54	Randomised trial	Chow diet 25g (daily), red palm oil-supplement 25g chow + 2ml (daily), red palm oil-supplement 25g chow + 4ml (daily), for 60 days	↓ Attenuate the oxidative stress-induced sperm damage due to organic hydroperoxide exposure	↑ Fertility
Kochikuzhyil et al., 2010	Palm oil	Male Wistar rats (neonatal; 28 days old), total n= not reported	Non-randomised controlled trial	Palm oil diet (40% PA), groundnut oil diet, coconut oil diet, 10% of energy from fats, for 45 days	↑ Impaired glucose tolerance ↑ Total cholesterol, triglycerides ↑ Atherogenic index ↑ Glucose levels	↑ T2D ↑ Atherosclerosis ↑ Hyperglycaemia
Boateng et al., 2006	Red palm oil	Fisher 344 male weanling rats (3-4 weeks old), total n= 32	Randomised controlled trial	Red palm oil (14% of diet), soybean oil (14% of diet), for 13 weeks	↓ Inhibition of azoxymethane (AOM) (carcinogen)-induced lesions	↓ Colon cancer
Ganafa et al., 2002	Palm oil (rapeseed oil blend)	Sprague-Dawley rats (4-5 weeks old), total n= 24	Non-randomised trial	Palm oil + rapeseed oil blend, water, PO: 5g/kg daily, for 5 weeks	↓ BSO-induced oxidative stress	↓ Hypertension
Rand et al., 1988	Palm oil	Male Wistar rats, total n= not reported	Non-randomised controlled trial	50% sunflower seed oil (6% PA), 50% palm oil (44% PA), for 8 weeks	↓ Decreased blood platelet aggregation ↓ TXA_2 formation	↓ Stroke ↓ Blood clots ↓ Arterial thrombosis

Donaldson et al., 2017	Palm oil	Coturnix coturnix japonica (Japanese) male quail (4 weeks old), total n= 57	Non-randomised trial	Palm oil (22% of diet), for 12 weeks	— No adverse effects on quail.	— Metabolic homeostasis
Wang et al., 2021	Palm oil	Honey bees (0 days old), total n= 15-30	Non-randomised trial	Palm oil (5% of diet), soybean oil (5% of diet), sucrose, casein, for 7 days	↑ Weight gain ↓ Lower survival rates ↑ Fat accumulation ↑ Upregulated expression level of TNF-α	↑ Weight gain ↑ Hyperglycaemia ↑ Inflammation
Nikouli et al., 2021	Palm oil	Atlantic salmon, total n= 200	Randomised trial	Fish oil diet (7.2% fish oil, 2.3% tuna oil), vegetable oil diet (2.9% rapeseed oil, 2.4% linseed oil, 4.2% palm oil, for 93 days	— No significant differences in gut microbiota — No significant differences in growth performance parameters - wet weight and total length	— Microbial homeostasis
Nyquist et al., 2013	Palm oil Red palm oil	Ross 308 broiler male chickens (0 days old), total n=250	Randomised trial	Palm oil, red palm oil, soybean oil, rapeseed oil, linseed oil, selenium, for 4 weeks	— Gizzard, gut health ↑ Increased early growth	— Microbial homeostasis
Patterson et al., 2014	Palm oil	C57BL/6J male mice (21 days old), total n= 54-60	Randomised trial	Palm oil, olive oil, safflower oil, flaxseed/fish oil, maize starch, sucrose, for 16 weeks	↓ Bacteroidetes at phylum level ↑ Weight gain, body fat mass ↑ Plasma glucose, leptin concentrations	↑ Obesity ↑ T2D ↑ Hepatocellular carcinoma

					↑ Hepatic expression of Fas and Srebp-1c	
Just et al., 2018	Palm oil	C57BL/6N male mice (8-10 weeks old), total n= 22	Randomised trial	Palm oil (20% of diet), lard, for 8 weeks	— Weight gain	— Obesity
Sales et al., 2018	Palm oil Hybrid palm oil	Male mice (C57Bl/6J), total n= 24	Randomised trial	Palm oil, olive oil, hybrid palm oil, (41% of energy from fats), for 8 weeks	↑ Liver fat accumulation ↑ Hepatocyte ballooning ↑ Lobular inflammation	↑ Obesity ↑ Steatosis ↑ MASLD ↑ NASH
Ghezzal et al., 2020	Palm oil	Male C57BL/6J mice (3 months old), total n= not reported.	Non-randomised trial	Palm oil (200 µL), for 1-5 days	↑ Intestinal permeability ↓ Abundance of Akkermansia muciniphila, segmented filamentous bacteria, Clostridium leptum ↑ Increased inflammatory cytokine expression	↑ Rheumatoid arthritis ↑ Neurodegeneration ↑ T2D ↑ Dysbiosis ↑ Inflammation
Ye et al., 2020	Palm oil	Sprague Dawley male rats (8 weeks old), total n= 48	Randomised controlled trial	Palm oil, leaf lard oil, rapeseed oil, sunflower oil, linseed oil, for 6 weeks	↑ Highest levels of triglycerides ↑ TC, HDL-C, and LDL-C ↑ Up-regulates the colon inflammation related gene expression levels ↓ Down-regulates the MUC2 expression levels	↑ Dyslipidaemia ↑ Hyper-cholesterolemia ↑ Colon cancer ↑ IBD ↑ Crohn's disease

					↓ Significantly decreases colon villus length and crypt depth	
Yang et al., 2020	Palm oil	Duroc × (Landrace × Yorkshire) Pigs (21-28 days old), total n= 18	Randomised controlled trial	Palm oil (6% of diet), soybean oil (6% of diet), encapsulated palm oil (7.5% of diet), for 28 days	↑ Encapsulated palm oil improved digestibility	↑ Digestion ↓ Malabsorption
Ruan et al., 2021	Palm oil	Sprague Dawley rats (8 weeks old), total n= 30	Randomised trial	Unheated palm oil, heated palm oil, unheated canola oil, heated canola oil, (23% of diet), for 3 months	↑ Heated palm oil stimulated the proliferation of Lactobacillus and Roseburia ↑ Induced the expression of IL-22 in gut.	↑ Eubiosis
Ye et al., 2021	Palm oil	Sprague Dawley rats (2-8 weeks old), total n= 48	Randomised trial	Palm oil, leaf lard oil, rapeseed oil, sunflower oil, linseed oil, for 6 weeks	↑ Higher level of serum LPS level ↑ Inflammatory cytokine expression level ↑ Significantly higher TLR4 and MyD88 expression level in the ileum tissue	↑ MetS ↑ Liver disease ↑ CVD ↑ Hyper-cholesterolemia
Selmin et al., 2021	Palm oil	C57BL/6J mice (<30 days old), total n= 10	Non-randomised trial	Western diet (11% palm oil), n-6 high-fat diet (20% soybean oil), for 10-16 weeks	↓ Colonic COX-2 expression ↓ Accumulation of colonic	↓ Colorectal cancer ↓ Colonic IBD

					inflammatory and hyperplastic lesions	
Conlon et al., 2020	Oil palm preparation (OPP)	Sprague-Dawley rats (~200g weight), total n= 56	Randomised trial	Western diet, oil palm preparation added to feed (OPP-F), oil palm preparation added to drink (OPP-D), high amylose maize starch added to feed, green tea added to drink, (n=10-13/group), for 4 weeks	OPP-D: ↑ Caecal digesta mass, caecal digesta concentrations of total SCFA, acetate and propionate OPP-F: ↑ Caecal butyrate concentration, caecal digesta abundance of bacteria (Faecalibacterium prausnitzii, Akkermansia muciniphila and Ruminococcus gnavus)	↑ Eubiosis ↑ Immune response
Budin et al., 2009	Tocotrienol-rich fraction (TRF) of palm oil	Male Sprague Dawley rats, total n= 23	Non-randomised trial	Palm oil tocotrienol-rich fraction (TRF), non-TRF fraction, oil, for 8 weeks	TRF: ↑ Improved blood glucose ↓ Improved dyslipidaemia ↓ Oxidative stress markers	↓ Oxidative stress ↓ T2D
Cheng et al., 2015	Palmitic acid	Male C57BL/6J mice (10 weeks old, body weight: 22.74 ± 3.22g), total n= 24	Randomised trial	Palmitic acid (25 pmol), for <20 days	↑ Central leptin resistance ↑ Impaired hypothalamic leptin signalling (JAK2-STAT3, PKB/Akt-FOXO1) ↑ Pro-inflammatory	↑ Obesity ↑ Inflammation

					response (TNF-α, IL1-β, IL-6 and IκBa) in the medio basal hypothalamus and paraventricular hypothalamic nuclei	
Ye et al., 2019	Palmitic acid	Zebrafish, total n= not reported	Randomised controlled trial	Palmitic acid (1.6 mM + 1.6% bovine serum albumin)	↑ Free fatty acid receptor desensitization — Did not induce endoplasmic reticulum (ER) stress in the digestive tract	↑ Dysbiosis — Glucose metabolism ↑ Inflammation
Xin et al., 2020	Palmitic acid	Sprague Dawley rats (6 weeks old), total n= 24	Randomised trial	High-fat diet (88% standard diet, 10% lard (23.8% palmitic acid), and 2% cholesterol), folic acid, for 16 weeks	↑ Lobular inflammation ↑ Hepatocellular ballooning ↓ Decreased microbial diversity	↑ Obesity ↑ Dyslipidaemia ↑ Steatosis ↑ MASLD ↑ NASH
Saraswathi et al., 2020	Palmitic acid	Male wild type C57BL/6 mice, total n= 6	Randomised trial	Palmitic acid high-fat diet (3% of diet), lauric acid high-fat diet (3% of diet), for 12 weeks	↑ Adipose tissue inflammation ↑ Systemic insulin resistance ↑ Liver injury	↑ Obesity ↑ T2D ↑ CVD ↑ MetS
Liu et al., 2019	Palmitic acid	Kunming male mice, total n= 50	Non-randomised controlled trial	Decaisnea insignis seed oil (DISO) (palmitic acid 12.25%, palmitoleic acid 55.25%, oleic	↑ Relative abundance of Lactobacillus ↑ Intestinal permeability	↑ Eubiosis

				acid 28.74%), low (3g/kg), med (6g/kg), high (12g/kg), for 12 weeks		
Zhang et al., 2019	Palmitate	C57Bl/6J male mice (6 weeks old), total n= 72	Randomised trial	100 g/kg palmitate (10% of diet, 300 g/kg fat (30% of diet), for 22 weeks	↑ Hepatic inflammation ↓ Gut microbial diversity ↑ Impaired recognition and spatial memory	↑ Fibrosis ↑ Cirrhosis ↑ Obesity ↑ Dysbiosis ↑ Neurodegeneration
Jena et al., 2020	Palmitate	C57BL/6 male mice, total n= 12	Randomised trial	Palmitate diet (29% palmitate, fructose, cholesterol), control diet (6.2% fat), for 5 months	↑ Liver fat accumulation ↑ RORγ and IL-17A signalling (liver, brain) ↑ Encephalitis	↑ Hepatic steatosis ↑ Hepatitis ↑ Neurodegeneration
Razolli et al., 2020	Palmitate	Mice (Swiss) (5-8 weeks old), total n= 10	Randomised trial	Palmitate (2.0 mM), for 4 weeks	↑ PCSK1/PC1/3 expression ↑ C1/3, PC2, CPE, PRCP	↑ Obesity
Reichardt et al., 2017	Palmitate	C57Bl/6 mice (6 weeks old), total n= not reported	Non-randomised controlled trial	Western diet (35% palmitate), purified regular diet (16.9% fat) for 3, 6, 9 and 12 weeks	↑ Nitrergic myenteric neuro-degeneration in the proximal colon ↑ TLR4 signalling	↑ IBD ↑ Inflammation

Anitha et al., 2016	Palmitate	C57BL/6 male mice (8 weeks old), total n= not reported	Non-randomised controlled trial	High-fat diet (60% kcal fat, palmitate), control diet (18% kcal fat), for 13 weeks	↑ Circulating LPS ↑ TLR4, JNK signalling pathways	↑ Hyperlipidaemia ↑ Dysbiosis
Wan et al., 2017	Sn-2 palmitic acid	Male Sprague-Dawley rats, total n= 27	Non-randomised trial	Sn-2 palmitic acid diet (56.3% sn-2 PA), sn-2: docosahexaenoic acid (DA) diet (45% DA) (90:10), sn2 (Sn2PA: arachidonic acid diet (47% ARA) (90:10), for 6 weeks	↑ Lipid metabolism ↑ Abundance of Lactobacillus	↓ CVD ↓ T2D ↑ Eubiosis
Wan et al., 2016	Sn-2 palmitic acid	Sprague Dawley male Rats (21 days old), total n= 36	Non-randomised trial	Low sn-2 diet (12.1% PA), medium sn-2 diet (40.4% PA), high sn-2 (56.3% PA), for 4 weeks	High sn-2 PA: ↓ Fatty acid soaps and calcium	↑ Digestion ↓ Malabsorption
Idris et al., 2014	Palm Vitamin E Oil palm phenolics	Male New Zealand White rabbits (5 months old), total n= 32	Non-randomised controlled trial	High-fat atherogenic diet (AD), AD + palm vitamin E (VIT E), AD + oil palm phenolics (OPPh), AD + VIT E + OPPh, for 100 days	↓ Development of atherosclerotic lesions	↓ Stroke ↓ Coronary heart disease
Boon et al., 2013	Palm olein Red palm olein	Spontaneously hypertensive rats, Wistar-Kyoto rats, (3 weeks old), total n= 24	Randomised controlled trial	Palm olein (39.5% PA), red palm olein (39.1% PA), super olein (35.6% PA), 15% from fat, for 15 weeks	Palm olein, super olein: ↑ Bradycardia ↓ Heart size reduction Red palm olein: ↑ Improved blood lipid profiles	↑ Myocardial infarction ↓ Atherosclerosis

Table 2.7. Characteristics of studies investigating palm oil, palmitic acid, and their derivatives' health effects in vitro.

Author(s)	Intervention	Cell type	Study type	Treatment	Outcomes	Implications
Nesaretnam et al., 1995	Tocotrienol-rich fraction (TRF) of palm oil	Human (breast) cancer MDA-MB-435 cell line	Non-randomised trial	Tocotrienol-rich fraction (TRF) of palm oil, 1 mg/mL, 500 ug/mL, 250 ug/mL, 200 u/mL, 180 ug/mL, 160 u/mL, 140 u/mL, 120 u/mL, 60 u/mL, 30 u/mL	↓ Inhibited cell growth positively correlates with TRF concentrations	↓ Breast cancer proliferation
Laugerette et al., 2012	Palmitic acid	Mouse (embryonic fibroblast) 3T3-L1 cells	Randomised trial	Dulbecco's modified eagle medium with or without 0.1 µg/ml LPS Experiment a: DMEM; 5 ug/ml insulin + one of following FAs: myristic, palmitic, linoleic, linolenic acids (+ 50 µmol BSA) Experiment b: 1 of the FAs with 0.1g/ml of LPS from Escherichia coli (E. coli), for 24 hours	↑ IL-6 accumulation ↑ Exacerbates IL-6 production with LPS	↑ Inflammation ↑ T2D ↑ CVD
Ghezzal et al., 2020	Palmitic acid	Human (colon) adenocarcinoma Caco-2/TC7 cell line	Non-randomised trial	Palmitic acid, oleic acid, for 1-5 days	↑ Disrupts cell junctions ↑ Increases paracellular permeability ↑ Induces IL-8 expression	↑ Rheumatoid arthritis ↑ IBD ↑ Inflammation

					and secretion ↑ Intestinal barrier defect	
Mayneris-Perxachs et al., 2021	Palmitic acid	Human (liver) primary hepatocytes	Non-randomised trial	Palmitic acid (200 µM PA) following iron treatment, for 24 hours	↑ Triglyceride accumulation	↑ Dyslipidaemia
Dong et al., 2020	Palmitic acid	Human (liver) hepatic stellate cells (HSCs)	Non-randomised trial	Palmitic acid (200 µm), for 6h	↑ Stimulates NLRP3 inflammasome activation (TLR4-NF-κB signal pathway)	↑ Inflammation ↑ Insulin resistance ↑ T2D ↑ CVD ↑ Hepatocellular carcinoma
Gowd et al., 2018	Palmitic acid	Human (liver) HepG2 cells	Non-randomised controlled trial	Palmitic acid (0.2 mM PA), for 24 hours	↑ Over-production of reactive oxygen species (ROS) ↑ Mitochondrial dysfunction	↑ Oxidative stress ↑ CVD ↑ Alzheimer's ↑ Parkinson's
Park et al., 2014	Palmitic acid	Human (HeLa cell derivative) Chang cells	Non-randomised controlled trial	PA (conjugated to 5% BSA), for 24 hours	↑ Cytotoxicity ↑ Mitochondrial dysfunction ↑ Endoplasmic reticulum stress	↑ Fibrosis ↑ Cirrhosis ↑ Hepatocellular carcinoma ↑ Alzheimer's ↑ Parkinson's
Ricchi et al., 2009	Palmitic acid	Human (liver, liver cancer, uterine cervix) HepG2,	Non-randomised trial	Palmitic acid (0.33 + 0.66 mM), oleic acid (0.66 + 1.32 mM), for 24 hours	↑ Apoptosis ↑ Increased PPARα expression	↑ MASLD ↑ Fibrosis ↑ Cirrhosis

		HuH7, WRL68 cells			↑ Impaired insulin signalling	↑ Hepatocellular carcinoma
Joshi-Barve et al., 2007	Palmitic acid	Human (liver) HepG2 cells	Non-randomised trial	Palmitic acid complexed with bovine serum albumin (BSA), for 12 hours	↑ Apoptosis ↑ IL-8 production, via activation of JNK/AP-1, and NF-κB	↑ MASLD ↑ Fibrosis ↑ Cirrhosis ↑ Hepatocellular carcinoma
Zhou et al., 2021	Palmitic acid	Human (faecal) inocula	Non-randomised trial	Palmitic acid, stearic acid, lauric acid, myristic acid	— Did not promote harmful bacteria (Bilophila, Fusobacterium and Dialister)	↑ Eubiosis
Wu et al., 2014	Palmitic acid	Rat (aortic) vascular smooth muscle cells (VSMCs)	Non-randomised trial	Palmitic acid (+ BSA), for 10 minutes	↑ CRP, TNF-α and iNOS expression	↑ T2D ↑ Inflammation ↑ Atherosclerosis
Sergi et al., 2021	Palmitic acid	Human (skeletal muscle) primary myotubes, myoblasts	Non-randomised trial	Palmitic acid (500 µM), lauric acid, for 24 hours	↑ NF-κB activation ↑ Mitochondrial fragmentation ↓ Mitochondrial membrane potential	↑ Inflammation ↑ Oxidative stress ↑ Alzheimer's ↑ Parkinson's
Sergi et al., 2021	Palmitic acid	Human (skeletal muscle) primary myotubes	Non-randomised trial	Palmitic acid (500 µM), glucose, eicosapentaenoic acid (EPA), for 24 hours	↑ NF-κB signalling ↑ Deleterious effects on mitochondrial health	↑ Inflammation ↑ Hyperglycaemia ↑ Oxidative disease ↑ Neurodegeneration

Rampersaud et al., 2020	Palmitic acid	Human (placenta) HTR8/SVneo cells Human (aortic) primary SMCs Human (uterine) microvascular endothelial cells Human (embryonic kidney) (HEK)-293T cells	Non-randomised trial	Palmitic acid (20 mM): BSA (30%) (2:1) to, palmitic acid Oleic acid (20 mM): BSA (30%) (2:1), for 24 hours	↑ IL6, CXCL8 ↑ Vasoactive (EDN1) ↑ Fibrogenic (SERPINE1) proteins (all increased in serum of obese patients).	↑ Inflammation ↑ Blood clots ↑ Fibrosis
Razolli et al., 2020	Palmitate	Human hypothalamic neurons	Randomised trial	Palmitate (2.0 mM), for 4 weeks	↑ PC1/3, PC2, CPE, PRCP expression	↓ Obesity
Laine et al., 2007	Palmitic acid	Human (histiocytic lymphoma) isolated U937 monocytes	Non-randomised controlled trial	Palmitic acid, lauric acid, oleic acid, linoleic acid, eicosapentaenoic acid, arachidonic acid, (sodium salts, 150 uM), for 48 hours	↑ IL-8, IP-10, MCP-1, COX-2, and MIG expression ↑ Induced NF-κB	↑ Inflammation
Tafti et al., 2019	Palmitic acid Palmitoleic acid	Human (bacterial cell) B. fragilis ATCC 2374 B. thetaiotaomicron ATCC 10774	Non-randomised trial	Palmitic acid (20 mg/ml) Palmitoleic (20 mg/ml), for 12-24 hours	Palmitic acid ↑ B. fragilis and B. thetaiotaomicron growth ↑ Outer membrane vesicles (OMV) production	↑ Eubiosis ↑ Immune response

Lee et al., 2021	Palmitate	Human (colon) adenocarcinoma Caco-2 cells	Non-randomised trial	Palmitate (2.5, 5, and 7.5 mM), BSA, for	↑ Increased the reactive oxygen species (ROS) levels in the colon epithelium	↑ Oxidative stress ↑ IBD
Rumora et al., 2019	Palmitate	Mouse (spinal column) primary dorsal root ganglion (DRG) neurons	Non-randomised trial	Palmitate (31.25 - 250 µm), for 24 hours, palmitate: oleate (1:1) (125: 125 µM), palmitate: oleate (2:1) (250: 125 µM), for 12 hours	↑ Mitochondrial transport impairment	↑ Alzheimer's ↑ Parkinson's
Yang et al., 2013	Palmitate	Mouse (skeletal muscle) C2C12 cells	Non-randomised trial	Palmitate, oleate, BSA, for 24-36 hours	↓ Akt phosphorylation ↑ Glucose uptake ↑ GLUT4 expression ↑ Myotube loss ↓ Akt phosphorylation ↓ Expression of FNDC5, CTRP15 and FGF21 genes	↑ Apoptosis ↑ T2D ↑ Insulin resistance ↑ Atrophy ↑ Neurodegeneration
Wang et al., 2018	Palmitate	Pig (liver) hepatocytes (injected with LPS)	Non-randomised trial	Palmitate (2 mM), for 24 hours	↑ Production (hepatic) of acetyl coenzyme A, pyruvate, citrate, oxaloacetate, lactate ↑ MAS activity ↑ Hepatic MDH2 and GOT2 hyperacetylation	↑ Nutrition-related cancers ↑ MASLD ↑ Pancreatic cancer
Ahmad et al., 2018	Palmitate	Human (monocytic leukaemia) THP-1,	Non-randomised trial	Palmitate (200 mM) and TNF-a (10 ng/ml), for 8 hours	↑ TNF-a activation ↑ Elevated NF-kB/AP-1	↑ Inflammation

		primary human monocytes, THP1-XBlue cells			activity ↑ CCL2	↑ Nutrition-related cancers ↑ Nutrition-related cancers
Lee et al., 2021	Palmitate	Human (colorectal adenocarcinoma) caco-2 cells	Non-randomised trial	Palmitate (2.5, 5, 7.5 mM) for 6-10 hours	↑ Reactive oxygen species (ROS) levels in the colon epithelium (dose-dependent manner)	↑ Oxidative stress
Hasan et al., 2019	Palmitate	Human (monocytic leukaemia) THP-1 cell line n=58 adults	Non-randomised trial	Palmitate (200μM), TNF-α (10 ng/mL), for 24 hours	↑ TNF-α activation ↑ Excessive production of IL-8	↑ Obesity ↑ Inflammation
Reichardt et al., 2017	Palmitate	Mice (gastrointestinal) IM-FEN cells	Non-randomised controlled trial	Palmitate (100mM), lipopolysaccharide (LPS), for 24 hours	↑ Palmitate and lipopolysaccharide (LPS) acted synergistically to induce enteric neurodegeneration	↑ Enteric neurodegeneration
Anitha et al., 2016	Palmitate	Mouse enteric neurons, (IM-PEN) cell line	Non-randomised trial	PA (0.5, 1 mmol/L) for 24 hours	↑ TLR4-dependent neuronal loss	↑ Inflammation ↓ Delayed intestinal motility
Ishii et al., 2015	Palmitate	Human (liver) HepG2 cells	Non-randomised trial	Sodium palmitate, for 2 hours	↓ Phosphorylation of key insulin signalling molecules: insulin receptor, insulin receptor substrate-1, Akt	↑ Insulin resistance ↑ T2D

Wang et al., 2021	Calcium palmitate (CP)	Human (gastrointestinal) infant gut microbiota	Non-randomised trial	Calcium palmitate (0.03 or 0.01 mg/ml), for 16-18 hours	↓ Calcium palmitate was shown to inhibit the growth of bifidobacteria and faecalibacteria	↑ Dysbiosis

2.6 Discussion

This systematic review evaluated the literature on the effects of palm oil, palmitic acid, and their derivatives on various components of metabolic health. Despite palm oil's increasing prevalence especially within Westernised diets, their exact roles within chronic disease appear conflicting, or unclear. The aims and objectives of this systematic review were to analyse and synthesise the studies investigating palm oil, palmitic acid, and its derivatives across various, distinct (yet related) populations (human, animal, in vitro), providing a synopsis of their reported outcomes on several cardiometabolic health risk factors. It was evident that the majority reporting unfavourable health effects, particularly for palm oil and palmitic acid/palmitate. In human infants, sn-2 palmitate/β-palmitic acid demonstrated favourable effects on infant general growth, neurodevelopment, bone health, intestinal diversity, as well as fatty acid and calcium absorption. In human adults, the majority of studies reported unfavourable effects on chronic disease related risk factors in the presence of palm oil and palmitic acid/palmitate, chiefly abnormalities/imbalances in cholesterol, inflammation, gut microbial dysbiosis, insulin resistance, oxidative stress, mitochondrial dysfunction, and triglyceride/lipid accumulation. Investigations within animal models and in vitro trials provided insight into the feasible effects of palm oil and palmitic acid/palmitate on human health, in vivo. Animal models mostly shared the outcome effects of human adult interventions (favourable, neutral, and unfavourable), whereas outcomes in vitro were almost exclusively unfavourable, observing disproportionate inflammatory cytokine production, gut microbiome dysbiosis, neurodegeneration, as well as widespread cell disruption and cell death (apoptosis). Although, it is important to consider that effects observed in vitro may not accurately depict health effects in vivo; due to the omission of various complex systemic, physiological and biochemical interactions with, and responses to, stimuli (including but not limited to, other foods/nutrients). Understanding the effect of palm oil, palmitic acid and their derivatives has potential for health recommendations.

Palm oil and palmitic acid were observed to possess hypercholesterolaemic properties, inducing increased total (serum) cholesterol, LDL cholesterol and apolipoprotein B levels, key risk factors in the development of atherosclerosis (Mortensen et al., 2023; Singh & Prabhakaran, 2024). Cardiovascular disease(s) in particular were strongly associated with palm oil and palmitic acid intake, via induced increased factor VII coagulant activity, circulating harmful

sphingolipids, and higher plasma cholesterol ester transfer protein (CETP) activity, proposed biomarkers of cardiovascular disease (Ansell & Hobbs, 2006; Bernardi & Mariana, 2021; Kovilakath et al., 2023).

Unexpectedly, palm oil was found to have minimal direct effects on inflammatory cytokines within human adults, although research was limited to one study (Engelberts et al., 1993). However, investigations in animal and cell models provided evidence to suggest that palm oil due to its high content of palmitic acid, plays a prominent role in chronic inflammation. A few of the palmitic acid induced inflammatory mediators includes cytokines, chemokines, interleukins and tumour necrosis factors (*TNFα, IL-1, IL1-β, IL-6, IL-8/CXCL8, MIG/CXCL9, MCP-1/CCL2, IP-10, IL-17A*), via toll-like receptor proteins; toll-like receptor 2 (TLR-2) and toll-like receptor 4 (TLR4), as well as the activation of key transcription factor nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB). Excessive levels of inflammatory cytokines are a risk factor for several chronic metabolic diseases, including obesity, type 2 diabetes, cardiovascular disease, and certain cancers (Greten et al., 2019; Henein et al., 2022; Rohm et al., 2022).

Surprisingly, none of the reviewed sources on human adults investigated the effects of palm oil or palmitic acid within the gut microbiome. Nevertheless, investigations within animal trials observed palm oil inducing various unfavourable effects on gut microbiome health, predominantly decreases in microbial diversity (dysbiosis), and an increase in intestinal permeability, both implicated in various diseases including rheumatoid arthritis (RA), neurodegenerative diseases, and diabetes (Macura et al., 2024). Palmitic acid also increased reactive oxygen species (ROS) levels in the colon epithelium, contributing to oxidative stress, strongly associated with increased risks of inflammatory bowel diseases, as well as colorectal (colon) cancer (Bardelčíková et al., 2023).

Palm oil and palmitic acid also demonstrated several adverse contributions to the pathogenesis of various other disorders/diseases. Concerning hepatic health, palmitic acid induced lipid/triglyceride accumulation (dyslipidaemia), typically associated with metabolic dysfunction-associated steatotic disease (MASLD), or non-alcoholic steatohepatitis (NASH) (Caldwell et al., 2010; Kawano & Cohen, 2013; Liu et al., 2010). Palmitic acid was also observed to impair insulin sensitivity, through the elevation of glucose concentrations (hyperglycaemia), as well as leptin concentrations, associated with insulin resistance and type

2 diabetes, or obesity, respectively (Obradovic et al., 2021). Lastly, palmitic acid is suggested to play a role in neurodegenerative disorders, most notably via impaired recognition and spatial memory, closely associated with Alzheimer's disease (Silva & Martínez, 2023).

Beneficial health effects of palm oil were observed predominantly with crude/red palm oil, (palm oil in its most natural state), or fractions/extractions of palm oil containing similar nutritional compositions (tocotrienol-rich fraction, palm vitamin E, oil palm preparation). Evidently, these forms of palm oil mitigated many of the effects typically associated with a high-fat diet, such as the amelioration of lipid accumulations (dyslipidaemia), oxidative stress, and hypertension, increased HDL cholesterol and apolipoprotein-AI concentrations, as well as the inhibition of metastasis and growth of cancer cells, particularly in breast and colon cancers. Most likely owing to their high vitamin A and/or vitamin E content, well-established beneficial nutrients (Alouache, 2025; Fatima et al., 2025; Pyo & Kwon, 2025). Unfortunately, red palm oil is not commonly consumed/used as a cooking oil in Westernised diets, with most everyday foods (particularly processed foods) containing refined version(s) of palm oil. Additionally, in the formation of refined palm oil, the majority of vitamin A (primarily carotenoids, or β-carotene) and vitamin e (tocopherols, tocotrienols) content is removed during the production process (Manorama et al., 1996; Oguntibeju et al., 2009). Continued advancements in technology may allow for greater amount(s) of these bioactive compounds being retained/preserved during processing, or increase their bioavailability through methods such as encapsulation, both as part of crude/red palm oil, or in an isolated form, post-extraction (Madoromae et al., 2025; Olivares-Tenorio et al., 2024). Exploration of bioactive compounds isolated directly from red palm oil, when compared to both red, and/or processed palm oil as a whole, may provide valuable insight into their effects on health. Peripheral studies have explored this, however via components such as synthetic vitamin A, or synthetic carotenoids (Souganidis et al., 2013; Van Stuijvenberg et al., 2001). Therefore, the majority of palm oil being consumed in the United Kingdom is more than likely refined, and lacking in these nutrients.

Interestingly, reviewed evidence suggests that the various unfavourable effects of palm oil, could principally be attributable to palmitic acid alone. Based on the characteristic tables, studies investigating palm oil observed varying health effect outcomes, including cholesterol, inflammation, insulin resistance, and gut microbiome status, with favourable, neutral, and unfavourable outcomes. Palmitic acid however, elicited a much higher proportion of

unfavourable health outcomes. While palm oil is the largest source of palmitic acid (~42-47%) and contains a high content of saturated fatty acids (~50%), it also contains comparable amounts of unsaturated fatty acids (~50%), such as oleic acid (~41-43%) (Chapter 4). Oleic acid is well-established in providing generally beneficial effects on cholesterol (Lopez-Huertas, 2010), inflammation (Santa-María et al., 2023), insulin sensitivity (Zhao et al., 2024) and the gut microbiome (Zhao et al., 2019). Additionally, oleoylethanolamide (OEA), derived from oleic acid, has been reported to possess 'atheroprotective' (Fan et al., 2014), and anti-oxidant effects (Tutunchi et al., 2023). Therefore, it would be reasonable to assume that the various unfavourable effects of palmitic acid on metabolic health are somewhat attenuated by unsaturated fatty acids, particularly oleic acid when consumed as whole palm oil. Oleic acid reportedly lessens, or modulates many detrimental health effects caused by saturated fatty acids. Oleic acid promotes the esterification of fatty acids into triglycerides, reducing the number of toxic FFA's in the body (Listenberger et al., 2003). Additionally, oleic acid promotes the formation of anti-inflammatory cytokines, inhibiting the production of inflammatory cytokines, and the activation of inflammatory pathways, such as the NF-κB pathway (Santa-María et al., 2023). Oleic acid can also regulate cholesterol metabolism, reducing LDL-cholesterol levels via downregulation of cholesterol synthesis (cholesterologenesis) (Natali et al., 2007), as well as protect against insulin resistance and cancer progression, via regulation of gene expression associated with the IRS1/P13K signalling pathway (López-Gómez et al., 2020). These findings align with the well-established knowledge that high-fat diets, particularly saturated fat intake, is strongly associated with various risk factors of chronic disease, and diets should be focused more on increased unsaturated fats (Hooper et al., 2015). However, perhaps palmitic acid and oleic acid share a unique relationship, where dietary requirements necessitate a balanced ratio of the two acids for optimal metabolic health. Several studies have examined their associations, although further research is needed as to how these two fatty acids interact at the metabolic levels within the cell (Ahn et all., 2013; Chen et al., 2018; Gao et al., 2009).

Further strengthening these interpretations are the favourable outcomes observed in palm olein (high in oleic acid). Palm olein reportedly inhibits LDL-cholesterol, increases HDL-cholesterol, apolipoprotein A-1 and A-II levels, as well as induces negligible effects on inflammatory markers, insulin resistance, glucose homeostasis - markers of reduced risks of metabolic syndrome and coronary artery disease (Karjalainen et al., 2020; Yi et al., 2012). Perhaps due not only to high oleic acid content, but also, its' triacylglycerol structure. Palm olein contains ~37% saturated fat and ~56% unsaturated fat content, predominantly palmitic acid (~37%),

and most importantly, oleic acid (~45%), respectively (Sampaio et al., 2011). Moreover, 60-70% of the oleic acid content in palm olein is at the sn-2 position of the glycerol backbone (Wan Rosnani et al., 2008), leading to improved fat digestion and absorption (Mehrotra et al., 2019). Effective lipid digestion, especially unsaturated fatty acids, is crucial in reducing the risks of metabolic disease, as it can have favourable effects on inflammation, insulin resistance, and gut microbiota, as well as support energy homeostasis regulation, and cellular structure/function (Omer & Chiodi, 2024). Conversely, palm stearin possesses higher palmitic acid content (~46%), in addition to lower oleic acid content (~36%), and (although limited to one study) had adverse effects on total cholesterol and HDL-cholesterol concentrations.

Over the past decade, there has been extensive research on the effects of sn-2 palmitate (β-palmitic acid) on infants. However, there are no available studies within the last few decades investigating the direct effects of dietary palm oil, crude, processed or otherwise, on human infants. Perhaps due to concerns that palmitic acid/palmitate found within both naturally occurring and refined palm oil is present at the sn-1 and sn-3 positions (Mattson & Lutton, 1958). Sn-1 and sn-3 palmitic acid has been associated with adverse health effects in infants, particularly digestive issues, and the formation of insoluble calcium soaps (Havlicekova et al., 2015). While not investigated in human infants, red palm oil has been investigated in the maternal diet, showing an increase in vitamin A carotenoids in the mothers' breastmilk (Canfield et al., 2001).

Sn-2 palmitate/β-palmitic acid intake, either naturally through breast milk or artificially manipulated (using enzymatic interesterification) demonstrated opposing effects to palmitic acid typically found in foods (sn-1, sn-3), mostly within infant trials. Growing interest in the research of sn-2 palmitate on infant health specifically is perhaps due to a greater understanding of the natural exposure infants have to large amounts of palmitic acid (16:0). Human infant diets typically require 50% energy from fats, in which 10% of the daily energy is provided (from breast milk, or formula) by sn-2 16:0 (Innis, 2011). The developing human foetus accumulates large amounts of 16:0 fatty acid during the third trimester of gestation through the process of foetal lipogenesis (Herrera & Ortega-Senovilla, 2010). Such an accumulation of 16:0 fatty acid particularly assists in supporting the early growth and development of organs such as the brain and adipose tissues (Desoye & Herrera, 2021). Hence, the palmitic acid derivative, sn-2 palmitic acid/β-palmitate, present endogenously during pre- and post-birth, as well as those supplied exogenously through diets, play an important role to meet the demand

of metabolic energy during the early growth and development stage of infants (Innis, 2016). These wholly beneficial outcomes suggest that dietary intervention through manipulated (sn-2 palmitate/β-palmitic acid) infant formulae is necessary to mitigate the potential, in theoretical cases evident, harmful effects of sn-1 and sn-3 palmitic acid on human infants. An increase in the diversity of gut microflora in human infants, alongside the bifidogenic factor of sn-2 palmitate/β-palmitic acid is a crucial finding identified in this review, as gut microbiome health status not only plays a major role in the early life of the infant, but also has long-term health consequences during adulthood (Yao et al., 2021).

Discernibly, breast milk/formula is seldom investigated within human adult studies. However, although minimal, investigations of sn-2 palmitate/β-palmitic in human adults observed favourable effects on cholesterol. Favourable health outcomes in animal trials (again, minimal), reported favourable effects on the lipid metabolism, and the abundance of beneficial gut bacteria. While is it possible for sn-2 palmitic acid to be present in the human adult diet (in small amounts), refined palm oil used in pre-packaged or processed foods does not utilise this positioning, perhaps due to shelf-life, taste/texture, or cost (commercial viability).

In summary, several lines of evidence suggested a potential relationship between palm oil, palmitic acid and various risk factors of chronic disease, predominantly type 2 diabetes, cardiovascular disease, stroke and certain cancers. It was further evident that red palm oil, palm olein, as well as sn-2 palmitate/β-palmitic acid induces various beneficial metabolic health effects. More research is needed to i) assess the dietary intake of palm oil/palmitic acid, ii) understand the chemical composition of palm oil and its presence in common foods, iii) establish the cause-and-effect relationship between palm oil and its chemical constituents at the whole organism, organ/tissue and cell levels.

2.6.1 Limitations of the available literature

There are considerable gaps in the available evidence. Firstly, no literature on human adults investigated the effects of palm oil, palmitic acid, or any of their derivatives on the health of the gut microbiome. Secondly, outside of human infant studies, the number of available sn-2 palmitate/β-palmitic acid studies in human adults, animals or in vitro, is very low. The risk of bias assessment(s) concluded that several studies originating from Malaysia (second leading producer of palm oil, worldwide), or studies where the author, affiliation, or sponsor has a

vested interest in palm oil from a business perspective, mostly reported neutral or favourable effects; a high likelihood of bias or influence(s) in both the measurement and reporting of outcomes (perhaps knowingly, or unconsciously). Surprisingly, no studies originated from the largest producer of palm oil, Indonesia. Inherent limitations of animal and in vitro trials should also be noted. In animal trials, the main limitations are environmental differences to how humans interact with foods day-to-day, i.e., rapidly induced effects of one intervention within animal models, compared to a varied composition of naturally occurring dietary effects, over time. For in vitro trials, their intrinsic simplification of investigations within controlled, artificial environments, can lead to false-positive results. Available review literature appears both limited and deficient. Solely analysing animal studies due to lack of human data (Syarifah et al., 2020), self-reported low quality of evidence (Ismail et al., 2018), alongside neutral or favourable effects being attributed to palm olein alone (Voon et al., 2019); raising concerns in the validity, reliability, and scope of their conclusions.

2.6.2 Strengths and limitations of the review

This review provides a comprehensive overview of a large number of studies available within the literature, covering a wide range of related interventions, populations, and outcomes to provide greater insight into the effects of palm oil, palmitic acid and their derivates on metabolic health, as well as their role in the risks of chronic disease. Critical appraisal of the full text of studies, alongside four different risk of bias assessment(s) increased review reliability and validity, as well transparency of certain research limitations/practices. Review conclusions were based on the outcomes within each study as a consensus, presenting favourable, unfavourable, and neutral health effects to produce an unbiased, objective summary of the current evidence. Although the strengths are evident, this review is not without limitations. Firstly, there were challenges in generalising a high number of diverse health outcomes, more specifically, the summarisation, in addition to postulation of findings and their implications. Perhaps focusing on one intervention, or one specific health outcome, such as palmitic acid's effects on human adult inflammation, may have produced a more cohesive review of the literature, and allowed for meta-analysis (although, this would have not met the reviews aims and objectives). Similarly, if review methodology was to be adapted or replicated in future research, the search strategy could be less restrictive in its approach, including specific health outcomes such as 'cholesterol', or 'type 2 diabetes', in place of broader terms such as

‘metabolic health’ could aid in additional publication identification. Lastly, publications from this year (2025) were not included.

2.7 Conclusions

Based on this review of the available literature, the results conclude that the majority of studies provide evidence to suggest that palm oil, or more specifically palmitic acid intake, contributes significantly to the deterioration of various components of metabolic health. Predominantly inducing disruption(s) of cholesterol homeostasis, excessive inflammatory cytokine production/chronic inflammation, hyperglycaemia, impaired insulin signalling/insulin resistance, oxidative stress, hepatic lipid (triglyceride) accumulation, gut microbial disturbance(s) and dysbiosis, as well as neurodegeneration - key risk factors in various chronic diseases associated with metabolic syndrome, such as type 2 diabetes, cardiovascular disease(s), stroke, and nutrition-related cancers. Oleic acid, a conceivable ameliorator of adverse effects observed in palmitic acid interventions, is in high content in both palm oil and palm olein - although the extent of their various physiological interactions is not fully understood. Further research is needed to evaluate the cause-and-effect relationship between the palm oil and its derivatives at the system, organ and cell levels.

2.7.1 Conflicting interests and funding statement

The author declares no conflicting or competing interests, nor any funding.

Chapter 3. Awareness and perceptions of the health effects of palm oil and palmitic acid

3.1 Chapter Overview

Palm oil/palmitic acid are abundantly present in the food consumed worldwide and there are growing concerns on their potential health risks. However, little is known of the public perceptions, specifically its effects on health and the extent of intake of palmitic acid in different dietary habits (omnivores, plant-based, vegans). The aim of this research was to assess the awareness of the United Kingdom public of palm oil and palmitic acid; their existence, their prevalence, as well as their health effects. A total of 251 members of the United Kingdom public completed a questionnaire containing demographic information, details regarding their anthropometry, exercise frequency/intensity, sleeping patterns and dietary habits, as well as their perceptions, behaviours and beliefs towards palm oil and palmitic acid. A total of 62 of these 251 respondents also provided a 1-week (4-day) food diary. Respondent survey data was collected via the survey website Qualtrics, as well as through Microsoft Word document. Dietary intake information was calculated using the nutritional analysis software Nutritics, cross-referenced with The McCance & Widdowson's The Composition of Foods Integrated Dataset 2021 to obtain palmitic acid intake. IBM SPSS Statistics was used for all statistical analysis. The majority of respondents are aware of the existence of palm oil within their foods (87%), the majority also don't know the effects of palm oil on health (63%). Responses indicated that there is no relationship between dietary habit and palm oil health awareness. Significance was observed among gender, with more females reportedly unsure of the health effects of palm oil and palmitic acid ($\chi^2_{(4)} = 16.476$, $P = 0.002$). Among age groups, significantly more 18–25-year-olds were also unsure of these health effects ($\chi^2_{(4)} = 11.919$, $P = 0.018$). Significance was observed in dietary habit timeframe, with respondents consuming a particular diet for >5 years being particularly unsure of the health effects of palm oil and palmitic acid ($\chi^2_{(6)} = 15.160$, $P = 0.019$). Finally, palmitic acid content was found to be higher in diets containing meat and/or dairy, than without; a plant-based/vegan diet ($t(54) = 2.245$, $P = 0.029$). This study provides evidence to suggest that palm oil/palmitic acid awareness should be recognised as an important component of health and nutrition literacy. Providing consumers increased knowledge and understanding of the health effects of the ingredients they consume, particularly ingredients with the prevalence of palm oil and palmitic acid, will allow for

consumers to make more informed food choices, improving their eating habits, disease/co-morbidity management and prevention, what is more by extension; their metabolic health.

3.2 Introduction

As of 2025, domestic importation of palm oil to the United Kingdom (360,000 metric tonnes) has more than tripled since 1964 (117,000mt) (United States Department of Agriculture, 2024). Palm oil is widely used in food and non-food products. In addition to its' use as cooking oil and cooking oil blend, palm oil is extensively used in processed foods, such as chocolate, cookies, margarine, ice cream, packaged bread and pizza dough. Non-food (hygiene, cosmetic) items such as shampoo, soap, detergent and lipstick may also contain variations of palm oil and/or palm oil derivatives. Palmitic acid, the main component of palm oil, is naturally found in large amounts in high-fat animal-based products such as butter, milk, meat and cheese (Valsta et al., 2005).

In the 2024/25 period, approximately 80 million (metric) tonnes of palm oil will be produced globally (Statista, 2025). Over 80% of palm oil production takes place in Southeast Asia, with the top two countries being Indonesia and Malaysia, respectively (Mohd Hanafiah et al., 2021). Oil palm production is of major economic importance for Indonesia and Malaysia. The increasing global demand for palm oil has resulted in a sharp rise in production over the past few decades, with recent annual production in Indonesia alone consisting of 46.5 million tonnes (United States Department of Agriculture, 2024). While yields of palm oil are larger per hectare - due to its' popularity, palm oil ultimately uses more land than its' vegetable/edible oil equivalents. As a consequence, palm oil is directly linked to deforestation: accounting for around 18% globally (alongside soybeans). Beef production accounts for the most deforestation at 41% (Pendrill, et al., 2019). In Southeast Asia specifically, oil-palm agriculture has been named the greatest immediate threat to biodiversity (Wilcove & Koh, 2010).

Recently, the popularity of a plant-based or vegan diet has increased in the western world. In addition to ethical and environmental reasons behind avoiding foods from animal origin by vegans, a plant-based diet is often seen as a healthier choice (Wang et al., 2023). A vegan/plant-based diet is described as solely focusing on the consumption of plants/plant-based foods such as fruits, vegetables, nuts, whole grains, beans and oils, and no animal-based or animal-derived

foods (Fehér et al., 2020). Growing evidence suggests that a plant-based diet provides metabolic health benefit, due to dietary habits such as higher intake of fruits and vegetable and whole grain, while lower intake of dietary saturated fat and cholesterol (Kahleova et al., 2017; Trautwein & McKay, 2020). With the growing demands of plant-based foods - specifically plant-based alternatives to animal-origin (meat and dairy) food items - food industries responded in the form of providing processed versions for the demand and convenience of the consumer. These food products are convenient for vegans as they are free from dairy and meat, yet provide a similar taste, texture and mouth-feel. However, they fall within the category of ultra-processed foods - leading to plant-based/vegan diets also being susceptible to considerable consumption(s) of palm oil. The United Kingdom is reportedly one of the leading countries in the world to have highest number of vegan products, overtaking Germany to take the number one spot (Mintel, 2018).

Use of vegetable oils in processed foods is almost a universal phenomenon. Addition of oil/fat in food processing helps provide flavour, stability and longer shelf-life to food items (Martín-Torres et al., 2022). Palm oil in particular, is extremely versatile as a food ingredient, is odourless, colourless, has a longer shelf-life. All these desirable properties otherwise sought after by the processed food industries substantially contributed to its ever-growing popularity, and use in many every day food and non-food items. Palm oil contains predominantly the saturated fatty acid palmitic acid (16:0), alongside lower levels of myristic acid (C14:0) and stearic acid (C18:0), whereas most other vegetable oils contain higher contents of unsaturated fatty acids such as oleic acid (C18:1), linoleic acid (C18:2) and α-linoleic acid (C18:3) (Table 3.1).

Table 3.1. Fatty acid composition (% total) of common edible oils.

Edible oil	Fatty acid (%)					
	C14:0	**C16:0**	C18:0	C18:1	C18:2	C18:3
[1]Palm oil	**0.71**	**42.05**	**5.77**	**43.20**	**10.69**	**0.33**
Rice oil	0.26	**15.23**	2.06	44.93	30.26	0.93
Canola/palm oil blend	0.29	**12.53**	2.37	57.35	18.66	6.01
Corn oil	0.03	**11.06**	1.78	28.38	56.31	0.80
Olive oil	0.00	**10.27**	3.15	77.86	6.17	0.63
Peanut oil	0.03	**9.47**	3.53	59.26	19.96	0.50
Sesame oil	0.02	**9.32**	5.77	40.05	43.19	0.00

	Grapeseed oil	0.07	**8.06**	3.80	25.39	60.45	0.85	
	Sunflower oil	0.07	**6.62**	3.27	30.40	58.25	0.05	
	Flaxseed oil	0.00	**5.87**	4.35	20.38	15.85	52.65	
	Rapeseed oil	0.05	**4.05**	1.93	64.47	18.47	8.08	
	[1]Chapter 4; Fabiszewska & Bialecka-Florianczyk, 2014.							

Palmitic acid is predominantly found in meat and/or dairy food products such as beef, pork, milk, butter and cheese; all of which are absent within a plant-based/vegan diet. However, it is expected that plant-based diets likewise contain high levels of palm oil and consequently, the saturated fatty acid palmitic acid. This is due to palm oil being a major ingredient in ultra-processed food items such as vegan meat and/or dairy alternatives. Increased consumption of ultra-processed foods positively correlates with risks of cardiovascular disease (Srour et al., 2019), as well as nutrition-related cancer (Chang et al., 2023; Fiolet et al., 2018). As outlined in Chapter 1 of this Thesis, palmitic acid influences several molecular processes that lead to metabolic health risk factors such as inflammation, oxidative stress and insulin resistance; significant in the development of diseases within the metabolic syndrome spectrum including obesity, type-2 diabetes, heart disease and certain cancers.

With the widespread presence of palm oil, palmitic acid and their derivatives, particularly in processed foods, the growing evidence of their implications in the development chronic inflammatory conditions in humans, together with the absence of data concerning exact content in food labels, the metabolic health status of the United Kingdom is of great concern. Furthermore, (to date) there is no information available within the literature on consumer awareness of palm oil or palmitic acid, their prevalence or health effects. Perhaps the most comprehensive study of palm oil awareness found that awareness of palm oil was reportedly 77% after assessing 1695 British adults (Ostfeld et al., 2019). However, this study mainly focused on the effects of awareness of palm cultivation on the environment, particularly deforestation and climate change. Therefore, this study is limited to providing insight into consumer awareness of palm oils' environmental impact.

While minimal, efforts in recent years have been made to further educate consumers concerning palm oil in everyday food products. In 2014, the European Parliament introduced the 'Food Information to Consumers' (FIC) Regulation; requiring specificity of exact ingredients used, rather than just a generic, umbrella term (European Parliament, 2014). For

example, ‘palm oil’ was now mandatory on food labelling, replacing ‘vegetable oil’. Furthermore, over the last decade, in order to educate the general public about the presence of palm oil in food products, a smartphone app has been released. ‘PalmOil Scan’ (World Association of Zoos and Aquariums, 2025) was designed to enable users to search food databased using keywords, or directly scanning the barcodes of food items. This app provides information mostly relevant to the sustainability of palm oil production in the form of a *Sustainable Palm Oil Rating.* Using a quasi-traffic-light system, foods/companies in green are considered ‘excellent’, yellow indicates ‘good’ and orange indicates ‘poor’. This rating of ‘sustainability’ meaning the specific product (company) handles production of palm oil, with the minimal (the lowest possible) impact on the environment - as well as if they are a registered Roundtable on Sustainable Palm Oil (RSPO) member.

The Roundtable on Sustainable Palm Oil (RSPO; rspo.org) was formed in 2004 in order to find solutions to alleviate the effects that palm oil production was having on the environment. To this day however, it is questionable and unclear as to whether RSPO truly implements protocols that have positive, or lessen the destructive, effects on the environment (Gatti et al., 2019). Additionally, the largest palm oil trader in the world, Wilmar, despite being a member, has been criticised for dealing with plantations that do not adhere to guidelines set by RSPO, as well as their own ‘no deforestation’ policy, such as the treatment and abuse of personnel, as well as limited attempts to lessen environmental destruction (Pye et al., 2016). Ultimately, this app appears crucial in strengthening consumer awareness of how their palm oil is sourced, aiding in the avoidance of unsustainable palm oil practices. However, as it does not provide any information on the exact content of palm oil within foods, this contributes further to the evidence concerning a universal lack of palm oil and palmitic acid health effect and nutritional composition information being shared with the general public.

Substantial evidence suggests that palm oil and palmitic acid is fundamental in the development of many chronic diseases. Despite this, there is a continued absence of palm oil and/or palmitic acid information being widely available and distribute, leading to the inevitable deficiency in consumer knowledge of their content within foods, or their health implications. This is further exacerbated by palm oil and palmitic acids’ ever-growing prevalence and inclusion within every day foods, particularly processed and ultra-processed food items. The absence in consumer awareness concerning both the presence and health effects of these conceivably harmful substances in high-abundance within their diets could be the leading cause of several

metabolic health problems. Therefore, there is a strong need of a systematic assessment of consumer palm oil and palmitic acid awareness. It is hypothesised that consumers are aware of the existence of palm oil (and to a lesser extent, palmitic acid), yet unaware, or unsure of both their prevalence and health effect(s). The aim of this research was to assess the perceptions of palm oil/palmitic acid within the UK household, with the following objectives:

- To assess the consumers' awareness on palmitic acid/palm oil and its effect on health.
- To estimate the intake of palmitic acid/palm oil in common Western diets.

3.3 Methodology

3.3.1 Ethics application

This research was approved by the University of Central Lancashire Ethical Committee (Reference: HEALTH 0263).

3.3.2 Survey on awareness of palm oil/palm oil derivatives

Participants included in this survey were predominantly current UK residents living within UK (219) and ordinarily UK residents but currently out of the country (32). A total of 251 participants participated in this study. Participant information sheets (Appendix 3.1) were provided and consent forms (Appendix 3.2) were required, alongside the following the inclusion criteria: no pre-existing disease conditions and should be in good health; Must be aged 18+, any gender, any ethnicity; Access to a scale/balance (weight) and measuring tape (for height, waist and hip circumference). The data protection checklist is available to view in Appendix 3.3. Respondents were recruited primarily through social media such as Reddit and Facebook, or communication platforms such as Microsoft Teams, through related subreddits, groups or university-affiliated networks within each, respectively. The principal recruitment method used was a poster/flyer (Appendices 3.4a, 3.4b). Respondents were asked to complete a questionnaire designed to include 18 questions, divided into five sections, 1: Information about the participants, 2: Anthropometry, 3: Physical Activity and Lifestyle, 4: Dietary Habit, 5: Awareness on Palm Oil. The questionnaire circulated and the responses were collected during a period of 9 weeks (August - October 2022). Respondents were directed to complete the questionnaire via the survey website Qualtrics (uclan.eu.qualtrics.com), or via Microsoft Word document (Appendix 3.5). For the analysis of data for the awareness survey, the five

sections were broken down using five sections from the questionnaire, 1: Information about the participants; participant gender, age range, ethnicity. 2: Anthropometry; height, weight. 3: Physical Activity and Lifestyle; daily physical activity, physical activity type, sleep schedule and duration. 4: Dietary Habit; dietary habit, dietary habit duration, self-perceived health. 5: Awareness on Palm Oil; awareness of and opinion on/attitude towards palm oil/palmitic acid, in addition to potential reasons/motivations to avoid palm oil and its derivatives in the future (health, ethical, environmental, economic). Responses were collected from participant questionnaires and compiled within a Microsoft Excel spreadsheet.

Analyses of the questionnaire were primarily based on chi-square (χ^2) tests of association for categorical variables. A conventional *a priori* power calculation for χ^2 tests requires specification of an anticipated effect size (commonly Cohen's w) and degrees of freedom. In this study area, there were no sufficiently comparable published contingency tables to provide a credible basis for specifying an expected effect size, and an additional external pilot study was not feasible within the constraints of the doctoral programme. Accordingly, the questionnaire component was framed as exploratory.

Post-hoc, (observed) power calculations were not used to justify the achieved sample size or to interpret non-significant findings as they were deemed to be unsuitable. The methodological literature has repeatedly noted that 'observed power' is problematic, since once the test statistic and p-value are known, the calculated post-hoc power largely restates that information and does not add evidential value (Hoenig & Heisey, 2001). This redundancy has been explained particularly clearly within the contemporary literature, illustrating that for standard two-sided testing, p-values greater than 0.05 necessarily map onto post-hoc power values below 50%, irrespective of sample size (Nuzzo, 2021); consequently, large p-values will always imply low observed power even in well-designed studies. Similarly, it has been highlighted why this is especially problematic when post-hoc power is used to explain non-significant results, since it cannot meaningfully distinguish between: a) a genuinely underpowered study, and b) a study where there is no meaningful association to detect (Althouse & Chow 2019).

3.3.3 Dietary intake of palm oil/palmitic acid derivatives

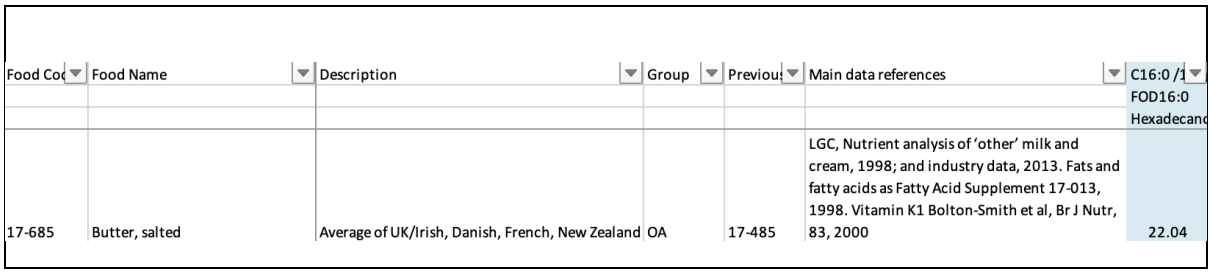
Participants were asked to complete a 4-day (over 1 week) dietary intake survey/food diary (Appendices 3.6). A total of 62 participants participated in the dietary intake survey. To provide a good representation of food consumption within a typical week, participants completed a 4-day food diary that records all foods consumed during a total of three weekdays (Monday, Wednesday, Friday) and one weekend day (Saturday). Food diaries recorded information including food or drink item, brand, as well as amount or size through early morning meal or breakfast, mid-morning, lunch, afternoon, evening meal or dinner and supper. All participants included in the dietary intake survey (62) also contributed to the survey study. Participants were recruited mainly from the local area (Preston, Lancashire) and around the University (UCLan) campus, via digital interactions (posts on university message boards, email) or physical advertisement (flyers/posters, used previously). Additional participants were sought for recruitment via social media (Facebook, Reddit), from various locations within the United Kingdom. The food intake data collected through diet diaries was analysed using nutrient analysis software Nutritics (nutritics.com). Split into the four-day diary structure of the food diary, food items were inputted and their size and/or gram amount recorded. In the instance where an exact food product was not contained within the Nutritics database, a food item closest the original food items' nutritional breakdown would be chosen. Data was exported, itemising detailed information on participant calorie, macronutrient and micronutrient intake. A Microsoft Excel spreadsheet was used to compile the relevant exported data; including energy (kcal), total carbohydrates, fibre, sugars, free sugars, total fats, saturated, trans-fatty acids, monounsaturated, polyunsaturated, protein, as well as vitamins A, D, E and K.

As only 2 of the 62 participants who provided food diary data chose the category: 'palm oil is healthy' from the survey, the three groups were condensed into two: 'palm oil is healthy/I don't know' and 'palm oil is unhealthy', for both the dietary data and the palmitic acid intake data.

3.3.4 Palmitic acid intake

To determine the content of palmitic acid and its derivatives in the food consumed by each participant, food diary data was analysed alongside information from the McCance & Widdowson's The Composition of Foods Integrated Dataset 2021 (Public Health England, 2021). This food composition database (FCD) compiles data on over 3000 UK-specific foods from various sources, including the Quadram Institute Food Labelling dataset (Quadram

Institute, 2021), and is considered the most comprehensive free source of information for the content of palmitic acid (16:0) in the UK. Available to the public as a as a searchable website (Quadram Institute, 2019), or downloadable Microsoft Excel spreadsheet (Figure 3.1).



Food Co	Food Name	Description	Group	Previou	Main data references	C16:0 /1
						FOD16:0
						Hexadecanc
17-685	Butter, salted	Average of UK/Irish, Danish, French, New Zealand	OA	17-485	LGC, Nutrient analysis of 'other' milk and cream, 1998; and industry data, 2013. Fats and fatty acids as Fatty Acid Supplement 17-013, 1998. Vitamin K1 Bolton-Smith et al, Br J Nutr, 83, 2000	22.04

Figure 3.1. Example food item entry within the Composition of Foods Integrated Dataset.
Note: From Public Health England, 2021.

An enhanced version of this data set: 'NutriticsGB23', proposed to address specific limitations such as accuracy, consistency and missing data (Innih et al., 2024), has been available since November 2023. However, data is not accessible without a Nutritics (nutritics.com) subscription.

Due to the lack of information on palmitic acid content of several food items consumed by the participants, particularly plant-based/vegan foods (meat/dairy alternatives): a fatty acid analysis of a selected panel of foods (Chapter 4 of this Thesis) was performed during this PhD research. Palmitic acid content from this newly reported dataset (Chapter 4 of this Thesis) was used (alongside the McCance and Widdowson dataset) to calculate the palmitic acid content within foods in the common UK household. For palmitic acid intake analysis, mean (g/day) intake (including standard error) was calculated for the 'Palm oil is healthy/Don't know' and the 'Palm oil is unhealthy' groups.

3.3.5 Statistical analysis

In order to obtain critical information regarding consumers' palm oil awareness from the questionnaire responses, food diary results and palmitic acid intake data, variables were categorised into three groups based on the answers to the question 'In relation to the health effect of palm oil and its derivatives, please select as appropriate'; 1: 'palm oil is unhealthy', 2: 'palm oil is healthy' and 3: 'I don't know'. Statistical analysis of the questionnaire data was performed using SPSS v29.0.2.0 (IBM, SPSS). Analysis of the questionnaire data was achieved

using one-way chi-squared tests, in addition to two-way cross-tabulation analysis via Pearson's chi squared tests. Formal tests of normality were not deemed necessary to determine whether parametric analyses could be applied. Inference on mean differences is typically robust to non-normality, and early modelling work shows that the t-test and the ANOVA F-ratio can maintain acceptable Type I error even under markedly non-normal outcome distributions in many scenarios (Lumley et al., 2002).

A univariate analysis of variance was performed for body mass index, as well as hip/waist circumference, mean and standard error values were also calculated and compared among the three 'palm oil health awareness' groups. When the data was condensed into two groups: 'palm oil is healthy/I don't know' and 'palm oil is unhealthy', an independent samples (two-tailed) T-test was performed. This analysis provided mean figures, in addition to the standard error and standard error of the mean, for each variable within the two groups.

Mean palmitic acid intake per (5) dietary habit, (omnivore, flexitarian, vegetarian, plant-based vegan, other), per (2) group ('palm oil is healthy/I don't know' and 'palm oil is unhealthy') was analysed, a univariate analysis of variance was adopted. Two-way cross-tabulation analysis was performed on each of the five diets to determine respondent count; both actual and expected values across the aforementioned two groups. A one-way analysis of variance was performed to determine mean intake per each diet, external of the two groups. As the main focus of this thesis is on palmitic acid within plant-based diets; an independent samples t-test was performed to determine mean palmitic acid intake for the diets including meat and/or dairy products (4; as a collective), versus those (1) without (plant-based/vegan diet).

3.4 Results

3.4.1 Participant characteristics and lifestyles

A chi-squared test was performed on the frequencies of respondent selections within the health awareness of palm oil question in the survey. Total count (n; %) for each variable within each section(s) of the questionnaire are described as follows: baseline demographic information in Table 3.2, physical activity and sleep information in Table 3.3, dietary habit information in Table 3.4. The respondents had 67% females and 28% males, 81% fall within the 18-34y (year) age group and 74% white ethnic background. There were 39% omnivore, 30% participants

plant-based/vegans and 11% vegetarians. 76% participants have been on their respective dietary habits for >2 years while 58% had for >5 years. 60% participants have sound nutritional health with 29% taking nutritional supplements and 7% has known nutritional deficiencies. 63% participants are physically active with 2 hours or more physical activity per day while the 37% participants were mostly sedentary (less than 1 hour per day). 51% has walking as their main type of physical activity, while 14% has running and 16% has weightlifting. 70% participants go to bed before the midnight during the weekdays, while 33% sleep 8-9 hrs.

Table 3.2. Respondent selection(s) within baseline demographic/characteristics parameters.

Variables	n (%)	*P-value*
Gender		**≤0.001**
Male	69 (27.5)	
Female	169 (67.3)	
Prefer not to say	13 (5.2)	
Age (y)		**≤0.001**
18-25	94 (37.5)	
25-34	109 (43.4)	
>35	48 (19.1)	
Ethnicity		**≤0.001**
White	187 (74.5)	
Asian British	26 (10.4)	
African British	6 (2.4)	
Other	32 (12.7)	

Note: P-values were determined by chi-square (χ^2) tests, measuring the probability that observed results (differences from expected results) occurred by chance.

Table 3.3. Respondent selection(s) within physical activity and sleep parameters.

Variables	n (%)	*P-value*
Physical activity/d		**<0.001**
Sedentary (<1)	93 (37.1)	
Moderate (2-3h)	129 (51.4)	
High (4h+)	29 (11.6)	
Physical activity type		**<0.001**
Walking	129 (51.4)	
Running	34 (13.5)	
Cycling	13 (5.2)	
Swimming	7 (2.8)	
Weight Training	41 (16.3)	
Other	25 (10)	
Unknown	2 (0.8)	
Sleep start (wk days)		**<0.001**
18:00-20:59	7 (2.8)	
21:00-23:59	174 (69.3)	
0:00-02.59	63 (25.1)	
03:00-05:59	3 (1.2)	
06:00-17:59	2 (0.8)	
Unknown	2 (0.8)	
Sleep start (free days)		**<0.001**
18:00-20:59	4 (1.6)	
21:00-23:59	120 (47.8)	
0:00-02.59	112 (44.6)	
03:00-05:59	9 (3.6)	
06:00-17:59	2 (0.8)	
Unknown	4 (1.6)	
Sleep duration (wk d)		**<0.001**
<4 h	5 (2)	
4 to 8	157 (62.5)	
8 to 9	83 (33.1)	
> 9h	6 (2.4)	
Sleep duration (free d)		**<0.001**
<4 h	5 (2)	
4 to 8	119 (47.4)	
8 to 9	106 (42.2)	
> 9h	16 (6.4)	
Unknown	5 (2)	

Table 3.4. Respondent selection(s) within dietary habit parameters.

Variables	n (%)	*P-value*
Dietary habit		**<0.001**
Omnivore	97 (38.6)	
Flexitarian	35 (13.9)	
Vegetarian	28 (11.2)	
Plant-based/vegan	75 (29.9)	
Other	16 (6.4)	
Dietary adaptation		**<0.001**
<3 m	15 (6)	
3m-2 yr	45 (17.9)	
2-5 y	46 (18.3)	
> 5 y	145 (57.8)	
Nutritional health status		**<0.001**
Enjoy(s) sound nutritional health	151 (60.2)	
Has a nutritional deficiency	17 (6.8)	
Takes supplement/medication	72 (28.7)	
Unknown	11 (4.4)	

3.4.2 Participants awareness of palm oil/palmitic acid and their derivatives

Of the total 251 participants including in the survey, 87% of the participants (P <0.001) are aware of palm oil. Although 45% of the participants don't really mind having the presence of palm oil in the foods they consume, 22% consumer eat foods that rarely contain any palm oil while 28% totally avoid food containing palm oil. Among the main underlying reason of avoidance of food containing palm oil, health reason (28%) and environmental reason (22%) are the dominant motives. The survey also found that 28% participants consider palm oil to be unhealthy while only 9% think healthy, 63% don't know whether palm oil is healthy or unhealthy (P <0.001). The palm oil awareness and preference(s) information of participants included in this survey are presented in Table 3.5.

Table 3.5. Respondent selection(s) within awareness and preferences on palm oil parameters.

Variables	n (%)	*P-value*
PO existence awareness		**<0.001**
Yes	218 (86.9)	
No	33 (13.1)	

	PO preference		**<0.001**
	My foods don't contain PO	11 (4.4)	
	My foods rarely contain PO	55 (21.9)	
	I avoid PO containing foods	71 (28.3)	
	I don't mind	112 (44.6)	
	PO health awareness		**<0.001**
	Don't know	158 (62.9)	
	Palm oil is healthy	23 (9.2)	
	Palm oil is unhealthy	70 (27.9)	
	Motivation of avoidance of PO		**<0.001**
	Health	70 (27.9)	
	Ethics	22 (8.8)	
	Environment	54 (21.5)	
	Economics	1 (0.4)	
	Health, Ethics & Environment	98 (39.0)	
	Unknown	6 (2.4)	

3.4.3 Participant demographic/lifestyle together with palm oil health perceptions

The most vital data observed within the questionnaire concerning the aims of this study is the respondents' awareness on the health effects of palm oil and palmitic acid. The three categories analysed were 'Don't know', 'Palm oil is healthy' and 'Palm oil is unhealthy'. As mentioned above, a significant difference ($\chi^2_{(2)} = 112.263$, $P < 0.001$) was observed, indicating that more respondents (158; 62.9%) don't know the effects of palm oil or palmitic acid on health. These three groups were then analysed concerning their significance across all survey responses.

For the 'Information about you' section, two-way cross-tabulation analysis was performed within the three categories: 'palm oil is healthy', 'palm oil is unhealthy', 'I don't know'. A significant difference ($\chi^2_{(4)} = 16.476$, $P = 0.002$) in the responses to 'In relation to the health effect of palm oil and its derivatives, please select as appropriate' as a function of 'Gender' was observed, indicating that fewer respondents who chose 'Prefer not to say', 'Don't know' the effects of palm oil and palmitic acid. A significant difference ($\chi^2_{(4)} = 11.919$, $P = 0.018$) was also observed as a function of 'Age (year)', indicating that predominantly 18-25 years olds are unfamiliar with these health effects. There was no significant difference observed within 'Your ethnicity'.

For the 'Anthropometry' section, a one-way univariate ANOVA compared the effect of the three choices within the 'In relation to the health effect of palm oil and its derivatives, please select as appropriate' question on 'BMI (kg/m2)' and 'Hip/Waist ratio'. A significant difference in 'BMI (kg/m2)' between at least two groups ($F_{(2, 220)} = 3.319$, $P = 0.038$, $\eta^2 = 0.03$) was observed. Post-hoc, Tukey's HSD Test for multiple comparisons found that the mean value of 'BMI (kg/m^2)' was significantly different between 'Don't know' and 'Palm oil is unhealthy' ($P = 0.038$) indicating that those who believe palm oil to be unhealthy, possess a lower ('healthier') BMI score. There was no significant difference between 'Don't know' and 'Palm oil is healthy', or 'Palm oil is healthy' and 'Palm oil is unhealthy'. There was also no significant difference within 'Hip/Waist ratio'.

Full outcome information of the 'Information about you' and 'Anthropometry' analyses are presented in Table 3.6.

Table 3.6. Respondent selection of palm oil awareness group within demographic parameters.

Parameters	Don't know	Palm Oil is healthy	Palm Oil is unhealthy	*P-value*
[1]BMI (kg/m2)	25.36 ± 0.42 (139)	25.61 ± 1.14 (20)	23.65 ± 0.45 (64)	[3]**0.038**
[1]Waist/Hip	0.91 ± 0.02 (112)	0.88 ± 0.03 (8)	0.92 ± 0.04 (30)	[3]**0.873**
[2]**Demographic**				
Gender				[4]**0.002**
Male	38 (43.3)	8 (6.3)	23 (19.2)	
Female	117 (106.4)	11 (15.5)	41 (47.1)	
Prefer not to say	3 (8.2)	4 (1.2)	6 (3.6)	
Age (y)				[4]**0.018**
18-25	71 (59.2)	7 (8.6)	16 (26.2)	
25-34	59 (68.6)	13 (10.0)	37 (30.4)	
>35	28 (30.2)	3 (4.4)	17 (13.4)	
Ethnicity				[4]**0.741**
White	116 (117.7)	17 (17.1)	54 (52.2)	
Asian British	19 (16.4)	2 (2.4)	5 (7.3)	
African British	3 (3.8)	0 (0.5)	3 (1.7)	
Other	20 (20.1)	4 (2.9)	8 (8.9)	

[1]Mean ± Standard Error of the Mean (n=). [2]Actual Values (Expected Values).

Note: [3]P-values were determined by ANOVA, measuring the probability that observed variations (between group means) occurred by chance. [4]P-values were determined by two-way cross-tabulation analysis, measuring the probability that observed associations (between categorical variables) occurred by chance.

Using two-way cross-tabulation analysis, the 'Physical activity and Lifestyle' section of the survey saw no significant differences in 'How would you describe the level of your daily physical activity (in hr)?', 'Select the main type of physical activity/Exercise you perform on daily basis', 'In a typical evening, you go to sleep at' weekdays or weekend, 'In a typical night, how many hours do you sleep?' weekdays or weekend. Full outcome information of this analysis is presented in Table 3.7.

Table 3.7. Respondent selection of palm oil awareness group within physical activity and sleep parameters.

Parameters	Don't know	Palm Oil is healthy	Palm Oil is unhealthy	*P-value*
Physical activity				
Physical activity/d				**0.859**
Sedentary (<1)	61 (58.5)	9 (8.5)	23 (25.9)	
Moderate (2-3h)	78 (81.2)	11 (11.8)	40 (36.0)	
High (4h+)	19 (18.3)	3 (2.7)	7 (8.1)	
Physical activity type				**0.194**
Walking	88 (81.2)	11 (11.8)	30 (36.0)	
Running	23 (21.4)	3 (3.1)	8 (9.5)	
Cycling	11 (8.2)	1 (1.2)	1 (3.6)	
Swimming	3 (4.4)	1 (0.6)	3 (2.0)	
Weight Training	18 (25.8)	4 (3.8)	19 (11.4)	
Other	13 (15.7)	3 (2.3)	9 (7.0)	
Unknown	2 (1.3)	0 (0.2)	0 (0.6)	
Sleep habits				
Sleep start (wk days)				**0.194**
18:00-20:59	4 (4.4)	2 (0.6)	1 (2.0)	
21:00-23:59	109 (109.5)	13 (15.9)	52 (48.5)	
0:00-02.59	41 (39.7)	6 (5.8)	16 (17.6)	
03:00-05:59	2 (1.9)	1 (0.3)	0 (0.8)	
06:00-17:59	0 (1.3)	1 (0.2)	1 (0.6)	
Unknown	2 (1.3)	0 (0.2)	0 (0.6)	

Sleep start (free days)				**0.210**
18:00-20:59	3 (2.5)	1 (0.4)	0 (1.1)	
21:00-23:59	76 (75.5)	8 (11.0)	36 (33.5)	
0:00-02.59	69 (70.5)	13 (10.3)	30 (31.2)	
03:00-05:59	8 (5.7)	0 (0.8)	1 (2.5)	
06:00-17:59	0 (1.3)	1 (0.2)	1 (0.6)	
Unknown	2 (2.5)	0 (0.4)	2 (1.1)	
Sleep duration (wk d)				**0.132**
<4 h	3 (3.1)	1 (0.5)	1 (1.4)	
4 to 8	107 (98.8)	13 (14.4)	37 (43.8)	
8 to 9	47 (52.5)	8 (7.6)	28 (23.1)	
> 9h	1 (3.8)	1 (0.5)	4 (1.7)	
Sleep duration (free d)				**0.175**
<4 h	3 (3.1)	1 (0.5)	1 (1.4)	
4 to 8	80 (74.9)	15 (10.9)	24 (33.2)	
8 to 9	62 (66.7)	6 (9.7)	37 (29.6)	
> 9h	8 (10.1)	1 (1.5)	7 (4.5)	
Unknown	4 (3.1)	0 (0.5)	1 (1.4)	

Actual Values (Expected Values).

Note: P-values were determined by two-way cross-tabulation analysis, measuring the probability that observed associations (between categorical variables) occurred by chance.

The 'Dietary habit' section saw no significant difference as a function of 'Based on your dietary habit, you are'. A significant difference ($\chi^2_{(6)} = 15.160$, $P = 0.019$) was observed as a function of 'You are on your present dietary habit since', indicating that respondents on their respective dietary habit for >5 years are unsure of the health effects of palm oil and palmitic acid. There was no significant difference observed within 'Which of the following describe your nutritional health?'. Full outcome information of this analysis is presented in Table 3.8.

Table 3.8. Respondent selection of palm oil awareness group within dietary parameters.

Parameters	Don't know	Palm Oil is healthy	Palm Oil is unhealthy	*P-value*
Dietary parameters				
Dietary habit				**0.128**
Omnivore	64 (61.1)	9 (8.9)	24 (27.1)	

Flexitarian	20 (22.0)	4 (3.2)	11 (9.8)	
Vegetarian	18 (17.6)	6 (2.6)	4 (7.8)	
Plant-based/vegan	44 (47.2)	4 (6.9)	27 (20.9)	
Other	12 (10.1)	0 (1.5)	4 (4.5)	
Dietary adaptation				**0.019**
<3 m	9 (9.4)	4 (1.4)	2 (4.2)	
3m-2 y	23 (28.3)	8 (4.1)	14 (12.5)	
2-5 y	27 (29.0)	3 (4.2)	16 (12.8)	
> 5 y	99 (91.3)	8 (13.3)	38 (40.4)	
Nutritional health status				**0.284**
Sound nutritional health	92 (95.1)	14 (13.8)	45 (42.1)	
Nutritional deficiency	12 (10.7)	3 (1.6)	2 (4.7)	
Supplement/medication	44 (45.3)	6 (6.6)	22 (20.1)	
Unknown	10 (6.9)	0 (1)	1 (3.1)	

Actual Values (Expected Values).

For the 'Awareness on Palm Oil' section, there was no significant difference as a function of 'Have you heard of palm oil/palm kernel oil/palmitic acid?'. A significant difference ($\chi^2_{(8)} = 32.184$, $P < 0.001$) as a function of 'In relation to the presence of palm oil/palm kernel oil in food, please select as appropriate' was observed. No significant difference as a function of 'If you are to avoid the foods containing palm oil and its derivatives, which of the following would motivate you?' was observed. Full outcome information of this analysis is presented in Table 3.9.

Table 3.9. Respondent selection of palm oil awareness group within palm oil awareness parameters.

Parameters	Don't know	Palm Oil is healthy	Palm Oil is unhealthy	*P-value*
Awareness of palm oil				
Existence awareness				**0.052**
Yes	131 (137.2)	22 (20.0)	65 (60.8)	
No	27 (20.8)	1 (3.0)	5 (9.2)	
Preference				**<0.001**

My foods don't contain PO	6 (6.9)	1 (1)	4 (3.1)	
My foods rarely contain PO	24 (34.6)	7 (5.0)	24 (15.3)	
I avoid PO containing foods	39 (44.7)	3 (6.5)	29 (19.8)	
I don't mind	87 (70.5)	12 (10.3)	13 (31.2)	
Unknown	2 (1.3)	0 (0.2)	0 (0.6)	
Motivation of avoidance				**0.057**
Health	42 (44.1)	12 (6.4)	16 (19.5)	
Ethics	19 (13.8)	1 (2.0)	2 (6.1)	
Environment	36 (34.0)	3 (4.9)	15 (15.1)	
Economics	0 (0.6)	0 (0.1)	1 (0.3)	
Health, Ethics & Environ.	58 (61.7)	7 (9.0)	33 (27.3)	
Unknown	3 (3.8)	0 (0.5)	3 (1.7)	
Actual Values (Expected Values).				

3.4.4 Dietary data

An independent samples t-test was performed to compare the mean of each of the macronutrients and micronutrients across participant food diaries. As this was not the main focus of the study, outcomes are included as appendices (Appendices 3.7a, 3.7b, 3.7c, 3.8). An independent samples t-test (two-tailed) was performed to compare the mean of each of the macronutrients and micronutrients within two of the palm oil awareness groups (refer to the Methodology): 'Palm oil is healthy/Don't know' and 'Palm oil is unhealthy'. There were no significant differences between any of the variables within the two groups (Table 3.10).

Table 3.10. Respondent selection of palm oil awareness group within dietary parameters.

Parameters	Palm oil is healthy /Don't know	Palm Oil is Unhealthy	*P-value*
Energy (kcal)			
Daily intake (kcal)	1845.98 ± 57.25	1940.04 ± 123.92	**0.478**
Target intake (kcal)	2349.01 ± 56.02	2401.11 ± 142.46	**0.697**
Carbohydrates (g)			
Total	197.37 ± 7.04	206.28 ± 16.79	**0.592**
Fibre	21.98 ± 1.16	31.48 ± 4.74	**0.075**

Sugars	63.92 ± 3.61	79.02 ± 9.65	**0.088**
Free sugars	7.16 ± 1.14	15.35 ± 3.79	**0.059**
Calories	789.48 ± 28.16	825.12 ± 67.18	**0.592**
Macronutrient (%)	43 ± 1	43 ± 3	**0.999**
Fats (g)			
Total	76.99 ± 3.61	85.16 ± 6.96	**0.319**
Saturated	23.85 ± 1.22	25.13 ± 3.61	**0.674**
Saturated (% of Total)	32 ± 1	30 ± 3	**0.384**
Saturated (kcal)	214.63 ± 11.02	226.21 ± 32.50	**0.674**
Trans-fats	0.48 ± 0.06	0.61 ± 0.17	**0.353**
Monounsaturated	14.85 ± 1.02	14.78 ± 2.16	**0.976**
Polyunsaturated	8.38 ± 0.60	10.28 ± 1.68	**0.202**
Calories	692.93 ± 32.56	766.47 ± 62.62	**0.319**
Macronutrient (%)	37 ± 1	39 ± 2	**0.300**
Protein (g)			
Total	74.33 ± 2.80	72.69 ± 8.39	**0.818**
Calories	297.31 ± 11.18	290.79 ± 33.57	**0.818**
Macronutrient (%)	16 ± 1	16 ± 1	**0.315**
Vitamin(s)			
Vitamin A (ug)	579.16 ± 59.85	843.87 ± 137.72	**0.076**
Vitamin D (ug)	2.12 ± 0.27	2.62 ± 0.56	**0.412**
Vitamin E (mg)	6.22 ± 0.48	7.63 ± 0.83	**0.182**
Vitamin K (ug)	38.72 ± 5.12	73.22 ± 16.27	**0.064**

Mean ± Standard Error of the Mean.

Note: P-values were determined by two tailed t test, measuring the probability that observed variations (between group means) occurred by chance.

3.4.5 Palmitic acid content

Mean palmitic acid intake (including standard error) was calculated for both groups: 'Palm oil is healthy/Don't know' (n =46) and 'Palm oil is unhealthy' (n =10). There were no significant differences in palmitic acid intake between these two groups. A two-way ANOVA was conducted to examine the effect of dietary habit ('Omnivore', 'Flexitarian', 'Vegetarian', 'Plant-based/vegan' and 'Other') and the perceptions of the health effects of palm oil ('Palm oil is healthy/Don't know' and 'Palm oil is unhealthy') on palmitic acid intake. There was no significant interaction between the effects of dietary habit and perceived palm oil/palmitic acid health effects on palmitic acid intake. Full information on both of these analyses is presented in Table 3.11. A one-way ANOVA was performed to compare the effects of dietary habit on dietary palmitic acid intake. No significant differences were observed concerning palmitic acid intake between the five dietary groups (Table 3.12). A significant difference between mean palmitic acid intake and dietary habit was observed ($t_{(54)} = 2.245$, $P = 0.029$), when condensed into two groups (1: diets containing meat and/or dairy, 2: plant-based/vegan diets), indicating that individuals consuming diets containing meat and/or dairy consume more palmitic acid than those without (Table 3.13).

Table 3.11. Respondent palmitic acid daily intake per awareness group.

Parameters	Palm oil is healthy/Don't know	Palm Oil is unhealthy	P-value
Daily intake			
Palmitic acid (16:0)	8.11 ± 0.60	7.64 ± 1.60	**0.754**
Dietary parameter(s)			
PA intake by dietary habit			**0.646**
Omnivore	9.00 ± 0.74	10.10 ± 3.49	
Flexitarian	10.11 ± 2.53	6.64 ± [1]0.00	
Vegetarian	6.36 ± 1.02		
Plant-based/vegan	5.29 ± 0.83	5.88 ± 1.47	
Other	6.27 ± 1.02		

Mean ± Standard Error of the Mean. [1]n=1.

Note: P-values were determined by two-way ANOVA, measuring the probability that observed variations (between group means) occurred by chance.

Table 3.12. Palmitic acid intake (grams per day) by dietary habit groups.

PA intake by dietary habit	*n*	Mean ± SE	*P-value*
Omnivore	27	9.16 ± 0.78	
Flexitarian	8	9.68 ± 2.23	
Vegetarian	7	6.36 ± 1.02	**0.065**
Plant-based/vegan	11	5.56 ± 0.77	
Other	3	6.27 ± 1.02	
Mean ± Standard Error of the Mean.			
Note: P-values were determined by ANOVA, measuring the probability that observed variations (between group means) occurred by chance.			

Table 3.13. Palmitic acid intake (grams per day) between two dietary groups (meat/dairy; non-meat/non-dairy).

PA intake by dietary habit (cont.)	*n*	Mean ± SE	P-value
Diet(s) containing meat and/or dairy	45	8.63 ± 4.33	**0.029**
Plant-based/vegan	11	5.56 ± 2.55	
Mean ± Standard Error of the Mean.			

3.5 Discussion

The prevalence of palm oil and palmitic acid within everyday food items cannot be understated, with around 50% of packaged foods including palm oil or one of its derivatives. Together with extensive reports of the adverse health effects of palm oil and palmitic acid, the growing concern for the metabolic health status of the common consumer necessitates intervention. To our knowledge, there is no information available within the literature on the United Kingdom general public's awareness of palm oil and palmitic acid, in particular their credibly damaging effects on health. This study was designed to be the first to investigate (as well as provide insight into the relationships between personal characteristics/behaviours and the awareness of) consumer awareness of the prevalence and health effects of palm oil and palmitic acid. In addition, an analysis of palmitic acid content within common dietary habits was implemented.

Analysis of the responses indicated that the majority of respondents (87%) are aware of the existence of palm oil. Considering its' prevalence within everyday foods, it is expected that respondents had previously heard of palm oil. Concerningly however, the majority (63%) of respondents also 'don't know' palm oil and palmitic acid health effects. While there is a lack of evidence outside of this study to suggest that the awareness of palm oil and palmitic acid within the United Kingdom is insufficient, it is reported that approximately 10 million adults in the UK, and 43% of England alone, possess deficient health literacy (NHS England, 2023; NHS England, 2025; Rowlands et al., 2015). Therefore, it is reasonable to assume that the UK does indeed possess a lack of palm oil awareness, in addition to strong associations with low health literacy. Both of which have serious potential health implications. Health literacy is an individual's ability to access, evaluate and understand health-related information in order to apply practices that promote and maintain their overall condition and well-being (Sørensen et al., 2015). Accessibility and effectiveness of health literacy can be impacted by many intrinsic factors such as country of origin, race, age, innate intelligence, sex/gender, as well as extrinsic factors such as education (reading, listening, writing) wealth and socio-economic status (Coughlin et al., 2020).

Several studies have evaluated the relationship between health literacy, nutritional knowledge and dietary habits/intake (Billany et al., 2023; Spronk et al., 2014; Taylor et al., 2019). Low health literacy is typically associated with poor eating habits and poor health outcomes (Shahid et al., 2022), in addition to impaired disease prevention and management skills (Davis et al., 2002; Osborne et al., 2022). Improved health literacy has previously reported to indicate positive effects on an individual's health behaviour(s) (Šulinskaitė et al., 2022). In alignment with the literature, the outcomes of questionnaire analysis indicated that BMI was significantly higher in respondents who chose 'Don't know', when compared to those who chose 'Palm oil is unhealthy'. While conducive to the growing evidence that improved health literacy contributes significantly to an individuals' metabolic health markers, it is important to note that some studies have found no association with health literacy and BMI (Boariu et al., 2024; Cunha et al., 2017).

While health literacy has been observed as relatively low amongst most age groups (Klinger et al., 2023), the outcomes of this study indicate that young adults (18-25y) in particular, are unsure of palm oil and palmitic acid health effects. Recent studies have linked young adults in particular with low health literacy (Sarhan et al., 2023). The most prominent and easily-

accessible source of an important aspect of health literacy - food ingredient/nutritional information, can be found on food packaging labels (Campos et al., 2011). However, it is reported that approximately two thirds of young adults (including college/university students) reported that they frequently do not pay attention to nutritional food labelling (Christoph et al., 2016). In young children, eating habits and health outcomes are improved when nutrition literacy is higher amongst themselves, in combination with their parents (Asakura et al., 2017).

The outcomes of this study also indicate respondents engaging with a specific dietary pattern for >5 years are principally those who are unsure of the aforementioned palm oil and palmitic acid health effects. Suggesting that individuals who practice dietary patterns for many (5+) years, become inflexible in their food choices/habits after a certain period of time has passed. According to recent studies: dietary complacency, described as the absence of concern for nutritional composition of foods an individual consumes, nor their health or disease implications, is common phenomena and often associated with health literacy (Silva et al., 2023). Correspondingly, these findings also suggest that those high health and nutrition literacy, are more willing to change their dietary pattern for the betterment of their health, aligning with many recent studies (Duarte et al., 2024; Siopsis et al., 2021; Zwierczyk et al., 2022).

Around half of respondents (45%) in this study reported that they don't mind the existence of palm oil in their foods. Additionally, significantly more respondents who don't know the health effects of palm oil and palmitic acid don't mind its presence within their foods. This could be observed as an inconsistency in respondent behaviour towards palm oil and palmitic acid, where the minority (<10%) believe palm oil is healthy, yet 45% don't mind the presence of palm oil. However, dietary complacency appears to affect more than just those practicing a dietary habit for many years. Predominant factors in food choice(s) are not generally influenced by nutritional concerns, but instead by factors such as taste, cost and convenience (Goyal & Deshmukh, 2018). Processed foods not only contain high levels of salt, sugar and fat (applied to improve taste) - but are also relatively cheaper to produce than fresh or whole foods. Likewise, 'fast foods' (classed as ultra-processed foods) are well-known and typically sought after due to their convenience. Contrary to these outcomes, while this survey did not require respondents to disclose rationale behind their food choices, specifically if they were motivated by factors such as taste, cost or convenience - the outcomes of questionnaire analysis did indicate that should respondents avoid palm oil at some point in the future, health and environment factors would be their chief motivation(s) for doing so.

Based on the results of this study, palmitic acid was significantly more prevalent within diets containing animal-products (meat and/or dairy) versus a plant-based/vegan diet. In addition, diets containing meat and/or dairy consume approximately 9.73 ± 4.84g of palmitic acid per day, whereas in plant-based/vegan diets, intake is approximately 4.33 ± 1.62g of palmitic acid per day (Chapter 5). In disagreement with recent findings where palmitic acid was reported to be present (in any diet) at around 20g per day (Murru et al., 2022), more than double. With palm oil (and incidentally, palmitic acid) being a predominant ingredient in ultra-processed foods, the results from this study would suggest that diets containing meat and/or dairy consume more ultra-processed foods than their plant-based/vegan counterparts. Interestingly however, according to a recent study; ultra-processed foods are typically consumed more within plant-based/vegan diets (39.5% of total food intake) than those containing animal-products such as omnivore, pescetarian and vegetarian (33%, 32.5 and 37%, respectively) - most likely due to the increased consumption of animal-product replacements such as plant-based meats or dairy (butter, milk) substitutes (Gehring et al., 2021). It is possible that the respondents of this survey whom practice a plant-based/vegan diet simply avoid the consumption ultra-processed foods, although not representative of the typical consumer. Regardless, in most instances, dietary intake of palm oil and palmitic acid is intrinsic, irrespective of diet.

This research is in agreement with recent studies utilising similar methodologies (Metoudi et al., 2025; Sinclair et al., 2022): an obligation is needed to transform the general public's perceptions and beliefs concerning health and nutrition, into actual nutritional choices and behaviour(s). In the case of palm oil and palmitic acid awareness, education practices in health literacy need to be implemented, with data becoming easily and freely accessible, accurate, and up-to-date. If respondents are indeed unaware of the palm oil and palmitic acid content that is within the foods they are consuming (due to lack of health literacy, or available resources), it is reasonable to assume that their self-perceived intake versus their actual intake, could be further contributing to the deterioration of public metabolic health. To provide a theorem: a much-improved, synergistic effort across governmental, clinical and academic organisations to increase efforts in improving health and nutrition literacy could substantially reduce risk factors of various chronic diseases. The effectiveness of health literacy interventions has been previously attributed to personalised/tailored to the needs of the individual (Visscher et al., 2018). Therefore, in order to appeal to the demographic of young adults most unsure about the

effects of palm oil (18-25y) - utilising aspects of their everyday lives such as mobile phone applications or social media platforms may prove effective. For most other age groups (except children): outside of detailed nutritional information distribution through national health initiatives, advances in dietary programs, as well as being taught more extensively within school, college and university food and nutrition courses; perhaps the swiftest, most direct-to-consumer and cost-effective approach includes further regulations on food labelling. Food labels are often (evidently) either uninteresting or confusing to the average consumer, especially with the high rate of low health and nutrition literacy occurrences within the UK. Therefore, clearer and more detailed presentation of ingredients could improve palm oil awareness and health outcomes significantly.

3.5.1 Limitations

Outside of the expectable biases that are synonymous with self-reported documentation such as selection/confirmation bias (awareness survey), or underreporting/participation-reactivity bias (food diary), this study has other potential limitations. Firstly, this research was exploratory: although many parametric tests are robust to non-normality in moderate-to-large samples, extreme skew/outliers and heteroscedasticity can still affect inference in some settings; therefore, findings should be interpreted with appropriate caution and future work could incorporate tests for normality. If low health literacy is as widespread amongst all age groups as the literature suggests, self-perceived health and/or food reporting could be further (unintentionally) affected. Although perhaps unavoidable, asymmetry in certain participant information such as sex/gender, age-range(s), ethnicity, dietary habit; proved difficult to control. Additionally, while dietary data provided great insight into palm oil/palmitic acid awareness and health literacy of participants/respondents, it is possible that a higher total quantity of included food diaries may have improved accuracy of data, being more representative of the UK at large. These limitations help provide strong recommendations for future studies to assess palm oil and palmitic acid awareness, to improve the health and nutrition literacy and overall metabolic health.

3.6 Conclusions

This study provides evidence to suggest that palm oil/palmitic acid awareness should be recognised as an important component of health and nutrition literacy. Overwhelmingly, the

results of this study indicate that the majority of the general public within the United Kingdom are aware of the existence of palm oil within their foods, yet concerningly, don't know the effects of palm oil or palmitic acid on their metabolic health. Conceivably harmful substances that are in high-abundance within dietary habits containing characteristics (high-fat, ultra-processed foods) of a 'Westernised diet'. As chronic disease is the leading cause of death worldwide, improving the health and nutrition literacy of the United Kingdom is crucial in determining the nationwide trajectory in the future of metabolic health. Providing consumers increased knowledge and understanding of both the prevalence and health effects of the food products they consume on a daily basis, particularly ingredients with the prevalence of palm oil and palmitic acid, will allow for 1: consumers to make more informed food choices, improving their eating habits, 2: aid in disease/co-morbidity management and prevention; and by extension 3: ameliorate widespread chronic disease. Lastly, limitations within the available databases, particularly plant-based/vegan foods such as meat or dairy alternatives, provide clear and concise rationale for a comprehensive analysis of the fatty acid composition of various prevalent, everyday food items.

Chapter 4. Fatty acid composition of common foods consumed in Westernised diets

4.1 Chapter Overview

The 'Western' or 'Western pattern' diet is the most common diet in the United Kingdom; containing high levels of saturated dietary fat, alongside an over-reliance on processed food items. Palmitic acid, found in both animal and vegetable fats, is the most abundant saturated fatty acid in the Western dietary patterns. Palm oil, the primary major source of palmitic acid, is a common constituent of many processed food items, regardless of dietary inclination. The primary objective of this study was to analyse the fatty acid composition of common food items, with an emphasis on palmitic acid content and its' health implications. The secondary objective was to mitigate the absence of various plant-based/vegan food alternatives from within both the literature and national databases, providing the fatty acid composition of a list of household foods. A total of 70 food samples, representing common food groups (vegetable/seed oils, palm-based foods, animal-origin foods, dairy foods, plant-based/vegan alternatives, nut/seed-based foods, miscellaneous/snack food items) were analysed for their fatty acid composition. A fatty acid profile of 38 different fatty acids was developed using gas chromatography. The highest content of palmitic acid was observed within palm oil itself (pure palm oil ~47%, unrefined palm oil ~42%), followed by palm-based foods including palm nut soup (~18%), palm nut cream (~18%), palm fruit pulp extract (~15%), and palm kernel oil (~6%). Non-palm foods with notably high content of palmitic acid included dark chocolate (~16%), and margarine (~15%). Other foods with relatively low content of palmitic acid included several of the edible oils including extra virgin olive oil (~12%), coconut oil (~10%), sesame oil (~10%), and sunflower oil (~6%), dairy-foods including parmesan cheese (~10%), cheddar cheese (~8%), goat cheese (~8%), and butter (~8%), nut/seed-based foods including Brazil nuts (~9%), and tahini (~8%), plant-based/vegan food alternatives including vegan chocolate cookie (~10%), vegan butter (~10%), and vegan chocolate (~9%), and some of the miscellaneous/snack foods including milk chocolate (~9%), and oatcakes (~8%). Animal-origin foods contained low contents of palmitic acid (~1-4%), instead the dominant fatty acid is oleic acid (~2-6%) While palmitic acid is in abundance within most everyday foods, across a variety of food groups, it is difficult to classify a food item as 'unhealthy' simple due to high palmitic acid content alone, as there are various other factors that need to be considered.

4.2 Introduction

Dietary lipids are essential nutritional components required for various physiological processes concerning human metabolic health and homeostasis. Lipids are an important source of energy stored within the body. In addition, lipids also play vital structural (cell membrane structure) and functional (signalling molecule, hormone and transportation) (Abumrad et al., 2016; Basson et al., 2021; Clandinin et al., 1991; Chianese et al., 2018; Jump & Clarke, 1999). Naturally-occurring dietary fatty acids include saturated, mono-unsaturated and poly-unsaturated fats. Trans-fats are unsaturated fatty acids have been linked to severe adverse health effects, in particular coronary heart disease; although, in recent years most countries reportedly consume trace (<1% of total energy intake) amounts (Wanders et al., 2017). High-fat dietary patterns are associated with an increased likelihood for a myriad of chronic diseases: including obesity (Rakhra et al., 2020), diabetes (Rice Bradley, 2018), atherosclerosis, heart/cardiovascular disease (CVD) (Wang et al., 2020), kidney disease (Hariharan & Kramer, 2015) gut dysbiosis (Martinez et al., 2017), as well as certain cancers such as colorectal cancer (Chun et al., 2015).

Saturated fats are typically considered the most harmful type of fat to human health because they play role in the development of diabetes (Luukonen et al., 2018), heart/cardiovascular disease (Maki et al., 2021) and different types of cancers (breast, prostate and colorectal cancers) (Mei et al., 2024). The adverse effects of saturated fats appear to predominantly be linked with high/over-consumption of certain saturated fatty acids. While a few recent studies also reported no association between saturated fat intake (within a balanced diet) and diseases such as diabetes, or cardiovascular disease (Gaeini et al., 2022; Heileson, 2020; Valk et al., 2022). In some instances, saturated fatty acids were even reported to have protective effects on human health (DiNicolantonio et al., 2016). The World Health Organisation (WHO) recommends <30% of total energy intake from fats, including <10% from saturated fats (WHO, 2023[a]; WHO, 2023[b]). Conflicting reports within the literature and recommended dietary restrictions or guidelines can feasibly cause confusion for the average consumer, leading to underestimations of the detrimental effects caused by dietary fats, particularly saturated fatty acids.

A diet high in saturated fats, or >11% of total energy intake (Public Health England, 2019), alongside increased refined sugar content and deficiencies in essential nutrients such as vitamins A, D and E, as well as dietary fibre, is colloquially referred to as the 'Western' or 'Western pattern' diet. Habitually practiced across the United States of America (USA), Canada and Western Europe, the Western diet is rapidly gaining prominence worldwide. Many countries are adopting the dietary characteristics of the Western diet, specifically the increased consumption of high-saturated fat, animal-origin foods, particularly processed meat and dairy products (Clemente-Suárez et al., 2023). As of 2025, the Western diet is the most common diet in the United Kingdom and in recent years, it has been reported that ultra-processed foods account for ~50% of all UK household family food purchases (Monteiro et al., 2017). Due to this over-reliance of pre-packaged/processed food items; high-saturated fat diets are similarly rich in polyunsaturated omega-6 (also known as ω−6, or *n*−6) fatty acids, yet contain scarce amounts (from red meat, processed meats) of omega 3 (ω−3, or *n*−3) fatty acids. Known as an essential fatty acid (ω−3: ω−6 ratio) imbalance: a surplus of ω−6 fatty acids, alongside a deficit of ω−3 fatty acids, has been linked to the pathogenesis of inflammatory and autoimmune diseases, as well as cancer and cardiovascular disease (DiNicolantonio et al., 2021; Gupta et al., 2013; Simopolous, 2008).

An increasingly popular diet within the Western world since the turn of the century is that of the plant-based/vegan diet. Similar to the 'Western' diet, the plant-based/vegan diet consists ~30% of total energy from fats (Bakaloudi et al., 2021; Cordain et al., 2005). Although, as animal-origin food products (meat, dairy) are not consumed; the main type of fatty acids in a plant-based/vegan diet are unsaturated fats, from sources such as nuts, seeds, fatty fruits (avocado, coconut) and various vegetable/seed oils (olive, sunflower, rapeseed). Further contrary to the Western diet, the plant-based/vegan diet is also particularly high in polyunsaturated fats (PUFAs), including ω−3 fatty acids. Due to this, the plant-based/vegan diet is oft-referred to and widely-perceived as a healthier option (Key et al., 2022). However, the ever-growing expansion, particularly over the past decade of the plant-based/vegan diet due to environmental factors, alongside the desire to fulfil certain palate preferences or nutrient requirements, has allowed it to become susceptible to many of the same health risks as most other Western diets. Increasing demand for plant-based/vegan alternatives to food products containing meat or dairy has led to an escalation in processed food production, and by implication an over-abundance of saturated fatty acids, and omega-6 likewise, appearing at the forefront.

Naturally, the content of fatty acids within food items, and therefore in any one individual's dietary preference(s), is varied and can affect metabolic health in different ways. Although, regardless of dietary inclination, common constituents of processed food items are refined vegetable oils (rapeseed oil, sunflower oil, soybean oil, maize oil), added to processed foods to improve their taste, texture and shelf-life. Over the past few decades, palm oil has become the frequently preferred ingredient in the processed food industry due to its efficiency (high crop yield per area of land), versatility (solidity at room temperature), resistance to oxidation, and cost-effectiveness, compared to other vegetable/seed oils. Crude palm oil is high in antioxidant (carotenoid/β-carotene, tocopherol and tocotrienol content); contributing to these structural and functional properties. However, the refining, bleaching and deodorisation of palm oil in the production process - in order to remove free fatty acids (non-triglycerides) to enhance palm oil's stability (at high temperatures), shelf-life and neutral colour/odour/flavour - also removes ~15-57% of these antioxidant nutrients (Posada et al., 2007).

Palm oil contains around 50% saturated fatty acid content, with the major saturated fatty acid being palmitic acid (16:0) at ~44%. Palmitic acid is the most common saturated fatty acid within human diets, accounting for ~50% of total saturated fatty acids. Palmitic acid is present in varying amounts in natural (minimally processed) or ultra-processed food products, including meat/dairy foods such as bacon, milk, cheese, butter and ice cream, in addition to plant/vegetable-origin foods such as most cereals, vegetable oils, nuts and seeds. Palmitic acid, irrespective of source, has various health implications associated with chronic disease; including inflammation, gut dysbiosis, oxidative stress and insulin resistance (Brodeur et al., 2013; Ghezzal et al., 2020; Korbecki & Bajdak-Rusinek, 2019; Reynoso et al., 2003). Interestingly, in some instances, palmitic acid's health effects appear independent of its' classification as a saturated fatty acid. For example, while palmitic acid is positively associated with the risk of type 2 diabetes (Prasad et al., 2022); an absent, or negative association is observed for lauric acid, stearic acid and myristic acid (Mancini et al., 2015), saturated fatty acids also present within palm oil.

Due to the global convergence of increased saturated fat and processed food consumption, as well as the mounting evidence of metabolic health risks associated with palmitic acid; it is essential to analyse the fatty acid composition of prevalent household food items. It is also reasonable to assume that the perception, or perhaps misconception, of plant-based/vegan diets

as an inherently healthy alternative, alongside the proliferation of ultra-processed food items, regardless of diet, has negatively contributed to the metabolic health status of the United Kingdom consumer. Furthermore, it is crucial that this information is easily accessible, to the United Kingdom general public, as a major initiative to tackle insufficiencies in health and nutrition literacy. Food composition databases such as the McCance and Widdowson Composition of Foods Integrated Dataset (CoFID) (Public Health England, 2021), one of the most prolific databases worldwide, are a valuable tool for both consumers as well as quantitative research (Church, 2006). Yet, while these databases provide detailed information on the nutritional composition of numerous foods, some of this information is incomplete, inaccurate/out-of-date or may not comprehensively cover the range of food/food ingredients consumed. Food products/ingredients evolve and change over time (particularly processed food items); therefore, there is a need of nutritional and chemical composition data. There is distinct lack of food compositional information particularly on plant-based/vegan food alternatives to animal-origin food items. Hence, the aim of this research was to assess the fatty acid composition (with particular emphasis on palmitic acid content) of common food items within UK diets, with the following objectives:

- To determine the fatty acid composition of a selected group of prevalent food items.
- To assess the extent of presence of palmitic acid content (16:0) in food items with an emphasis on health implications.

4.3 Methodology

This chapter was a collaborative project with the School of Health and Agricultural Sciences at University College Dublin. I performed all investigational, experimental and analytical procedures, with guidance and support from Dr Marco Garcia-Vaquero and Dr Raquel Cama-Moncunill.

4.3.1 Rationale

Following the analysis of food diaries (Chapter 3 of this thesis), assessment of palmitic acid intake in participants was attempted. However, due to a lack of palmitic acid content data for most of the food ingredients, particularly those of plant-based/vegan diets within the available databases (McCance and Widdowson composition of foods dataset), we assessed the fatty acid composition analysis of a selected set of food samples. This fatty acid composition analysis

provides the basis for the estimation of the palmitic acid intake in the participants and identification of gene expression biomarkers (Chapter 5).

4.3.2 Selection of food samples for fatty acid composition analysis

A total of 79 food samples were selected for the complete fatty acid analysis. Samples were initially selected based on their prevalence in the 62 food diaries (Chapter 3 of this thesis). The samples were selected based on firstly, the known fatty acid and saturated fatty content (High-fat foods). This group represents food items that contains ≥17.5% fat and/or ≥5% saturated fat content, respectively, per the United Kingdom Food Standards Agency (FSA) (food labelling) guidelines for 'high-fat' foods (Public Health England, 2019). Fat and saturated fat content of selected foods was obtained through Nutritics nutritional analysis software (nutritics.com) during analysis of food diaries (Chapter 3 of this thesis). Consumption of high-fat containing foods has health complications and hence, these group of foods were prioritised. Several low-fat food items were also identified through the analysis of food diaries, however, due to the technical limitation associated with the extraction and analysis of the fatty acid profile of low-fat foods, their inclusion was limited in this study. A total of 48 food items, selected based on prevalence, with high total fat and/or saturated fat content were included for the fatty acid analysis.

Additional samples were chosen based on their relevance to the study. Fat spread, with olive oil (Tesco) was chosen as a higher unsaturated fat, lower saturated fat alternative to butter. Three samples were also selected based on their stated absence of any palm oil, 1: 'Palm oil free' peanut butter (Meridian); 2: 'Palm oil free' multigrain bread (Jacksons); 3: 'Palm oil free' plant-based spread (Naturli). In addition, one relatively low-fat content food item was included in this list as a comparison to 'palm oil free' bread: seeds and grains bread (Warburtons). Secondly, several (7) plant-based/vegan samples were chosen due to their prevalence in the food diaries; however, many others (14) were chosen as alternatives (to be observed as a comparison) to meat or dairy food items already included within the initial selection. Plant-based/vegan food items in particular were absent from the McCance and Widdowson dataset (further contributing to their significance) leading to a total of 21 plant-based/vegan foods being selected. A total of 2 additional foods were chosen, 1: Brazil nuts (Tesco), chosen to include the majority of commonly consumed nuts within the analysis, 2: crisps (popped/straws) (Eat Real) as a 'healthier' comparison to potato crisps. Lastly, 10 food products focusing

largely on palm oil (or its' derivatives) were selected due to their evident relevance to this study. Fat content of additional foods was obtained via food product label, or brand/supplier website information. The full list of the initial selection of food items can be found in Table 4.1.

Once the selection of food items was finalised, the sourcing of food products could begin. All food items were sourced from UK or Ireland retailers. Chilled or frozen foods were purchased close to the vicinity of the laboratory (University College Dublin, Ireland) to avoid deterioration, stored in a freezer at -18° C when not in use. Food samples were categorised into different food groups including 1: edible oils; 2: dairy foods; 3: nut/seed-based foods; 4: animal-origin foods; 5: plant-based/vegan food alternatives; 6: miscellaneous/snack foods; 7: palm-based foods. Full sourced food information for each group can be found in Tables 4.2a, 4.2b.

Table 4.1. List of food items chosen within the selection phase.

Food code (McCance & Widdowson)	Food item description (Nutritics/McCance & Widdowson)	Prevalence (count)	[b]Fat content (g/100g)	[b]Saturated fat content (g/100g)
12-346	Cheese, Cheddar, English	19	34.9	21.7
17-661	Butter, unsalted/salted	18	82.2	52.1
17-671	Potato crisps, fried in sunflower oil	17	31.0	2.5
14-892	Peanut butter, smooth	13	52.0	6.8
17-654	Mayonnaise, standard, retail	12	79.0	6.2
14-386	Avocado, Hass, flesh only	12	19.5	4.1
17-038	Oil, olive	11	91.3	15.5
14-889	Coconut milk, retail	11	15.0	13.2
19-509	Sausages, pork, chilled, grilled	9	15.0	5.5
13-433	Houmous	9	23.8	3.0
17-648	Chocolate, milk	8	30.0	18.0
	Butter, vegan	7	56.0	12.6
17-041	Oil, rapeseed	7	91.7	7.6
14-896	Almonds, with skin	7	55.8	4.4
	Ice cream, premium, vegan	7	11.0	6.6
	Fruit and/or nut bar, raw	7	20.0	4.2
11-938	Breakfast cereal, crunchy clusters type, without nuts, unfortified	6	29.3	7.5
19-503	Bacon rashers, middle, grilled	6	17.0	7.0
17-491	Chocolate, plain (dark)	6	42.0	25.0
	Chocolate protein bar, vegan	6	18.9	10.4
	Burger, meat-free, vegan	6	19.0	5.6

12-525	Cheese, feta	6	23.0	17.1
14-877	Peanuts, kernel only, plain, unsalted/salted	5	52.0	7.4
14-879	Walnuts, kernel only	5	68.5	7.5
11-936	Pizza, cheese and tomato, retail	5	11.0	5.7
12-555	Yogurt, Greek style, plain	5	9.3	6.1
18-469	Beef, mince, raw	5	14.5	6.5
	Cheese, vegan	5	26.0	24.0
12-511	Ice cream, dairy, luxury, with chocolate/caramel	4	13.0	7.8
17-622	Pesto, green	4	28.4	4.0
11-823	Oatcakes, plain, retail	4	19.9	8.6
17-045	Oil, sunflower	4	92.0	7.9
15-635	Coleslaw	4	20.0	1.9
	Chia seeds	4	27.7	3.2
	Almond butter	4	52.3	4.1
	Chocolate, vegan	4	36.0	20.0
	Mayonnaise, vegan	4	72.0	5.3
12-511	Cheese, spreadable, full fat, soft, white	4	27.0	19.0
19-491	Burger, beef, 98-99% beef, grilled	4	18.1	7.7
14-811	Cashew nuts, kernel only, plain	4	50.9	10.1
17-043	Oil, sesame	3	92.0	13.1
17-031	Oil, coconut	3	100.0	94.0
12-500	Margarine, catering	3	75.0	24.0
12-357	Cheese, goats' milk, full fat, soft, white rind	3	29.3	19.7
14-845	Sunflower seeds	3	47.5	6.6
12-342	Cream substitute, double	3	31.0	21.0
12-526	Cheese, Parmesan, fresh	3	29.7	19.6

14-847	Tahini paste	3	58.5	9.3
12-505	[a]Fat spread, with olive oil		59.0	16.0
	[a]'Palm oil free' peanut butter		46.0	8.2
	[a]'Palm oil free' multigrain bread		1.7	0.3
	[a]'Palm oil free' plant-based spread		75.0	26.0
11-980	[a]Seeds and grains bread		2.0	0.5
	[a]Cookie, vegan		27.0	11.0
	[a]Sausages, vegan		14.0	5.3
	[a]Sausage roll, vegan		12.7	6.0
	[a]Salad cream, vegan		23.1	1.7
	[a]Pie, meat-free, vegan		13.9	6.5
	[a]Pesto, vegan		36.8	4.0
	[a]Bacon, vegan		19.7	3.5
	[a]Mince, vegan		1.2	0.3
	[a]Nuggets, vegan		2.6	1.0
	[a]Pizza, vegan		5.9	3.7
	[a]Brownie, vegan		8.8	2.8
	[a]Chicken burger, vegan		12.0	3.6
	[a]Cream, vegan		31.0	22.0
	[a]Soft cheese spread, vegan		6.4	5.2
	[a]Brazil nuts		68.5	14.8
	[a]Crisps (popped/straws)		31.3	2.5
17-039	[a]Oil, palm		100.0	49.3
	[a]Oil, palm (unrefined)		99.9	45.3
	[a]Palm oil blend/mixture		92.0	14.3

[a]Pure palm oil	99.3	47.8
[a]Zomi palm oil	98.0	48.0
[a]Palm kernel oil	100.0	81.5
[a]Palm nut cream	52.6	5.7
[a]Palm nut soup	52.6	5.7
[a]Palm fruit pulp extract	41.2	33.3
[a]Palm sugar	0.0	0.0

[a]Food items chosen for their relation/relevance to the study (not prevalence).

[b]Fat, saturated fat content based on Nutritics, the McCance and Widdowson Composition of Foods Integrated Dataset (CoFID) data.

Table 4.2a. List of sourced food items for analysis.

Group	Food item	Brand/Supplier	[a]Fat content (g/100g)	Size	Source
Edible oils (6)	Extra Virgin Olive Oil	Napolina	91.4	500ml	Tesco Superstore, Penwortham, Preston, England
	Coconut Oil	KTC	99.9	1l	Asda Superstore, Fulwood, Preston, England
	Sesame Oil	Tesco	100.0	250ml	Tesco Superstore, Penwortham, Preston, England
	Red Palm Oil + Rapeseed Oil	Carotino	92.0	500ml	Tesco Superstore, Penwortham, Preston, England
	Sunflower Oil	Tesco	100.0	1l	Tesco Superstore, Penwortham, Preston, England
	Extra Virgin Rapeseed Oil	Mr Hugh's	95.5	500ml	Tesco Superstore, Penwortham, Preston, England

Dairy foods (10)	Butter	Dairypak	60.3	500g	Tesco Superstore, Dundrum, Dublin, Ireland
	Margarine	Stork	75.0	250g	Tesco Superstore, Dundrum, Dublin, Ireland
	Cheddar Cheese	Creamfields	34.9	200g	Tesco Superstore, Dundrum, Dublin, Ireland
	Feta Cheese	Tesco	23.0	200g	Tesco Superstore, Dundrum, Dublin, Ireland
	Parmesan Cheese	Tesco	29.7	100g	Tesco Superstore, Dundrum, Dublin, Ireland
	Goat's Cheese	Tesco Finest	31.8	200g	Tesco Superstore, Dundrum, Dublin, Ireland
	Greek Yoghurt	Tesco	9.5	1kg	Tesco Superstore, Dundrum, Dublin, Ireland
	Chocolate Ice Cream	Cadbury	11.0	425ml	Tesco Superstore, Dundrum, Dublin, Ireland
	Double Cream	Elmlea	31.0	270ml	Dunnes Stores, Leopardstown, Dublin, Ireland
	Coleslaw	Tesco	17.7	400g	Tesco Superstore, Dundrum, Dublin, Ireland
Nut/seed-based foods (10)	Brazil Nuts	Tesco	68.2	250g	Tesco, Penwortham, Preston, England
	Cashew Nuts	Tesco	46.5	300g	Tesco, Penwortham, Preston, England
	Almonds	Tesco	55.8	200g	Tesco, Penwortham, Preston, England
	Peanuts	Tesco	46.0	300g	Tesco, Penwortham, Preston, England
	Walnuts	Tesco	68.5	200g	Tesco, Penwortham, Preston, England
	Peanut Butter	Tesco	50.5	340g	Tesco, Penwortham, Preston, England
	Palm Oil Free Peanut Butter	Meridian	46.0	280g	Tesco, Penwortham, Preston, England
	Almond Butter	Pip and Nut	52.3	170g	Tesco, Penwortham, Preston, England
	Sunflower Seeds	Tesco	47.5	150g	Tesco, Penwortham, Preston, England
	Tahini	Tesco	59.9	300g	Tesco, Penwortham, Preston, England
Animal-origin foods (4)	Beef Mince (10%)	Tesco	9.7	420g	Tesco Superstore, Dundrum, Dublin, Ireland
	Beef Burger	Tesco	14.3	454g	Tesco Superstore, Dundrum, Dublin, Ireland
	Pork Sausages	Tesco	21.5	227g	Tesco Superstore, Dundrum, Dublin, Ireland
	Bacon Rashers	Better Naked	34.6	200g	Tesco Superstore, Dundrum, Dublin, Ireland

[a]Fat content based on food label/brand website information.

Table 4.2b. List of sourced food items for analysis (continued).

Group	Food item	Brand/Supplier	[a]Fat content (g/100g)	Size	Source
Plant-based/vegan food alternatives (18)	Vegan Mayonnaise	Heinz	60.2	800ml	Tesco Superstore, Dundrum, Dublin, Ireland
	Vegan Salad Cream	Heinz	23.1	400ml	Tesco Superstore, Dundrum, Dublin, Ireland
	Vegan Chocolate Cookie	Wicked Kitchen	23.5	300g	Tesco Superstore, Penwortham, Preston, England
	Vegan Chocolate	Nomo	33.7	200g	Tesco Superstore, Penwortham, Preston, England
	Vegan Butter	Flora	79.0	200g	Tesco Superstore, Dundrum, Dublin, Ireland
	Vegan Ice Cream	Wicked Kitchen	12.2	500ml	Tesco Superstore, Dundrum, Dublin, Ireland
	Vegan Mince	Henry Denny & Sons	9.7	500g	Tesco Superstore, Dundrum, Dublin, Ireland
	Vegan Chicken Nuggets	Plant-It	19.0	200g	Aldi, Dundrum, Dublin, Ireland
	Vegan Soft Cheese	Philadelphia	21.0	145g	Tesco Superstore, Dundrum, Dublin, Ireland
	Vegan Pie	Plant Chef	9.8	568g	Tesco Superstore, Dundrum, Dublin, Ireland
	Vegan Burger	Vemondo	14.5	226g	Lidl, Dundrum, Dublin, Ireland
	Vegan Cheese	Violife	24.0	200g	Tesco Superstore, Dundrum, Dublin, Ireland
	Vegan Chicken Burger	Plant Menu	17.6	227g	Aldi, Dundrum, Dublin, Ireland
	Vegan Brownie Bites	Oggs	22.8	126g	Dunnes Stores, Leopardstown, Dublin, Ireland
	Vegan Bacon Rashers	Better Naked	12.1	150g	Tesco Superstore, Dundrum, Dublin, Ireland
	Vegan Pizza	Goodfella's	5.1	387g	Tesco Superstore, Dundrum, Dublin, Ireland
	Palm Oil Free Vegan Spread	Naturli	75.0	450g	Dunnes Stores, Leopardstown, Dublin, Ireland
	Vegan Double Cream	Elmlea	31.0	250ml	Dunnes Stores, Leopardstown, Dublin, Ireland
Miscellaneous/snack	Ready Salted Crisps	Walkers	31.0	150g	Tesco Superstore, Penwortham, Preston, England
	Straw Crisps	Eat Real	31.3	110g	Tesco Superstore, Penwortham, Preston, England
	Milk Chocolate	Cadbury	30.0	180g	Tesco Superstore, Penwortham, Preston, England

		85% Dark Chocolate	Lindt	46.0	100g	Tesco Superstore, Penwortham, Preston, England
		Fruit & Nut Bar	Nakd	20.0	140g	Tesco Superstore, Penwortham, Preston, England
		Protein Flapjack	Trek	22.0	150g	Tesco Superstore, Penwortham, Preston, England
		Oatcakes	Nairn's	19.9	250g	Tesco Superstore, Penwortham, Preston, England
		Seeds & Grains Bread	Warburtons	1.7	700g	Tesco Superstore, Penwortham, Preston, England
		PO Free Multigrain Bread	Jacksons	3.1	800g	Tesco Superstore, Penwortham, Preston, England
		Margherita Pizza	Milano	8.2	455g	Tesco Superstore, Dundrum, Dublin, Ireland
		Granola	Lizi's	29.3	400g	Tesco Superstore, Penwortham, Preston, England
		Avocado	Tesco	22.0	145g	Tesco Superstore, Dundrum, Dublin, Ireland
		Pesto	Sacla	33.0	190g	Tesco Superstore, Penwortham, Preston, England
		Olive Spread	Tesco	59.4	500g	Tesco Superstore, Dundrum, Dublin, Ireland
	Palm-based foods (7)	Palm Sugar	Thai Boy	0.0	500g	Asia Continental, City Centre, Preston, England
		Unrefined Palm Oil	KTC	99.9	500ml	Asia Continental, City Centre, Preston, England
		Pure Palm Oil	Tropical Sun	99.0	1l	JB Supplies Ltd, City Centre, Preston, England
		Palm Kernel Oil	Mystic Moments	100.0	100g	Amazon.co.uk
		Palm Nut Cream	Ghana Fresh	52.6	800g	JB Supplies Ltd, City Centre, Preston, England
		Palm Nut Soup	Ghana Best	52.6	800g	JB Supplies Ltd, City Centre, Preston, England
		Palm Fruit Pulp Extract	Ghana Taste	41.2	400g	JB Supplies Ltd, City Centre, Preston, England
	[a]Fat content based on food label/brand website information.					

4.4 Fatty acid analysis

4.4.1 Preparation of food samples

All food samples were prepared at the Analytical laboratory based at the University College Dublin (UCD), Ireland during December 2022 and July 2023. Prior to the sampling for fatty acid analysis, food samples were homogenised as following:

For edible oils, each food item (excluding coconut oil) was poured into 2 different 100ml plastic sample bottles, shaken gently and rotated upside down gently multiple times, then poured into a 3rd (final) 100ml bottle, repeating the aforementioned process then left to stabilise. For coconut oil, a Philips 'ProBlend 5' Blender model: HR2162/91 (Philips, Amsterdam, Netherlands) was used to mix the contents together. For dairy foods, each food item (excluding the various cheese-type food items) was mixed together using a CH180 Compact Mini Chopper (Kenwood Ltd, Havant, UK). For the cheese-type food items, each food item was cut into pieces with a knife and crushed/grinded down using a mortar and pestle. For nut/seed-based foods, the whole nut/seed-type food items were broken down/ground using a mortar and pestle. The nut/seed spread-type food items were mixed using a Philips 'ProBlend 5' Blender model: HR2162/91 (Philips, Amsterdam, Netherlands) or the aforementioned mini chopper. For animal-based foods, each food item was cut into pieces with a knife, then further chopped/minced using the aforementioned mini chopper. For plant-based/vegan food alternatives, the main preparation methods were mortar and pestle, or either of the aforementioned food blenders. For miscellaneous/snack foods, as there were many different types of food; various the appropriate methods of sample preparation established in the previous food groups were used. For palm-based foods, each food item was mixed together using either the blenders, or the aforementioned mini chopper.

Concerning foods with high heterogeneity: after the appropriate preparation method(s), parts of the food sample from various surface and interior areas of the food was collected then further crushed, milled, blended, to ensure high homogeneity. Post-preparation, samples to be analysed on subsequent days were placed into a 100ml plastic sample bottle and stored as per freezer temperature requirements noted above. Food samples for analysis were collected post-preparation using a sampling micro spoon, laboratory forceps (tweezers) or a plastic pipette (washed thoroughly each time or discarded between each sample). Based on the lipid (fat)

content of the samples and the threshold requirement for the fatty acid analytical process, specific weight of the samples was required (Table 4.3). Each sample (sample a, sample b/duplicate) was then appropriately weighed and transferred into two separate MARS Xpress extraction vessels (CEM Corporation, Matthews, NC, USA) for preparation of FAMEs (Tables 4.4a, 4.4b).

Table 4.3. Food sample weight requirements for analysis.

Note: Adapted from University College Dublin, Appendix 4.1.

Sample type	Lipid content	Example	Sample weight
Wet Solid	Very High (80%)	Meat fat, butter	0.1- 0.2g
	High (50%)	Walnuts	0.3 - 0.5 g
	Average (25%)	Pepperoni, cheese	0.5 - 0.7 g
	Low (5%-10%)	Lean Meat, yoghurt	0.8 - 1 g
	Low	Crab	3 g
Dry Solid	Average	Milk powder	0.5 g
	Low	Grains	1.5 g
	Very low	Bread	2 g
Liquid	Average	Milk	1 ml
	High	Oil	3 drops (record weight)

Table 4.4a. Food sample fat content, weight requirements and sample weights.

Group	Food sample	Fat Content (g/100g)	To Weigh	Sample a Weight (g)	Sample b Weight (g)	Sample weight (mean)
Edible oils (6)	Extra Virgin Olive Oil	91.4	3 drops	0.0641	0.0540	0.0591
	Coconut Oil	99.9	0.08g	0.0891	0.0826	0.0859
	Sesame Oil	100.0	3 drops	0.0579	0.0670	0.0625
	Red Palm Oil + Rapeseed Oil	92.0	3 drops	0.0581	0.0667	0.0624
	Sunflower Oil	100.0	3 drops	0.0781	0.0828	0.0805
	Extra Virgin Rapeseed Oil	95.5	3 drops	0.0664	0.0604	0.0634
Dairy foods (10)	Butter	60.3	0.2g	0.2066	0.2598	0.2332
	Margarine	75.0	0.1g	0.1071	0.1311	0.1191
	Cheddar Cheese	34.9	0.4g	0.4144	0.3980	0.4062
	Feta Cheese	23.0	0.5g	0.6034	0.4743	0.5389
	Parmesan Cheese	29.7	0.4g	0.4038	0.3851	0.3945
	Goat's Cheese	31.8	0.4g	0.4767	0.3859	0.4313
	Greek Yoghurt	9.5	0.8g	0.7923	0.8639	0.8281
	Chocolate Ice Cream	11.0	0.7g	0.7251	0.6967	0.7109
	Double Cream	31.0	0.4g	0.4043	0.4372	0.4208
	Coleslaw	17.7	0.6g	0.5900	0.6359	0.6130

	Nut/seed-based foods (10)	Brazil Nuts	68.2	0.2g	0.1959	0.2061	0.2010	
		Cashew Nuts	46.5	0.3g	0.2975	0.3434	0.3205	
		Almonds	55.8	0.3g	0.3313	0.2873	0.3093	
		Peanuts	46.0	0.3g	0.3815	0.2781	0.3298	
		Walnuts	68.5	0.2g	0.1892	0.2301	0.2097	
		Peanut Butter	50.5	0.3g	0.3185	0.2895	0.3040	
		Palm Oil Free Peanut Butter	46.0	0.3g	0.3034	0.3952	0.3493	
		Almond Butter	52.3	0.3g	0.2957	0.2916	0.2937	
		Sunflower Seeds	47.5	0.3g	0.3018	0.3468	0.3243	
		Tahini	59.9	0.3g	0.3115	0.3305	0.3210	
	Animal-origin foods (4)	Beef Mince (10%)	9.7	0.8g	0.7710	0.7625	0.7668	
		Beef Burger	14.3	0.7g	0.6813	0.7023	0.6918	
		Pork Sausages	21.5	0.5g	0.4945	0.4470	0.4708	
		Bacon Rashers	34.6	0.4g	0.4663	0.5439	0.5051	

Table 4.4b. Food sample weight requirements and food sample weights (continued).

Group	Food item	Fat Content (g/100g)	To Weigh	Sample a Weight (g)	Sample b Weight (g)	Sample weight (mean)
Plant-based/vegan food alternatives (18)	Vegan Mayonnaise	60.2	0.3g	0.3498	0.2709	0.3104
	Vegan Salad Cream	23.1	0.5g	0.5504	0.5080	0.5292
	Vegan Chocolate Cookie	23.5	0.5g	0.2964	0.3343	0.3154
	Vegan Chocolate	33.7	0.5g	0.5429	0.5118	0.5274
	Vegan Butter	79.0	0.1g	0.0983	0.0742	0.0863
	Vegan Ice Cream	12.2	0.7g	0.6779	0.7627	0.7203
	Vegan Mince	12.0	0.7g	0.7020	0.6771	0.6896
	Vegan Chicken Nuggets	19.0	0.6g	0.6167	0.5888	0.6028
	Vegan Soft Cheese	21.0	0.5g	0.5602	0.5118	0.5360
	Vegan Pie	9.8	0.8g	0.8595	0.8141	0.8368
	Vegan Burger	14.5	0.7g	0.7440	0.8900	0.8170
	Vegan Cheese	24.0	0.5g	0.4807	0.4915	0.4861
	Vegan Chicken Burger	17.6	0.6g	0.5909	0.6574	0.6242
	Vegan Brownie Bites	22.8	0.5g	0.5007	0.4899	0.4953
	Vegan Bacon Rashers	12.1	0.7g	0.7143	0.7694	0.7419
	Vegan Pizza	5.1	1g	0.9174	1.0723	0.9949
	Palm Oil Free Vegan Spread	75.0	0.1g	0.1239	0.1934	0.1587

		Vegan Double Cream	31.0	0.4g	0.4741	0.4840	0.4791
	Miscellaneous/snack foods (14)	Ready Salted Crisps	31.0	0.5g	0.4944	0.5014	0.4979
		Straw Crisps	31.3	0.5g	0.4952	0.5250	0.5101
		Milk Chocolate	30.0	0.5g	0.4978	0.5116	0.5047
		85% Dark Chocolate	46.0	0.3g	0.3478	0.3516	0.3497
		Fruit & Nut Bar	20.0	0.5g	0.5074	0.5043	0.5059
		Protein Flapjack	22.0	0.5g	0.5038	0.5111	0.5075
		Oatcakes	19.9	0.5g	0.5009	0.5173	0.5091
		Seeds & Grains Bread	1.7	1g	0.9954	1.0063	1.0009
		PO Free Multigrain Bread	3.1	1g	1.0867	1.0363	1.0615
		Margherita Pizza	8.2	0.8g	0.8244	0.8193	0.8219
		Granola	29.3	0.5g	0.5361	0.5302	0.5332
		Avocado	22.0	0.5g	0.5675	0.5188	0.5432
		Pesto	33.0	0.5g	0.6094	0.5455	0.5775
		Olive Spread	59.4	0.2g	0.2486	0.2843	0.2665
	Palm-based foods (7)	Palm Sugar	0.0	1g	0.9985	0.9503	0.9744
		Unrefined Palm Oil	99.9	0.08g	0.0952	0.0868	0.0910
		Pure Palm Oil	99.0	0.08g	0.0853	0.1010	0.0932
		Palm Kernel Oil	100.0	0.08g	0.0802	0.0678	0.0740
		Palm Nut Cream	52.6	0.3g	0.3051	0.2977	0.3014
		Palm Nut Soup	52.6	0.3g	0.3208	0.2870	0.3039
		Palm Fruit Pulp Extract	41.2	0.3g	0.3159	0.3662	0.3411

4.4.2 Preparation of Fatty Acid Methyl Esters (FAMEs)

In order to determine the fatty acid composition of food samples, there are several high-accuracy methods: including liquid chromatography, near-infrared spectroscopy and NMR spectroscopy (Rahmania, 2024). However, out of all analytical techniques, gas chromatography (GC) is the most commonly used (Chiu & Kuo, 2020). Gas chromatography allows for the identification and quantification of fatty acids from food samples; however, it requires the separation of fatty acid methyl esters (FAMEs) for analysis (Shantha & Napolitano, 1992). To begin, all food samples were joined by a magnetic 10mm stir bar within their respective extraction vessel, then identifiably marked. Microwave-assisted sample preparation for fatty acid analysis was performed using the MARS 6 Express 40 position Microwave Reaction System (CEM Corporation, Matthews, NC, USA). FAME preparation protocol required use of (microwave-assisted) saponification and methyl esterification processes (outlined in Brunton et al., 2015):

Saponification

Saponification is the process in which individual fatty acids are broken away from triglycerides (resulting in free fatty acids), allowing for quantitative determination and individual identification. During this step, 10ml potassium hydroxide (≥85%; KOH) (Fisher Scientific International, Inc, PA, USA) placed in methanol solution (Sigma-Aldrich, MA, USA) and 100µL IS (internal standard) (tricosanoic acid, 23:0) solution (Sigma-Aldrich, MA, USA) were added to each sample, before being placed in the sample carousel then placed within the microwave. Purity of the IS was recorded during its preparation phase, where IS dissolved in 7ml chloroform (Fisher Scientific International, Inc, PA, USA) placed in a 40° water bath and shaken well. The samples were heated up to 130°C, over 4 minutes, with a hold at 130°C for a further 4 minutes to achieve saponification. If the temperature of 130°C was not reached, the process was repeated. Once complete, the samples were cooled within an ice bath.

Esterification

Methyl esterification converts the free fatty acids into fatty acid methyl esters (FAMEs); to aid in identification, quantification of fatty acid composition via gas chromatography (GC) analysis. Once sample vessels cooled, 15ml acetyl chloride (98%) (Fisher Scientific International, Inc, PA, USA) in methanol solution was added to each food sample vessel before again being placed in the microwave. The samples were, this time, heated up to 120°C for ~4

minutes; with the same temperature (reach and hold) protocol as previously, to achieve esterification. After completion, samples were once again cooled within an ice bath.

4.4.3 Extraction of Fatty Acid Methyl Esters (FAMEs)

Following saponification and esterification, 10ml pentane (≥99%) (Sigma-Aldrich, MA, USA) was then added to each vessel in order to separate the FAMEs from the rest of the remaining food sample. The sample vessel(s) were gently and repeatedly turned/rotated approximately 10 times, before roughly 20ml of saturated NaCl (sodium chloride) (Lennox, Dublin, Ireland) was carefully added to each sample, before repeating the turning/rotating process. Immediately followed by leaving for up to 30 minutes to allow the aqueous (bottom) and organic (top) layers to separate. Post-separation, FAMEs are present within the top (clear) layer. Extraction of FAMEs was performed by removing approximately 1ml from this layer (via glass pipette), before transferring the contents to an amber (1.5ml) GC vial containing around 0.2g anhydrous sodium sulphate (Na_2SO_4) (Fisher Scientific International, Inc, PA, USA), ready for GC analysis. The food sample vessels were then cleaned thoroughly using heptane, followed by a glass/dishwasher cycle - after the remaining food contents within each vessel were appropriately wasted. All addition(s) of reagents and extraction of FAMEs, in addition to disposal of waste, was conducted with the appropriate safety gear (gloves, goggles) and performed within a fume hood. The full list of reagents is presented within Table 4.5. Visualisation of the FAME preparation and extraction process can be viewed in Figure 4.1.

Table 4.5. List of reagents used within the preparation of FAMEs phase.

Solutions	Supplier	Product identifier
Methanol, HPLC grade, 2.5L	Merck	34860-2.5L-R
Potassium Hydroxide, ≥ 85%, pellets 1Kg	Fisher Scientific	10113190
Acetyle chloride, 98%, 1L	Fisher Scientific	10317520
Pentane, suitable for HPLC, ≥99%, 2.5L	Merck	34956-2.5L-M
Cloroform, for HPLC 2.5L	Fisher Scientific	10615492
Sodium Chloride SA (7647-14-5) >99.5% 5KG	Lennox	CLEN0076-5KG
Na_2SO_4 anhydrous (1 KG)	Fisher Scientific	10224640
Standards	**Supplier**	**Product identifier**
Supelco 37 FAME Mix	Merck	CRM47885
Supelco PUFA n.3	Merck	47085-U
Tricosanoic acid C23:0, free acid (IS)	Merck	T6543-1G

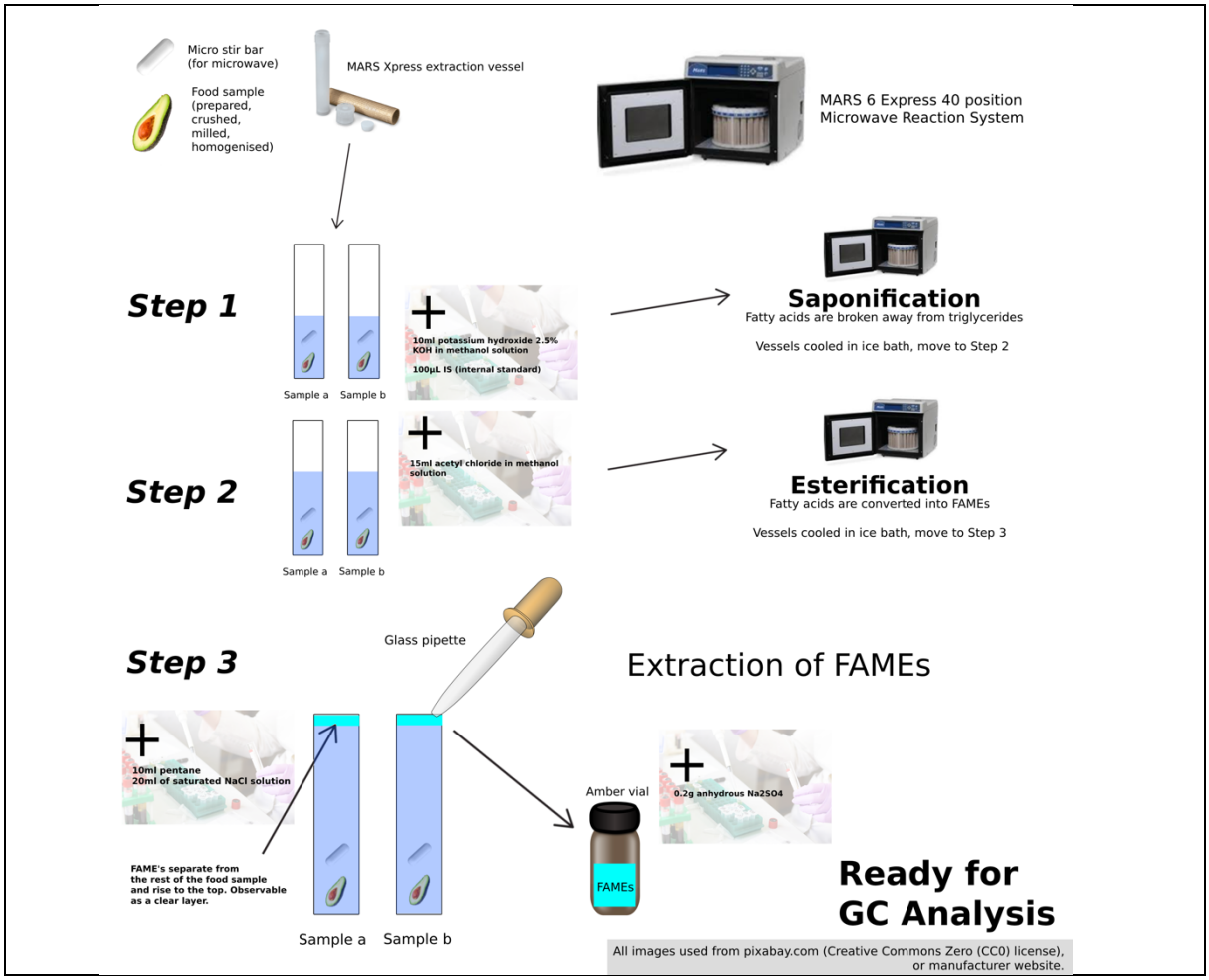


Figure 4.1. Esterification, saponification and extraction of FAMEs from food samples.

4.4.4 Analysis of Fatty Acid Methyl Esters (FAMEs)

Gas chromatography (GC) analysis

FAMEs were analysed in duplicate giving rise to a total of 158 samples (79 x 2). All fatty acid analysis was performed at the Analytical laboratory based at the University College Dublin (UCD), Ireland during December 2022 and July 2023. Analysis of fatty acid methyl esters (FAMEs) was conducted using Agilent Technologies 7890B GC equipped flame ionisation detector (Agilent Technologies, CA, USA). Each of the samples within the GC vials were added to the GC autosampler, alongside 1 blank (heptane), 1 Supelco FAME Mix (Supelco Inc, Pennsylvania, USA), and 1 Supelco PUFA n.3 standard (Supelco Inc, Pennsylvania, USA), to aid in fatty acid quantification and identification. Separation of FAMEs was performed using a CP-Sil 88 capillary 100 m × 0.25 mm (ID) × 0.2 µm (film thickness) (Agilent Technologies, CA, USA) column. Separation and detection of FAMEs required multiple hours (typically left

overnight) before full GC analysis is complete. A total of 38 different FAMEs were detected during the GC analysis phase (Table 4.6). The full breakdown of chromatographic conditions for the analysis of FAMEs is presented in Appendix 4.1.

Table 4.6. FAMEs (38) detected during GC analysis.

Saturated Fats (SFAs)	Monounsaturated Fats (MUFAs)	Polyunsaturated Fats (PUFAs)
C4:0 Butyric acid	C14:1 Myristoleic acid	C18:2n6t Linolelaidic acid
C6:0 Caproic acid	C15:1n5c Pentadecenoic acid	C18:2n6c Linoleic acid
C8:0 Caprylic acid	C16:1n7c Palmitoleic acid	C18:3n3 α-Linolenic acid
C10:0 Capric acid	C17:1n7c Heptadecenoic acid	C18:3n6c gamma-linolenic acid
C11:0 Undecanoic acid	C18:1n9t Elaidic acid	C20:2 cis-Eisodienoic acid
C12:0 Lauric acid	C18:1n9c Oleic Acid	C20:3n3c Eicosatrienoic acid
C13:0 Tridecanoic acid	C18:1n7c cis-Vaccenic acid	C20:3n6c Dihomo-γ-linolenic acid
C14:0 Myristic acid	C20:1n9c Eicosenoic acid	C20:4n6 Arachidonic acid
C15:0 Pentadecanoic acid	C22:1n9c Erucic acid	C20:5n3c Eicosapentaenoic acid
C16:0 Palmitic acid	C24:1n9c Nervonic acid	C22:2n6c Docosadienoic acid
C17:0 Heptadecanoic acid		C22:5n3c Docosapentaenoic acid
C18:0 Stearic acid		C22:6n3c Docosahexaenoic acid
C20:0 Arachidic acid		
C21:0 Henicosanoic acid		
C22:0 Behenic acid		
C24:0 Lignoceric acid		

Post-GC-analysis of FAMEs, output data is presented within OpenLAB CDS 2.1 Workstation software (Agilent Technologies, CA, USA) as a chromatogram (Figure 4.2), exhibiting spikes where each individual FAME data point is present. Manual calibration of each spike range was then required to align each spike with their respective fatty acid. Once chromatogram calibration is complete, quantitative determination of FAMEs to determine the content of each fatty acid (mg/g) within each food sample was performed following the equation in Figure 4.3. The peak area of the FAME is the highest point of each spike for that specific fatty acid within the chromatogram. Similarly, the peak area of the IS (internal standard) is the highest point for the internal standard within the chromatogram. IS purity was noted during the FAME preparation phase. After fatty acid content was determined, the mean of each food sample (food sample a, duplicate sample b) value was then calculated in order to determine the final fatty acid content (mg/g) per fresh food sample. All calculations were performed using a Microsoft Excel data sheet.

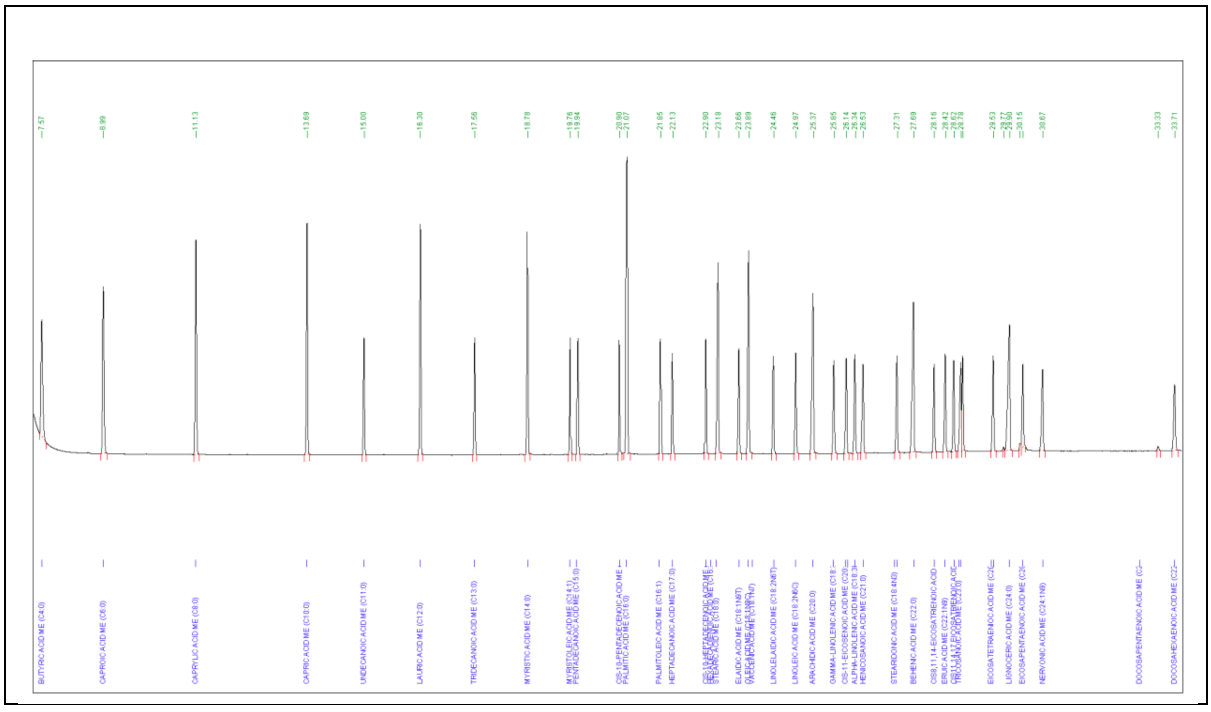


Figure 4.2. Example Chromatogram.

Note: From University College Dublin, Appendix 4.1.

$$\frac{PeakArea(FAME)}{PeakArea(ISTD)} \times \frac{Weight(ISTD)}{WeightSample} \times ISTDpurity \times 10 \times 0.96 = Content$$

Figure 4.3. Formula used to calculate fatty acid content (mg/g).

Note: From Appendix 4.1.

4.4.5 Expression of fatty acid content based on dry matter content

The dry matter content (dry weight basis) of food samples was also analysed. The dry weight of a food sample is the weight of the sample, excluding its water/moisture content, typically analysed to allow for fairer comparison(s) between food samples with varying levels of moisture content. Full protocol of dry matter content analysis is provided within the appendices (Appendix 4.2). The dry weight of food samples is presented within Appendix 4.3. Dry matter content analysis was performed using a Binder drying oven, model FD115/E2 (BINDER GmbH, Tuttlingen, Germany), with 2g (per duplicate) of food samples dried over consecutive periods, before weighing. While the food samples used in both analyses were derived from the same source/food, there was no overlap (i.e., food sample source produced: samples a and b - for fresh food analysis, and samples c and d - for dry weight analysis). The results of the analysis in fresh food were required to calculate the expression of fatty acid content (%) of food in dry matter. More specifically, fresh matter fatty acid content (%) was divided by the dry matter weight, then multiplied by 100.

4.4.6 Fatty acid composition data

From a food science perspective, the fatty acid composition of dry matter was important to analyze and include within the study, to allow for comparisons across food groups excluding their moisture content. However, 1: the drying of food items for dry matter analysis can influence changes in fatty acid profile. Heat-induced components of oven-drying such as increased temperature and prolonged duration(s) can degrade/alter levels of fatty acids, negatively affecting fatty acid composition (Caponio et al., 2003; Matin et al., 2024). 2: Dry matter fatty acid content does not represent foods in their practical, real-world setting. Therefore, from the nutritional science perspective of this thesis: potential degradation of fatty acid data, alongside the significance of presenting fatty acid composition as of foods as-

consumed; fresh weight was chosen as the focus for this study. Dry matter fatty acid content of foods is presented within Appendices 4.4, 4.5, 4.6, 4.7, 4.8a, 4.8b, 4.9, 4.10).

Fatty acid content for each group is presented in tabular format, displaying each food sample alongside a total of 38 fatty acids (mg/g of food), in addition to overall saturated, mono-unsaturated and polyunsaturated fatty acid content.

4.5 Results

4.5.1 Edible vegetable oils

The fatty acid composition of a total 6 edible oils (olive oil, coconut oil, sesame oil, red palm oil & rapeseed oil, sunflower oil and rapeseed oil) were analysed and is presented in Table 4.7. The major fatty acids in these edible oils were saturated fatty acid(s): C12:0 (lauric acid), C16:0 (palmitic acid) and C18:0 (stearic acid); monounsaturated fatty acid(s): C18:1n9c (oleic acid); and polyunsaturated fatty acid(s): C18:2n6c (linoleic acid). Of these 6 edible vegetable oils assessed, olive oil contains medium level of SFA (166.3 mg/g), high MUFA (875.5 mg/g), and a low level of PUFA (97.2 mg/g). Palmitic acid (119.3 mg/g), oleic acid (843.6 mg/g) and linoleic acid (87.9 mg/g) were the major SFA, MUFA and PUFA present in the olive oil, respectively. Coconut oil has a high level of SFA (855.5 mg/g), low MUFA (72.8 mg/g), and a low level of PUFA (18.5 mg/g). The major SFA, MUFA and PUFA present in the coconut oil are lauric acid (471.1 mg/g), oleic acid (70.5 mg/g) and linoleic acid (18.2 mg/g), respectively. Sesame oil contains medium level of SFA (160.4 mg/g), high MUFA (422.5 mg/g) and high PUFA (461.4 mg/g). The major SFA, MUFA and PUFA present in sesame oil are palmitic acid (97.7 mg/g), oleic acid (417 mg/g) and linoleic acid (456.8 mg/g), respectively. Red palm oil & rapeseed oil contains medium level of SFA (176 mg/g), high MUFA (572 mg/g) and medium PUFA content (251.4 mg/g). The major SFA, MUFA and PUFA present in red palm oil & rapeseed oil are palmitic acid (134.9 mg/g), oleic acid (555.1 mg/g) and linoleic acid (187.3 mg/g), respectively. Sunflower oil contains medium level of SFA (106.9 mg/g), high MUFA (290.8 mg/g) and medium PUFA content (584.9 mg/g). The major SFA, MUFA and PUFA present in red palm oil & rapeseed oil are palmitic acid (59.6 mg/g), oleic acid (287.7 mg/g) and linoleic acid (582.7 mg/g), respectively. Rapeseed oil contains medium level of SFA (66.1 mg/g), high MUFA (581.2 mg/g) and medium PUFA content (299 mg/g). The major SFA, MUFA and PUFA present in red palm oil & rapeseed oil are palmitic acid (41.3

mg/g), oleic acid (562.7 mg/g) and linoleic acid (195.9 mg/g), respectively. For the abundance of C16:0 (palmitic acid), high C16:0 in red palm oil & rapeseed oil (blend) (135.9 mg/g) and olive oil (119.3 mg/g), medium in coconut (100.8 mg/g) and sesame oil (97.7 mg/g) and low in sunflower (59.6 mg/g), and rapeseed oil (41.3 mg/g) were evident.

Table 4.7. Fatty acid composition of edible oils.

FAs [mg/g]	Olive Oil	Coconut Oil	Sesame Oil	Red Palm & Rapeseed Oil	Sunflower Oil	Rapeseed Oil
C4:0	nd	nd	nd	nd	nd	nd
C6:0	nd	nd	nd	nd	nd	nd
C8:0	nd	nd	nd	0.15	nd	nd
C10:0	nd	56.82	nd	0.21	nd	0.09
C11:0	nd	0.26	nd	nd	nd	nd
C12:0	nd	471.13	nd	1.39	nd	0.11
C13:0	nd	0.36	nd	nd	nd	nd
C14:0	0.29	194.67	0.44	3.76	0.79	0.51
C15:0	0.08	0.13	nd	0.29	0.14	0.18
C16:0	**119.25**	**100.84**	**97.65**	**134.88**	**59.63**	**41.26**
C17:0	0.95	0.16	0.58	0.76	0.40	0.53
C18:0	38.42	30.17	51.72	24.97	33.19	13.94
C20:0	4.91	1.00	5.82	5.41	2.47	5.10
C21:0	0.33	nd	0.20	0.25	0.15	0.17
C22:0	1.38	nd	2.51	2.53	7.51	3.06
C24:0	0.68	nd	1.46	1.39	2.61	1.15
C14:1	nd	nd	nd	nd	nd	nd
C15:1n5c	nd	nd	nd	nd	nd	nd
C16:1n7c	7.98	0.20	2.06	2.16	0.96	1.90
C17:1n7c	nd	nd	nd	nd	nd	nd
C18:1n7c	21.15	1.07	nd	nd	nd	nd
C18:1n9t	nd	0.57	1.03	nd	nd	nd

	C18:1n9c	843.64	70.49	416.98	555.14	287.72	562.70
	C20:1n9c	3.15	0.47	2.31	12.89	1.81	12.62
	C22:1n9c	nd	nd	0.11	0.47	0.13	2.14
	C24:1n9c	nd	nd	nd	1.31	0.21	1.80
	C18:2n6t	nd	nd	0.17	nd	0.09	nd
	C18:2n6c	87.85	18.16	456.75	187.28	582.67	195.86
	C18:3n3	8.19	0.11	3.68	62.83	1.67	101.61
	C18:3n6c	nd	nd	nd	nd	nd	nd
	C20:2	1.10	nd	0.75	1.33	0.46	1.45
	C20:3n3c	nd	nd	nd	nd	nd	0.08
	C20:3n6c	nd	nd	nd	nd	nd	nd
	C20:4n6	nd	nd	nd	nd	nd	nd
	C20:5n3c	nd	0.18	nd	nd	nd	nd
	C22:2n6c	nd	nd	nd	nd	nd	nd
	C22:5n3c	nd	nd	nd	nd	nd	nd
	C22:6n3c	nd	nd	nd	nd	nd	nd
	SFAs	166.30	855.54	160.38	175.97	106.89	66.12
	MUFAs	875.91	72.79	422.49	571.97	290.83	581.17
	PUFAs	97.15	18.46	461.35	251.44	584.89	299.01
	Data is presented as milligram (mg) total fatty acid (FA) content per 1 gram (g) fresh food. 'nd' indicates there were no FAs determined/detected. SFAs = saturated fatty acids, MUFAs = mono-unsaturated fatty acids. PUFAs = poly-unsaturated fatty acids.						

4.5.2 Dairy foods

A total of 10 dairy foods (butter, margarine, cheddar, feta, parmesan, goat cheese, Greek yoghurt, ice cream, double cream and coleslaw) were analysed and the fatty acid profile is presented in Table 4.8. The major fatty acids within dairy food items were saturated fatty acid(s): C12:0 (lauric acid), C14:0 (myristic acid), C16:0 (palmitic acid) and C18:0 (stearic acid); monounsaturated fatty acid(s): C18:1n9c (oleic acid); and polyunsaturated fatty acid(s): C18:2n6c (linoleic acid). Butter contains medium level of SFA (133.7 mg/g), medium MUFA (164.2 mg/g) and medium PUFA content (65.3 mg/g). The major SFA, MUFA and PUFA present in butter are palmitic acid (75.9 mg/g), oleic acid (151 mg/g) and linoleic acid (46.2 mg/g), respectively. Margarine contains high level of SFA (242.4 mg/g), high MUFA (305.5 mg/g) and medium PUFA content (124.4 mg/g). The major SFA, MUFA and PUFA present in margarine are palmitic acid (150.9 mg/g), oleic acid (305.5 mg/g) and linoleic acid (92.8 mg/g), respectively. Cheddar contains medium level of SFA (174.7 mg/g), low MUFA (60.6 mg/g) and low PUFA content (6.9 mg/g). The major SFA, MUFA and PUFA present in cheddar are palmitic acid (80.5 mg/g), oleic acid (52.9 mg/g) and linoleic acid (3.6 mg/g), respectively. Feta contains medium level of SFA (90.9 mg/g), low MUFA (29.8 mg/g) and low PUFA content (5.3 mg/g). The major SFA, MUFA and PUFA present in feta are palmitic acid (36.8 mg/g), oleic acid (28.1 mg/g) and linoleic acid (3.5 mg/g), respectively. Parmesan contains high level of SFA (208.1 mg/g), low MUFA (75.5 mg/g) and low PUFA content (12.2 mg/g). The major SFA, MUFA and PUFA present in parmesan are palmitic acid (101.6 mg/g), oleic acid (66.9 mg/g) and linoleic acid (8.6 mg/g), respectively. Goat cheese contains medium level of SFA (174.8 mg/g), low MUFA (54.1 mg/g) and low PUFA content (7.4 mg/g). The major SFA, MUFA and PUFA present in goat cheese are palmitic acid (78.7 mg/g), oleic acid (50.7 mg/g) and linoleic acid (5.8 mg/g), respectively. Greek yoghurt contains low level of SFA (17.3 mg/g), low MUFA (6.8 mg/g) and low PUFA content (0.7 mg/g). The major SFA, MUFA and PUFA present in Greek yoghurt are palmitic acid (8.3 mg/g), oleic acid (6.1 mg/g) and linoleic acid (0.1 mg/g), respectively. Ice cream contains low level of SFA (41.2 mg/g), low MUFA (13.5 mg/g) and low PUFA content (2.2 mg/g). The major SFA, MUFA and PUFA present in ice cream are palmitic acid (14.3 mg/g), oleic acid (13.3 mg/g) and linoleic acid (2 mg/g), respectively. Double cream contains medium level of SFA (102.9 mg/g), low MUFA (34.3 mg/g) and low PUFA content (13.2 mg/g). The major SFA, MUFA and PUFA present in double cream are lauric acid (42.9 mg/g), oleic acid (31.9 mg/g) and linoleic acid (9.8 mg/g), respectively. Coleslaw contains medium level of SFA (16.7 mg/g), low MUFA (78.7 mg/g)

and low PUFA content (42.5 mg/g). The major SFA, MUFA and PUFA present in coleslaw are palmitic acid (9.5 mg/g), oleic acid (73.1 mg/g) and linoleic acid (28.8 mg/g), respectively. For the abundance of C16:0 (palmitic acid), high C16:0 in margarine (150.9mg/g), parmesan (101.6 mg/g), medium in cheddar (80.5 mg/g), butter (75.92 mg/g) and goat cheese (78.7 mg/g), low in feta (36.8 mg/g), ice cream (14.3 mg/g), double cream (13.6 mg/g), coleslaw (9.5 mg/g), and Greek yoghurt (8.3 mg/g) were evident.

Table 4.8. Fatty acid composition of dairy-based foods.

FAs [mg/g]	Butter	Margarine	Cheddar	Feta	Parmesan	Goat Cheese	Greek Yoghurt	Ice Cream	Double Cream	Coleslaw
C4:0	nd	nd	0.88	0.29	0.53	0.30	0.07	0.01	nd	0.02
C6:0	nd	0.22	3.51	2.23	3.64	3.11	0.31	0.26	nd	0.13
C8:0	nd	3.12	2.86	2.85	2.95	4.78	0.24	2.77	nd	0.10
C10:0	3.74	3.15	7.65	9.98	8.04	20.49	0.56	2.55	5.09	0.27
C11:0	0.07	0.05	0.20	0.13	0.25	0.35	0.01	0.01	0.04	nd
C12:0	12.46	43.74	10.07	6.34	10.63	12.98	0.77	nd	42.87	0.38
C13:0	0.09	0.06	0.11	0.18	0.40	0.14	0.02	0.03	0.05	0.02
C14:0	17.32	17.78	32.37	15.28	36.11	28.54	2.62	9.58	17.59	1.27
C15:0	1.54	0.25	3.58	1.33	4.3	2.72	0.25	0.09	0.05	0.15
C16:0	**75.92**	**150.88**	**80.53**	**36.79**	**101.55**	**78.68**	**8.34**	**14.34**	**13.57**	**9.46**
C17:0	1.00	0.48	2.17	1.15	2.71	2.04	0.21	0.11	0.09	0.15
C18:0	18.93	19.64	30.05	13.24	34.8	19.1	3.65	10.89	22.85	3.39
C20:0	1.48	3.06	0.41	0.33	0.57	0.41	0.05	0.34	0.68	0.75
C21:0	1.16	nd	nd	0.62	1.19	1.01	0.14	0.04	nd	0.04
C22:0	nd	nd	0.18	0.14	0.26	0.11	0.02	0.07	0.01	0.40
C24:0	nd	nd	0.13	0.05	0.16	0.04	0.01	0.06	nd	0.15
C14:1	1.53	nd	2.79	0.23	2.98	0.64	0.18	0.07	0.02	0.10
C15:1n5c	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C16:1n7c	2.94	1.01	4.25	1.05	4.75	2.25	0.47	0.17	0.16	0.49
C17:1n7c	nd	nd	nd	nd	nd	nd	nd	nd	0.03	nd
C18:1n7c	6.22	12.29	0.28	0.27	0.82	0.50	nd	nd	1.39	4.58
C18:1n9t	nd	nd	0.04	0.01	0.05	0.02	0.09	nd	nd	nd

C18:1n9c	151.00	305.51	52.92	28.12	66.87	50.65	6.09	13.27	31.91	73.12
C20:1n9c	2.54	5.57	0.14	0.10	nd	nd	nd	nd	0.84	nd
C22:1n9c	nd	nd	0.08	0.02	0.04	0.02	nd	nd	nd	0.17
C24:1n9c	nd	nd	0.08	0.02	0.02	0.04	nd	nd	nd	0.25
C18:2n6t	nd	nd	0.02	0.01	nd	nd	nd	nd	nd	nd
C18:2n6c	46.07	92.81	3.63	3.51	8.6	5.75	0.4	2.04	9.84	28.83
C18:3n3	18.62	30.84	1.76	0.83	1.98	0.85	0.14	0.12	3.21	13.46
C18:3n6c	0.52	0.68	0.09	0.10	0.19	0.11	0.08	nd	0.18	nd
C20:2	nd	nd	0.14	0.05	0.16	0.10	0.02	0.02	nd	0.17
C20:3n3c	nd	0.03	0.04	0.01	0.03	0.02	nd	nd	nd	0.01
C20:3n6c	0.02	nd	0.22	0.04	0.43	0.07	0.01	0.01	nd	0.01
C20:4n6	nd	nd	0.25	0.34	0.58	0.42	0.03	0.01	nd	0.04
C20:5n3c	0.10	nd	0.22	0.08	0.18	0.10	0.04	0.00	nd	0.00
C22:2n6c	nd	nd	0.13	nd	nd	nd	nd	nd	nd	nd
C22:5n3c	nd	nd	0.33	0.18	nd	nd	nd	nd	nd	nd
C22:6n3c	nd	nd	0.03	0.18	nd	nd	nd	nd	nd	nd
SFAs	133.71	242.43	174.73	90.91	208.1	174.79	17.27	41.16	102.89	16.67
MUFAs	164.23	324.39	60.58	29.82	75.52	54.12	6.83	13.51	34.34	78.71
PUFAs	65.33	124.36	6.85	5.34	12.15	7.42	0.72	2.20	13.23	42.53

Data is presented as milligram (mg) total fatty acid (FA) content per 1 gram (g) fresh food.

'nd' indicates there were no FAs determined/detected.

SFAs = saturated fatty acids.

MUFAs = mono-unsaturated fatty acids.

PUFAs = poly-unsaturated fatty acids.

4.5.3 Nut/seed-based foods

A total of 8 nut/seed-based food items (Brazil nuts, cashews, almonds, peanuts, walnuts, peanut butter, 'palm oil free' peanut butter, almond butter, sunflower seeds, tahini) were analysed and the fatty acid profile is presented in Table 4.9. The predominant fatty acids within nut/seed-based food items were saturated fatty acid(s): C16:0 (palmitic acid), C18:0 (stearic acid); monounsaturated fatty acid(s): C18:1n9c (oleic acid); polyunsaturated fatty acid(s): C18:2n6c (linoleic acid). Brazil nuts contain medium level of SFA (150.5 mg/g), high MUFA (184.3 mg/g) and high PUFA content (237.2 mg/g). The major SFA, MUFA and PUFA present in Brazil nuts are palmitic acid (86.6 mg/g), oleic acid (181.8 mg/g) and linoleic acid (254.9 mg/g), respectively. Cashews contain medium level of SFA (83.5 mg/g), high MUFA (258.5 mg/g) and medium PUFA content (78.1 mg/g). The major SFA, MUFA and PUFA present in cashews are palmitic acid (40.72 mg/g), oleic acid (254.9 mg/g) and linoleic acid (77.5 mg/g), respectively. Almonds contain low level of SFA (38.1 mg/g), high MUFA (314.5 mg/g) and medium PUFA content (110.8 mg/g). The major SFA, MUFA and PUFA present in almonds are palmitic acid (28.4 mg/g), oleic acid (306.2 mg/g) and linoleic acid (110.7 mg/g), respectively. Peanuts contain medium level of SFA (86.5 mg/g), medium MUFA (165.2 mg/g) and high PUFA content (178.3 mg/g). The major SFA, MUFA and PUFA present in peanuts are palmitic acid (48.1 mg/g), oleic acid (159.3 mg/g) and linoleic acid (177.7 mg/g), respectively. Walnuts contain low level of SFA (34 mg/g), low MUFA (52.4 mg/g) and high PUFA content (273.6 mg/g). The major SFA, MUFA and PUFA present in walnuts are palmitic acid (23.4 mg/g), oleic acid (51.3 mg/g) and linoleic acid (221.4 mg/g), respectively. Peanut butter contains low level of SFA (61.3 mg/g), high MUFA (284.3 mg/g) and low PUFA content (31.5 mg/g). The major SFA, MUFA and PUFA present in peanut butter are palmitic acid (31.9 mg/g), oleic acid (270.8 mg/g) and linoleic acid (31 mg/g), respectively. PO free peanut butter contains low level of SFA (62.1 mg/g), high MUFA (271.9 mg/g) and low PUFA content (57.2 mg/g). The major SFA, MUFA and PUFA present in PO free peanut butter are palmitic acid (28.8 mg/g), oleic acid (262.2 mg/g) and linoleic acid (56.9 mg/g), respectively. Almond butter contains low level of SFA (44.3 mg/g), high MUFA (341.6 mg/g) and high PUFA content (151.8 mg/g). The major SFA, MUFA and PUFA present in almond butter are palmitic acid (35.3 mg/g), oleic acid (332.2 mg/g) and linoleic acid (151.7 mg/g), respectively. Sunflower seed(s) contain low level of SFA (67.8 mg/g), high MUFA (249.7 mg/g) and low PUFA content (0.3 mg/g). The major SFA, MUFA and PUFA present in sunflower seed(s) are palmitic acid (35.3 mg/g), oleic acid (332.2 mg/g) and linoleic acid (151.7 mg/g), respectively. Tahini

contains medium level of SFA (147.8 mg/g), high MUFA (374.3 mg/g) and high PUFA content (377.6 mg/g). The major SFA, MUFA and PUFA present in tahini are palmitic acid (84 mg/g), oleic acid (364.7 mg/g) and linoleic acid (374.8 mg/g), respectively. For the abundance of C16:0 (palmitic acid), medium C16:0 in Brazil nuts (86.6 mg/g) and tahini (84 mg/g), low in peanuts (48.1 mg/g), cashews (40.7 mg/g), peanut butter (31.9 mg/g), PO free peanut butter (28.8 mg/g), almond butter (35.3 mg/g), sunflower seed(s) (35.3 mg/g), almonds (28.4 mg/g), and walnuts (23.4 mg/g) were evident.

Table 4.9. Fatty acid composition of nut/seed-based food items.

FAs [mg/g]	Brazil Nut	Cashew	Almond	Peanut	Walnut	Peanut Butter	PO Free Peanut Butter	Almond Butter	Sunflower Seed(s)	Tahini
C4:0	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C6:0	nd	nd	nd	nd	nd	0.02	nd	nd	nd	nd
C8:0	nd	nd	nd	nd	nd	nd	nd	nd	0.02	nd
C10:0	nd	nd	nd	nd	nd	nd	nd	nd	0.02	nd
C11:0	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C12:0	0.03	nd	0.03	nd	0.02	0.05	0.01	0.03	0.16	0.03
C13:0	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C14:0	0.31	0.11	0.21	0.15	0.09	0.24	0.10	0.27	0.44	0.16
C15:0	0.08	0.06	0.07	0.06	0.06	0.04	0.03	0.07	0.09	0.03
C16:0	**86.58**	**40.72**	**28.37**	**48.12**	**23.42**	**31.93**	**28.81**	**35.28**	**36.19**	**84.04**
C17:0	0.54	0.74	0.40	0.47	0.23	0.44	0.45	0.40	0.29	0.51
C18:0	60.66	37.94	8.21	11.49	10.13	7.35	10.07	7.46	21.96	54.77
C20:0	1.60	2.66	0.46	5.91	nd	3.69	4.93	0.44	1.68	6.00
C21:0	0.08	0.03	0.11	0.10	nd	0.08	0.11	0.12	0.08	0.13
C22:0	0.42	0.57	0.14	13.33	nd	10.21	11.13	0.15	5.02	1.29
C24:0	0.19	0.69	0.08	6.87	nd	7.28	6.49	0.09	1.78	0.81
C14:1	nd	nd	0.01	nd	nd	nd	nd	nd	nd	nd
C15:1n5c	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C16:1n7c	1.96	1.39	2.11	0.16	0.24	0.22	0.24	2.32	0.65	1.06
C17:1n7c	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C18:1n7c	nd	1.42	5.64	nd	nd	1.79	1.59	6.55	3.43	6.81

	C18:1n9t	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
	C18:1n9c	181.81	254.89	306.22	159.28	51.28	270.77	262.17	332.16	244.35	364.71
	C20:1n9c	0.42	0.79	0.44	5.17	0.88	10.07	7.12	0.45	0.95	1.68
	C22:1n9c	0.03	nd	nd	0.39	nd	1.28	0.71	nd	0.02	0.02
	C24:1n9c	0.08	nd	0.08	0.23	nd	0.21	0.12	0.15	0.25	0.02
	C18:2n6t	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
	C18:2n6c	236.69	77.45	110.68	177.68	221.41	31.03	56.92	151.67	0.03	374.77
	C18:3n3	0.53	0.68	0.15	0.32	51.89	0.37	0.23	0.15	0.26	2.85
	C18:3n6c	nd	nd	nd	nd	0.24	0.03	nd	nd	nd	nd
	C20:2	nd	nd	nd	0.31	nd	nd	nd	nd	nd	nd
	C20:3n3c	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
	C20:3n6c	nd	nd	nd	0.01	nd	0.04	0.01	nd	nd	nd
	C20:4n6	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
	C20:5n3c	nd	nd	nd	0.02	nd	nd	nd	0.02	0.02	nd
	C22:2n6c	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
	C22:5n3c	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
	C22:6n3c	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
	SFAs	150.49	83.53	38.08	86.48	33.95	61.33	62.13	44.31	67.75	147.77
	MUFAs	184.30	258.49	314.50	165.23	52.40	284.34	271.94	341.63	249.65	374.31
	PUFAs	237.21	78.13	110.84	178.34	273.55	31.47	57.16	151.84	0.31	377.62
	Data is presented as milligram (mg) total fatty acid (FA) content per 1 gram (g) fresh food. 'nd' indicates there were no FAs determined/detected. SFAs = saturated fatty acids. MUFAs = mono-unsaturated fatty acids. PUFAs = poly-unsaturated fatty acids.										

4.5.4 Animal-origin foods

A total of 4 animal-origin foods (beef mince, beef burger, pork sausage, bacon rasher) were analysed and the fatty acid profile is presented in Table 4.10. The predominant fatty acids within animal-origin food items were determined to be saturated fatty acid(s): C16:0 (palmitic acid) and C18:0 (stearic acid); monounsaturated fatty acid(s): C18:1n9c (oleic acid). Beef mince contains low level of SFA (32.7 mg/g), low MUFA (28.1 mg/g) and low PUFA content (1.6 mg/g). The major SFA, MUFA and PUFA present in beef mince are palmitic acid (17.1 mg/g), oleic acid (24.6 mg/g) and linoleic acid (1.2 mg/g), respectively. Beef burger contains low level of SFA (72.5 mg/g), low MUFA (64.5 mg/g) and low PUFA content (3.1 mg/g). The major SFA, MUFA and PUFA present in beef burger are palmitic acid (37.8 mg/g), oleic acid (56.8 mg/g) and linoleic acid (2.24 mg/g), respectively. Pork sausage contains low level of SFA (37.9 mg/g), low MUFA (40.3 mg/g) and low PUFA content (16.5 mg/g). The major SFA, MUFA and PUFA present in pork sausage are palmitic acid (23.2 mg/g), oleic acid (37.3 mg/g) and linoleic acid (13.9 mg/g), respectively. Bacon rasher contains low level of SFA (19.3 mg/g), low MUFA (23.3 mg/g) and low PUFA content (10.3 mg/g). The major SFA, MUFA and PUFA present in bacon rasher are palmitic acid (12.3 mg/g), oleic acid (21 mg/g) and linoleic acid (8.5 mg/g), respectively. For the abundance of C16:0 (palmitic acid), low C16:0 in beef burger (37.8 mg/g), pork sausage (23.2 mg/g), beef mince (17.1 mg/g), and bacon rasher (12.3 mg/g) were evident.

Table 4.10. Fatty acid composition of animal-origin foods.

FAs [mg/g]	Beef Mince	Beef Burger	Pork Sausage	Bacon Rasher
C4:0	nd	nd	nd	nd
C6:0	nd	nd	nd	nd
C8:0	nd	nd	0.01	0.01
C10:0	nd	nd	0.08	0.05
C11:0	nd	nd	nd	nd
C12:0	0.06	0.10	0.09	0.05
C13:0	0.01	0.04	nd	nd
C14:0	1.81	4.06	1.27	0.64
C15:0	0.36	0.82	0.05	0.04
C16:0	**17.10**	**37.82**	**23.15**	**12.30**
C17:0	0.81	1.78	0.32	0.19
C18:0	12.48	27.65	12.70	5.91
C20:0	nd	0.25	0.18	0.09
C21:0	nd	nd	0.04	0.02
C22:0	0.05	nd	0.02	0.01
C24:0	nd	nd	0.01	0.01
C14:1	0.36	0.90	0.02	0.01
C15:1n5c	0.02	0.01	nd	0.01
C16:1n7c	2.19	4.97	2.22	1.81
C17:1n7c	0.00	nd	nd	nd
C18:1n7c	0.84	1.53	nd	nd

	C18:1n9t	nd	nd	nd	nd
	C18:1n9c	24.64	56.82	37.36	21.03
	C20:1n9c	nd	0.30	0.78	0.35
	C22:1n9c	nd	nd	0.03	0.02
	C24:1n9c	nd	nd	nd	nd
	C18:2n6t	0.00	0.01	nd	nd
	C18:2n6c	1.22	2.24	13.85	8.51
	C18:3n3	0.33	0.72	1.04	0.58
	C18:3n6c	0.03	0.06	0.06	0.04
	C20:2	nd	nd	0.63	0.32
	C20:3n3c	nd	nd	0.17	0.08
	C20:3n6c	nd	nd	0.12	0.10
	C20:4n6	nd	nd	0.41	0.47
	C20:5n3c	0.06	0.06	0.02	0.02
	C22:2n6c	nd	nd	0.02	0.02
	C22:5n3c	nd	nd	0.11	0.09
	C22:6n3c	nd	nd	0.03	0.03
	SFAs	32.70	72.52	37.92	19.32
	MUFAs	28.05	64.53	40.43	23.27
	PUFAs	1.64	3.09	16.46	10.26
	Data is presented as milligram (mg) total fatty acid (FA) content per 1 gram (g) fresh food. 'nd' indicates there were no FAs determined/detected. SFAs = saturated fatty acids. MUFAs = mono-unsaturated fatty acids. PUFAs = poly-unsaturated fatty acids.				

4.5.5 Plant-based/vegan food alternatives

A total of 18 plant-based/vegan food alternatives (vegan mayonnaise, vegan salad cream, vegan chocolate cookie, vegan chocolate bar, vegan butter, vegan ice cream, vegan mince, vegan nuggets, vegan soft cheese, vegan pie, vegan 'beef' burger, vegan cheese, vegan 'chicken' burger, vegan brownie, vegan bacon rasher, vegan pizza, vegan 'palm oil free' spread, vegan double cream) were analysed and the fatty acid profile is presented in Tables 4.11a, 4.11b. The predominant fatty acids within dairy food items were determined to be saturated fatty acid(s): C16:0 (palmitic acid), C18:0 (stearic acid), C14:0 (myristic acid); monounsaturated fatty acid(s): C18:1n9c (oleic acid); polyunsaturated fatty acid(s): C18:2n6c (linoleic acid), C18:3n3 (α-linoleic acid). Mayonnaise (v) contains low level of SFA (32.1 mg/g), high MUFA (279.6 mg/g) and high PUFA content (150.1 mg/g). The major SFA, MUFA and PUFA present in mayonnaise (v) are palmitic acid (20.2 mg/g), oleic acid (271.3 mg/g) and linoleic acid (100.9 mg/g), respectively. Salad cream (v) contains low level of SFA (19.8 mg/g), high MUFA (169.5 mg/g) and low PUFA content (71.5 mg/g). The major SFA, MUFA and PUFA present in salad cream (v) are palmitic acid (11.5 mg/g), oleic acid (152.4 mg/g) and linoleic acid (49.8 mg/g), respectively. Chocolate cookie (v) contains medium level of SFA (125.3 mg/g), medium MUFA (120.2 mg/g) and low PUFA content (37.4 mg/g). The major SFA, MUFA and PUFA present in chocolate cookie (v) are palmitic acid (98.1 mg/g), oleic acid (115.2 mg/g) and linoleic acid (32.9 mg/g), respectively. Chocolate (v) contains high level of SFA (227.6 mg/g), medium MUFA (130.1 mg/g) and low PUFA content (14.5 mg/g). The major SFA, MUFA and PUFA present in chocolate (v) are stearic acid (131.4 mg/g), oleic acid (127.6 mg/g) and linoleic acid (13.4 mg/g), respectively. Butter (v) contains high level of SFA (235.1 mg/g), medium MUFA (107.9 mg/g) and low PUFA content (42.1 mg/g). The major SFA, MUFA and PUFA present in butter (v) are palmitic acid (97.4 mg/g), oleic acid (105.6 mg/g) and linoleic acid (40.8 mg/g), respectively. Ice cream (v) contains low level of SFA (64.3 mg/g), low MUFA (15.3 mg/g) and low PUFA content (4 mg/g). The major SFA, MUFA and PUFA present in ice cream (v) are lauric acid (26.61 mg/g), oleic acid (15.2 mg/g) and linoleic acid (3.7 mg/g), respectively. Mince (v) contains low level of SFA (25.1 mg/g), low MUFA (48.5 mg/g) and low PUFA content (22.3 mg/g). The major SFA, MUFA and PUFA present in mince (v) are stearic acid (13.4 mg/g), oleic acid (45.6 mg/g) and linoleic acid (16.9 mg/g), respectively. 'Chicken' nuggets (v) contain low level of SFA (9.69 mg/g), low MUFA (30.3 mg/g) and low PUFA content (39.8 mg/g). The major SFA, MUFA and PUFA present in chicken nuggets (v) are palmitic acid (6.4 mg/g), oleic acid (28.8 mg/g) and linoleic acid (39.8

mg/g), respectively. Soft cheese (v) contains low level of SFA (55.8 mg/g), low MUFA (19 mg/g) and low PUFA content (5.6 mg/g). The major SFA, MUFA and PUFA present in soft cheese (v) are myristic acid (27.6 mg/g), oleic acid (18.5 mg/g) and linoleic acid (5.5 mg/g), respectively. Pie (v) contains low level of SFA (39.3 mg/g), low MUFA (31.4 mg/g) and low PUFA content (12.5 mg/g). The major SFA, MUFA and PUFA present in pie (v) are palmitic acid (34.8 mg/g), oleic acid (30.5 mg/g) and linoleic acid (11.3 mg/g), respectively. Burger (v) contains low level of SFA (25.1 mg/g), low MUFA (11 mg/g) and low PUFA content (13.5 mg/g). The major SFA, MUFA and PUFA present in burger (v) are myristic acid (9.4 mg/g), oleic acid (10.8 mg/g) and linoleic acid (13.3 mg/g), respectively. Cheese (v) contains low level of SFA (50 mg/g), low MUFA (9.7 mg/g) and low PUFA content (2.8 mg/g). The major SFA, MUFA and PUFA present in cheese (v) are myristic acid (20.3 mg/g), oleic acid (9.5 mg/g) and linoleic acid (2.7 mg/g), respectively. Chicken breast (v) contains low level of SFA (32.1 mg/g), low MUFA (64.2 mg/g) and low PUFA content (73.4 mg/g). The major SFA, MUFA and PUFA present in chicken breast (v) are palmitic acid (29.2 mg/g), oleic acid (63.7 mg/g) and linoleic acid (69.4 mg/g), respectively. Brownie (v) contains low level of SFA (70.3 mg/g), low MUFA (62 mg/g) and low PUFA content (22.3 mg/g). The major SFA, MUFA and PUFA present in brownie (v) are palmitic acid (48.6 mg/g), oleic acid (61.6 mg/g) and linoleic acid (18.6 mg/g), respectively. Bacon rasher (v) contains low level of SFA (8.1 mg/g), low MUFA (55 mg/g) and low PUFA content (25.9 mg/g). The major SFA, MUFA and PUFA present in bacon rasher (v) are palmitic acid (5.2 mg/g), oleic acid (51.5 mg/g) and linoleic acid (19.6 mg/g), respectively. Pizza (v) contains low level of SFA (2.9 mg/g), low MUFA (10.3 mg/g) and low PUFA content (9.7 mg/g). The major SFA, MUFA and PUFA present in pizza (v) are palmitic acid (2 mg/g), oleic acid (10.2 mg/g) and linoleic acid (8.2 mg/g), respectively. PO free spread (v) contains high level of SFA (154.3 mg/g), high MUFA (233.4 mg/g) and low PUFA content (88 mg/g). The major SFA, MUFA and PUFA present in PO free spread (v) are stearic acid (59.1 mg/g), oleic acid (221.2 mg/g) and linoleic acid (65.7 mg/g), respectively. Double cream (v) contains low level of SFA (68.6 mg/g), low MUFA (61.7 mg/g) and low PUFA content (24.5 mg/g). The major SFA, MUFA and PUFA present in double cream (v) are palmitic acid (40.2 mg/g), oleic acid (58.1 mg/g) and linoleic acid (17.7 mg/g), respectively. For the abundance of C16:0 (palmitic acid), medium C16:0 in chocolate cookie (v) (98.1 mg/g), butter (v) (97.4 mg/g), chocolate (v) (88.7 mg/g), low in brownie (v) (48.6 mg/g), double cream (v) (40.2 mg/g), pie (v) (34.8 mg/g), chicken breast (v) (29.2 mg/g), PO free spread (v) (25.7 mg/g), low in mayonnaise (v) (20.2 mg/g), soft cheese (v) (15.7 mg/g), ice cream (v) (14.4 mg/g), cheese (v) (13.1 mg/g), salad cream (v) (11.5 mg/g), burger (v) (7.7 mg/g), ‘chicken

nuggets' (v) (6.4 mg/g), mince (v) (6.1 mg/g), bacon rasher (v) (5.2 mg/g), and pizza (v) (2 mg/g) were evident.

Table 4.11a. Fatty acid composition of plant-based/vegan food alternatives.

FAs [mg/g]	Mayonnaise (v)	Salad Cream (v)	Chocolate Cookie (v)	Chocolate (v)	Butter (v)	Ice Cream (v)	Mince (v)	'Chicken' Nuggets (v)	Soft Cheese (v)
C4:0	nd	nd	nd	nd	nd	nd	nd	nd	nd
C6:0	nd	0.03	0.05	0.06	nd	nd	nd	nd	nd
C8:0	nd	nd	nd	nd	nd	nd	nd	nd	nd
C10:0	0.04	0.02	0.04	nd	nd	2.96	nd	nd	7.38
C11:0	nd	nd	nd	0.06	0.08	0.03	0.00	0.01	0.04
C12:0	0.06	0.03	0.44	0.05	89.57	26.61	3.16	0.02	0.01
C13:0	nd	nd	nd	nd	0.11	0.02	0.01	nd	0.05
C14:0	0.29	0.15	2.09	0.35	32.45	11.17	1.45	0.08	27.63
C15:0	0.09	0.06	0.14	0.10	0.13	0.02	0.03	0.02	0.02
C16:0	**20.17**	**11.49**	**98.05**	**88.65**	**97.40**	**14.40**	**6.14**	**6.43**	**15.70**
C17:0	0.35	0.51	0.99	1.44	0.25	0.37	0.09	0.07	0.03
C18:0	8.85	4.61	21.33	131.41	15.05	8.67	13.40	2.73	4.89
C20:0	nd	1.56	1.44	4.20	nd	nd	0.81	0.33	nd
C21:0	0.14	0.06	0.04	0.07	nd	nd	nd	nd	nd
C22:0	1.48	0.86	0.38	0.75	nd	nd	nd	nd	nd
C24:0	0.59	0.39	0.29	0.44	nd	nd	nd	nd	nd
C14:1	nd	nd	nd	nd	nd	nd	nd	nd	nd
C15:1n5c	nd	nd	nd	nd	0.03	nd	nd	nd	nd
C16:1n7c	0.99	0.56	0.49	0.88	0.35	0.07	0.15	0.12	0.08
C17:1n7c	nd	nd	nd	nd	nd	nd	nd	nd	nd
C18:1n7c	nd	7.62	3.02	1.29	1.88	nd	1.82	1.37	0.32
C18:1n9t	nd	nd	0.18	nd	nd	0.04	nd	nd	0.04

	C18:1n9c	271.32	152.37	115.23	127.64	105.60	15.15	45.58	28.78	18.54	
	C20:1n9c	5.91	4.35	1.07	0.33	nd	nd	0.96	nd	nd	
	C22:1n9c	0.50	4.01	0.11	nd	nd	nd	nd	nd	nd	
	C24:1n9c	0.87	0.64	0.11	nd	nd	nd	nd	nd	nd	
	C18:2n6t	nd	nd	nd	nd	nd	nd	0.01	0.02	nd	
	C18:2n6c	100.88	49.76	32.93	13.40	40.81	3.69	16.93	39.78	5.52	
	C18:3n3	48.42	21.24	4.23	0.65	1.16	0.30	5.18	3.12	0.04	
	C18:3n6c	nd	nd	0.19	nd	0.10	0.01	0.16	0.16	nd	
	C20:2	0.72	0.46	nd	nd	nd	nd	nd	nd	nd	
	C20:3n3c	0.04	0.03	nd	nd	nd	nd	nd	nd	nd	
	C20:3n6c	nd	nd	nd	nd	nd	nd	nd	nd	nd	
	C20:4n6	nd	nd	nd	nd	nd	nd	nd	nd	nd	
	C20:5n3c	nd	nd	nd	nd	nd	nd	nd	0.02	nd	
	C22:2n6c	nd	nd	nd	nd	nd	nd	nd	nd	nd	
	C22:5n3c	nd	nd	nd	nd	nd	nd	nd	nd	nd	
	C22:6n3c	nd	nd	nd	nd	nd	nd	nd	nd	nd	
	SFAs	32.06	19.78	125.27	227.56	235.06	64.26	25.10	9.69	55.75	
	MUFAs	279.60	169.54	120.20	130.14	107.87	15.26	48.51	30.27	18.97	
	PUFAs	150.06	71.48	37.35	14.05	42.07	4.00	22.28	43.10	5.56	

Table 4.11b. Fatty acid composition of plant-based/vegan food alternatives (continued).

FAs [mg/g]	Pie (v)	Burger (v)	Cheese (v)	'Chicken' Breast (v)	Brownie (v)	Bacon Rasher (v)	Pizza (v)	PO Free Spread (v)	Double Cream (v)
C4:0	nd	nd	nd	nd	nd	nd	nd	nd	nd
C6:0	0.01	0.17	0.44	nd	0.03	nd	nd	0.31	0.05
C8:0	0.00	2.60	6.64	0.03	0.34	0.02	nd	4.97	1.30
C10:0	nd	2.30	5.77	0.02	0.42	0.03	nd	4.59	2.06
C11:0	0.01	0.01	0.03	nd	nd	nd	nd	0.02	0.03
C12:0	0.15	nd	nd	0.12	5.62	0.03	0.02	38.28	nd
C13:0	nd	0.02	0.04	nd	0.01	nd	nd	0.03	0.05
C14:0	0.76	9.36	20.33	0.55	3.56	0.07	0.03	16.03	17.61
C15:0	0.05	0.01	0.02	0.06	0.07	0.03	0.01	0.09	0.06
C16:0	**34.76**	**7.65**	**13.12**	**29.21**	**48.64**	**5.21**	**1.99**	**25.71**	**40.17**
C17:0	0.11	0.02	0.02	0.11	0.16	0.06	0.02	0.26	0.11
C18:0	3.42	2.50	3.41	0.02	10.29	1.73	0.55	59.08	6.01
C20:0	nd	0.12	0.12	0.69	0.75	0.52	0.11	3.34	0.65
C21:0	nd	nd	nd	0.03	nd	nd	nd	nd	nd
C22:0	nd	0.18	0.04	0.87	0.24	0.28	0.10	1.05	0.29
C24:0	nd	0.18	0.05	0.33	0.14	0.14	0.04	0.53	0.15
C14:1	nd	nd	nd	nd	nd	nd	nd	nd	nd
C15:1n5c	nd	0.01	nd	nd	nd	nd	0.01	nd	nd
C16:1n7c	0.13	0.03	0.03	0.24	0.24	0.26	0.06	0.65	0.20
C17:1n7c	nd	nd	nd	nd	nd	nd	nd	nd	nd
C18:1n7c	0.83	nd	nd	nd	nd	2.95	nd	9.61	3.07
C18:1n9t	nd	nd	0.14	nd	nd	nd	0.02	nd	nd

C18:1n9c	30.45	10.82	9.54	63.74	61.58	51.52	10.15	221.23	58.05
C20:1n9c	nd	0.08	nd	nd	nd	nd	nd	nd	nd
C22:1n9c	nd	0.01	nd	0.14	0.13	0.11	0.06	1.39	0.25
C24:1n9c	nd	0.01	nd	0.08	0.07	0.13	0.03	0.48	0.14
C18:2n6t	0.01	0.01	nd	nd	nd	nd	nd	nd	nd
C18:2n6c	11.32	13.30	2.71	69.40	18.56	19.62	8.22	65.65	17.74
C18:3n3	1.10	0.18	0.06	3.86	3.65	6.12	1.39	21.66	6.60
C18:3n6c	0.05	nd	nd	nd	nd	nd	0.06	nd	nd
C20:2	0.01	0.01	0.01	0.13	0.12	0.11	0.02	0.64	0.13
C20:3n3c	nd	nd	nd	nd	nd	0.01	nd	0.03	0.01
C20:3n6c	nd	nd	nd	nd	nd	nd	nd	nd	nd
C20:4n6	nd	nd	nd	nd	nd	nd	nd	nd	nd
C20:5n3c	0.00	nd	nd	nd	nd	nd	nd	nd	nd
C22:2n6c	nd	nd	nd	nd	nd	nd	nd	nd	nd
C22:5n3c	nd	nd	nd	nd	nd	nd	nd	nd	nd
C22:6n3c	nd	nd	nd	nd	nd	nd	nd	nd	nd
SFAs	39.29	25.12	50.01	32.06	70.27	8.13	2.87	154.29	68.55
MUFAs	31.41	10.97	9.71	64.20	62.02	54.97	10.33	233.37	61.71
PUFAs	12.49	13.50	2.78	73.39	22.34	25.86	9.69	87.99	24.47

Data is presented as milligram (mg) total fatty acid (FA) content per 1 gram (g) fresh food.

'nd' indicates there were no FAs determined/detected.

SFAs = saturated fatty acids.

MUFAs = mono-unsaturated fatty acids.

PUFAs = poly-unsaturated fatty acids.

4.5.6 Miscellaneous/snack food items

A total of 14 miscellaneous/snack food items (ready salted crisps, vegetable straw crisps, milk chocolate, dark chocolate, fruit and nut bar, protein flapjack, oatcake, seeds & grains bread, 'palm oil free' bread, pizza, granola, avocado, pesto, olive spread) were analysed and the fatty acid profile is presented in Table 4.12. The predominant fatty acids within miscellaneous/snack food items were saturated fatty acid(s): C16:0 (palmitic acid), C18:0 (stearic acid); monounsaturated fatty acid(s): C18:1n9c (oleic acid); polyunsaturated fatty acid(s): C18:2n6c (linoleic acid). Ready salted crisps contain low level of SFA (25.9 mg/g), high MUFA (244.6 mg/g) and low PUFA content (32.6 mg/g). The major SFA, MUFA and PUFA present in ready salted crisps are palmitic acid (12.5 mg/g), oleic acid (232.3 mg/g) and linoleic acid (26.9 mg/g), respectively. Vegetable straw crisps contain low level of SFA (25.7 mg/g), high MUFA (213.4 mg/g) and medium PUFA content (83.6 mg/g). The major SFA, MUFA and PUFA present in vegetable straw crisps are palmitic acid (15.3 mg/g), oleic acid (194.9 mg/g) and linoleic acid (59.4 mg/g), respectively. Milk chocolate contains high level of SFA (188.9 mg/g), medium MUFA (99.4 mg/g) and low PUFA content (12.6 mg/g). The major SFA, MUFA and PUFA present in milk chocolate are palmitic acid (9.1 mg/g), oleic acid (96.6 mg/g) and linoleic acid (11.2 mg/g), respectively. Dark chocolate contains high level of SFA (406.7 mg/g), high MUFA (206.2 mg/g) and low PUFA content (19.4 mg/g). The major SFA, MUFA and PUFA present in dark chocolate are stearic acid (234.1 mg/g), oleic acid (106.7 mg/g) and linoleic acid (202.3 mg/g), respectively. Fruit & nut bar contains low level of SFA (37.5 mg/g), medium MUFA (108.6 mg/g) and low PUFA content (33.5 mg/g). The major SFA, MUFA and PUFA present in fruit & nut bar are palmitic acid (49.5 mg/g), oleic acid (73.6 mg/g) and linoleic acid (35.6 mg/g), respectively. Protein flapjack contains medium level of SFA (92.29 mg/g), medium MUFA (78.71 mg/g) and low PUFA content (42.9 mg/g). The major SFA, MUFA and PUFA present in protein flapjack are palmitic acid (49.5 mg/g), oleic acid (91.9 mg/g) and linoleic acid (46.3 mg/g), respectively. Oatcake contains medium level of SFA (88.3 mg/g), medium MUFA (95.2 mg/g) and low PUFA content (47.3 mg/g). The major SFA, MUFA and PUFA present in oatcake are palmitic acid (20.2 mg/g), oleic acid (271.3 mg/g) and linoleic acid (100.9 mg/g), respectively. Seeds & grains bread contains low level of SFA (9.4 mg/g), low MUFA (13 mg/g) and low PUFA content (20.4 mg/g). The major SFA, MUFA and PUFA present in seeds & grains bread are palmitic acid (6.5 mg/g), oleic acid (11.5 mg/g) and linoleic acid (18 mg/g), respectively. PO free bread contains low level of SFA (6.7 mg/g), low MUFA (5.3 mg/g) and low PUFA content (11.3 mg/g). The major SFA, MUFA and PUFA present in

PO free bread are palmitic acid (4.5 mg/g), oleic acid (4.6 mg/g) and linoleic acid (10.6 mg/g), respectively. Pizza contains low level of SFA (20.3 mg/g), low MUFA (11.9 mg/g) and low PUFA content (4.8 mg/g). The major SFA, MUFA and PUFA present in pizza are palmitic acid (11.1 mg/g), oleic acid (10.9 mg/g) and linoleic acid (68.8 mg/g), respectively. Granola contains high level of SFA (74.3 mg/g), medium MUFA (123.1 mg/g) and medium PUFA content (82.61 mg/g). The major SFA, MUFA and PUFA present in granola are palmitic acid (24.6 mg/g), oleic acid (19.3 mg/g) and linoleic acid (116.2 mg/g), respectively. Avocado contains low level of SFA (20.3 mg/g), medium MUFA (76 mg/g) and low PUFA content (12.1 mg/g). The major SFA, MUFA and PUFA present in avocado are palmitic acid (19.3 mg/g), oleic acid (68.7 mg/g) and linoleic acid (162.4 mg/g), respectively. Pesto contains medium level of SFA (50.1 mg/g), medium MUFA (124.2 mg/g) and high PUFA content (163.7 mg/g). The major SFA, MUFA and PUFA present in pesto are palmitic acid (28 mg/g), oleic acid (120 mg/g) and linoleic acid (162.4 mg/g), respectively. Olive spread contains medium level of SFA (119.4 mg/g), medium MUFA (251.2 mg/g) and medium PUFA content (67.6 mg/g). The major SFA, MUFA and PUFA present in olive spread are palmitic acid (85.4 mg/g), oleic acid (236.6 mg/g) and linoleic acid (52.1 mg/g), respectively. For the abundance of C16:0 (palmitic acid), high C16:0 in dark chocolate (160.4 mg/g), medium in milk chocolate (91.1 mg/g), olive spread (85.4 mg/g) and oatcake (77.5 mg/g), low in protein flapjack (49.5 mg/g), pesto (28 mg/g), granola (24.6 mg/g), avocado (19.3 mg/g), fruit & nut bar (18.2 mg/g), vegetable straw crisps (15.3 mg/g), ready salted crisps (12.5 mg/g), pizza (11.1 mg/g), seeds & grains bread (6.5 mg/g), and PO free bread (4.5 mg/g) were evident.

Table 4.12. Fatty acid composition of miscellaneous/snack food items.

FAs [mg/g]	Ready Salted Crisps	Vegetable Straw Crisps	Milk Chocolate	Dark Chocolate	Fruit & Nut Bar	Protein Flapjack	Oatcake	Seeds & Grains Bread	PO Free Bread	Pizza	Granola	Avocado	Pesto	Olive Spread
C4:0	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C6:0	nd	nd	nd	nd	0.12	0.08	nd	0.01	0.02	nd	nd	nd	nd	0.08
C8:0	0.04	nd	0.60	nd	0.04	nd	nd	0.01	0.01	nd	nd	nd	nd	0.75
C10:0	0.02	0.03	1.54	nd	0.01	1.00	0.03	nd	nd	nd	3.13	nd	0.44	0.80
C11:0	nd	nd	0.10	nd	0.02	0.05	nd	0.01	0.02	0.03	0.02	nd	0.02	nd
C12:0	0.02	0.03	2.24	0.05	0.03	21.48	0.33	0.02	0.01	1.12	25.67	0.01	0.60	9.87
C13:0	nd	nd	0.06	nd	0.01	0.04	nd	nd	nd	0.05	0.02	nd	0.04	0.02
C14:0	0.15	0.17	7.64	0.53	0.07	10.17	1.64	0.06	0.04	3.50	10.02	0.04	2.17	5.08
C15:0	0.05	0.08	0.85	0.19	0.04	0.07	0.09	0.03	0.02	0.39	0.05	0.01	0.27	nd
C16:0	**12.52**	**15.31**	**91.14**	**160.41**	**18.24**	**49.54**	**77.54**	**6.45**	**4.47**	**11.13**	**24.62**	**19.32**	**27.98**	**85.37**
C17:0	0.13	0.23	2.33	1.79	0.31	0.42	0.21	0.06	0.04	0.24	0.22	0.02	0.29	0.35
C18:0	8.35	6.12	78.70	234.12	16.82	7.99	7.38	2.34	1.83	3.82	8.58	0.53	13.85	13.87
C20:0	1.14	1.91	2.54	7.46	1.13	0.83	0.68	0.17	0.08	nd	0.91	0.06	0.98	1.96
C21:0	0.11	0.24	0.35	0.11	0.02	0.02	0.02	0.02	nd	nd	0.07	0.05	0.11	0.10
C22:0	2.44	1.20	0.48	1.30	0.27	0.39	0.17	0.15	0.06	nd	0.58	0.19	2.44	0.77
C24:0	0.90	0.41	0.30	0.77	0.32	0.21	0.22	0.10	0.05	nd	0.35	0.06	0.90	0.40
C14:1	nd	nd	0.66	nd	0.02	nd	nd	nd	nd	0.30	nd	nd	0.18	0.04
C15:1n5c	nd	nd	nd	nd	0.05	0.02	nd	nd	nd	nd	nd	nd	nd	0.15
C16:1n7c	0.42	0.65	1.74	1.51	0.61	0.29	0.37	0.19	0.13	0.59	0.48	6.99	0.92	1.70
C17:1n7c	nd	nd	nd	nd	0.02	nd	nd	nd	nd	nd	nd	nd	nd	nd

C18:1n7c	3.57	9.63	0.09	2.13	0.78	2.99	1.72	0.54	0.27	0.11	3.84	nd	2.32	9.60
C18:1n9t	nd	nd	nd	nd	nd	0.13	nd	0.30	0.01	0.01	nd	nd	nd	nd
C18:1n9c	238.26	194.94	96.57	202.28	106.72	73.55	91.91	11.54	4.61	10.88	116.24	68.74	120.01	236.58
C20:1n9c	1.88	5.71	0.31	0.30	0.37	1.50	1.00	0.26	0.14	nd	2.02	0.24	0.59	2.47
C22:1n9c	0.32	1.78	0.01	nd	0.01	nd	0.07	0.07	0.03	nd	0.26	0.01	0.02	0.34
C24:1n9c	0.17	0.67	0.03	nd	nd	0.22	0.16	0.09	0.06	nd	0.29	nd	0.13	0.36
C18:2n6t	0.01	0.04	nd	nd	0.01	nd	0.02	0.01	nd	nd	nd	nd	0.04	nd
C18:2n6c	26.90	59.40	11.22	18.24	33.06	35.57	46.33	18.02	10.55	3.97	68.84	11.12	162.40	52.07
C18:3n3	5.72	23.46	1.14	1.13	0.40	6.96	0.94	2.28	0.70	0.73	13.37	0.98	1.14	15.22
C18:3n6c	nd	nd	nd	nd	nd	0.33	0.05	0.03	0.01	0.04	0.38	nd	0.04	nd
C20:2	nd	0.63	nd	nd	nd	nd	nd	0.04	0.03	nd	nd	0.03	nd	0.28
C20:3n3c	nd	0.02	nd	nd	nd	nd	nd	nd	nd	nd	0.01	nd	nd	nd
C20:3n6c	nd	nd	0.06	nd	nd	nd	nd	nd	nd	nd	nd	nd	0.02	nd
C20:4n6	nd	nd	0.08	nd	nd	nd	nd	nd	nd	nd	nd	nd	0.03	nd
C20:5n3c	nd	nd	0.04	nd	nd	nd	0.01	0.01	nd	0.02	0.01	nd	nd	nd
C22:2n6c	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C22:5n3c	nd	nd	0.06	nd	nd	nd	nd	nd	nd	nd	nd	nd	0.02	nd
C22:6n3c	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
SFAs	25.88	25.72	188.87	406.74	37.47	92.29	88.29	9.44	6.66	20.28	74.25	20.31	50.11	119.42
MUFAs	244.61	213.38	99.42	206.22	108.57	78.71	95.24	12.99	5.25	11.90	123.13	75.98	124.18	251.24
PUFAs	32.64	83.56	12.61	19.38	33.47	42.85	47.34	20.39	11.30	4.77	82.61	12.13	163.69	67.57

Data is presented as milligram (mg) total fatty acid (FA) content per 1 gram (g) fresh food.

'nd' indicates there were no FAs determined/detected.

SFAs = saturated fatty acids.

MUFAs = mono-unsaturated fatty acids.

PUFAs = poly-unsaturated fatty acids.

4.5.7 Palm-based foods

A total of 7 palm-based food (palm sugar, unrefined palm oil, pure palm oil, palm kernel oil, palm oil nut cream, palm oil nut soup and palm oil fruit pulp) were analysed and the fatty acid profile is presented in Table 4.13. The predominant fatty acids within palm-based food items were determined to be saturated fatty acid(s): C16:0 (palmitic acid) and C18:0 (stearic acid); monounsaturated fatty acid(s): C18:1n9c (oleic acid); and polyunsaturated fatty acid(s): C18:2n6c (linoleic acid). Palm sugar contains low level of SFA (2 mg/g), low MUFA (0.1 mg/g) and low PUFA content (0.1 mg/g). The major SFA, MUFA and PUFA present in palm sugar are heptadecanoic acid (1.8 mg/g), oleic acid (0.1 mg/g) and linoleic acid (0.1 mg/g), respectively. Unrefined palm oil contains high level of SFA (492.9 mg/g), high MUFA (441 mg/g) and medium PUFA content (110.1 mg/g). The major SFA, MUFA and PUFA present in unrefined palm oil are palmitic acid (420.5 mg/g), oleic acid (432 mg/g) and linoleic acid (106.9 mg/g), respectively. Pure palm oil contains high level of SFA (543.4 mg/g), high MUFA (420.3 mg/g) and medium PUFA content (111.9 mg/g). The major SFA, MUFA and PUFA present in pure palm oil are palmitic acid (467.8 mg/g), oleic acid (410.6 mg/g) and linoleic acid (108.5 mg/g), respectively. Palm kernel oil contains high level of SFA (490.9 mg/g), medium MUFA (111.7 mg/g) and low PUFA content (17.9 mg/g). The major SFA, MUFA and PUFA present in palm kernel oil are lauric acid (292.7 mg/g), oleic acid (110.4 mg/g) and linoleic acid (17.5 mg/g), respectively. Palm nut cream contains high level of SFA (490.9 mg/g), medium MUFA (111.7 mg/g) and low PUFA content (17.9 mg/g). The major SFA, MUFA and PUFA present in palm nut cream are palmitic acid (176.4 mg/g), oleic acid (169 mg/g) and linoleic acid (43.2 mg/g), respectively. Palm nut soup contains high level of SFA (207.9 mg/g), high MUFA (175.8 mg/g) and low PUFA content (43.4 mg/g). The major SFA, MUFA and PUFA present in palm nut soup are palmitic acid (181.1 mg/g), oleic acid (172.6 mg/g) and linoleic acid (42.2 mg/g), respectively. Palm nut soup contains high level of SFA (207.9 mg/g), high MUFA (175.8 mg/g) and low PUFA content (43.4 mg/g). The major SFA, MUFA and PUFA present in palm nut soup are palmitic acid (181.1 mg/g), oleic acid (172.6 mg/g) and linoleic acid (42.2 mg/g), respectively. Palm fruit pulp contains high level of SFA (175.5 mg/g), high MUFA (148.1 mg/g) and low PUFA content (39.6 mg/g). The major SFA, MUFA and PUFA present in palm fruit pulp are palmitic acid (151 mg/g), oleic acid (145 mg/g) and linoleic acid (38.4 mg/g), respectively. For the abundance of C16:0 (palmitic acid), high C16:0 in pure palm oil (467.8 mg/g), unrefined palm oil (420.5 mg/g), palm nut soup (181.1 mg/g), palm nut cream

(176.4 mg/g), and palm fruit pulp (150.1 mg/g), medium in palm kernel oil (56.9 mg/g), low in palm sugar (0.1 mg/g) were evident.

Table 4.13. Fatty acid composition of palm-based foods.

FAs [mg/g]	Palm Sugar	Unrefined Palm Oil	Pure Palm Oil	Palm Kernel Oil	Palm Nut Cream	Palm Nut Soup	Palm Fruit Pulp
C4:0	nd	nd	nd	nd	nd	nd	nd
C6:0	0.04	nd	nd	nd	nd	nd	nd
C8:0	nd	nd	nd	nd	nd	nd	nd
C10:0	nd	0.16	0.21	19.95	nd	nd	nd
C11:0	0.05	nd	nd	0.26	nd	nd	nd
C12:0	0.01	1.56	2.52	292.66	1.23	1.92	1.01
C13:0	nd	nd	nd	0.34	nd	nd	nd
C14:0	0.01	7.14	10.91	103.57	3.70	3.92	3.34
C15:0	nd	0.51	0.54	0.10	0.18	0.20	0.17
C16:0	**0.10**	**420.53**	**467.84**	**56.93**	**176.37**	**181.11**	**150.95**
C17:0	1.80	1.02	1.18	0.16	0.43	0.43	0.37
C18:0	0.03	57.69	55.39	16.95	24.13	24.18	18.22
C20:0	nd	4.32	4.77	nd	1.89	1.90	1.48
C21:0	nd	nd	nd	nd	nd	nd	nd
C22:0	nd	nd	nd	nd	nd	nd	nd
C24:0	nd	nd	nd	nd	nd	nd	nd
C14:1	nd	nd	nd	nd	nd	nd	nd
C15:1n5c	0.01	nd	nd	nd	nd	nd	nd
C16:1n7c	nd	1.37	1.48	0.14	0.48	0.49	0.53
C17:1n7c	nd	nd	nd	nd	nd	nd	nd
C18:1n7c	nd	6.07	6.66	1.01	2.11	1.96	2.07
C18:1n9t	nd	nd	nd	0.16	nd	nd	nd

C18:1n9c	0.13	432.04	410.57	110.41	168.97	172.63	145.04
C20:1n9c	nd	1.50	1.60	nd	0.63	0.67	0.51
C22:1n9c	nd	nd	nd	nd	nd	nd	nd
C24:1n9c	nd	nd	nd	nd	nd	nd	nd
C18:2n6t	nd	nd	nd	nd	nd	0.01	nd
C18:2n6c	0.06	106.87	108.52	17.45	43.20	42.16	38.39
C18:3n3	0.01	3.25	3.36	0.40	1.28	1.21	1.17
C18:3n6c	nd	nd	nd	0.04	0.01	0.01	0.01
C20:2	nd	nd	nd	nd	nd	nd	nd
C20:3n3c	nd	nd	nd	nd	nd	nd	nd
C20:3n6c	nd	nd	nd	nd	nd	nd	nd
C20:4n6	nd	nd	nd	nd	nd	nd	nd
C20:5n3c	0.03	nd	nd	nd	nd	nd	nd
C22:2n6c	nd	nd	nd	nd	nd	nd	nd
C22:5n3c	nd	nd	nd	nd	nd	nd	nd
C22:6n3c	nd	nd	nd	nd	nd	nd	nd
SFAs	2.03	492.91	543.37	490.92	207.92	213.66	175.54
MUFAs	0.14	440.98	420.32	111.73	172.19	175.75	148.15
PUFAs	0.09	110.12	111.88	17.89	44.49	43.40	39.57

Data is presented as milligram (mg) total fatty acid (FA) content per 1 gram (g) fresh food.

'nd' indicates there were no FAs determined/detected.

SFAs = saturated fatty acids.

MUFAs = mono-unsaturated fatty acids.

PUFAs = poly-unsaturated fatty acids

4.6 Discussion

This study analysed the fatty acid composition of a total of 70 common everyday food items, based on their prevalence within Westernised diets, or their relevance to the thesis aims and objectives. As highlighted in Chapter 3 of this thesis, overall comprehension of palm oil and palmitic acids' presence/abundance within the diet is lacking within United Kingdom consumers. Additionally, the authors' own estimations of palmitic acid intake of the average consumer presented difficulties, based on the available literature. While fatty acid composition data (including palmitic acid) of a large number of foods is publicly available through databases such as the McCance and Widdowson Composition of Foods Integrated Dataset (CoFID); several of the food entries are incomplete, out-of-date, or absent entirely (particularly plant-based/vegan foods). Consequently, to the authors' knowledge, this study is the first to investigate the fatty acid composition of several plant-based/vegan foods, typically those available as alternatives to meat and/or dairy products.

Unsurprisingly, the highest content of palmitic acid was observed within palm oil itself followed by palm-based foods. Non-palm foods with notably high content of palmitic acid included dark chocolate and margarine. Other high palmitic acid foods included several of the edible oils, dairy-foods (butter, cheeses) nut/seed-based foods, particularly Brazil nuts and tahini, plant-based/vegan food alternatives including vegan chocolate cookie, butter, and chocolate, as well as some of the miscellaneous/snack foods including milk chocolate and oatcakes. Animal-origin foods contained low contents of palmitic acid, yet high contents of oleic acid. A visualisation of mean palmitic acid content within all food group categories is available to view in Figure 4.4.

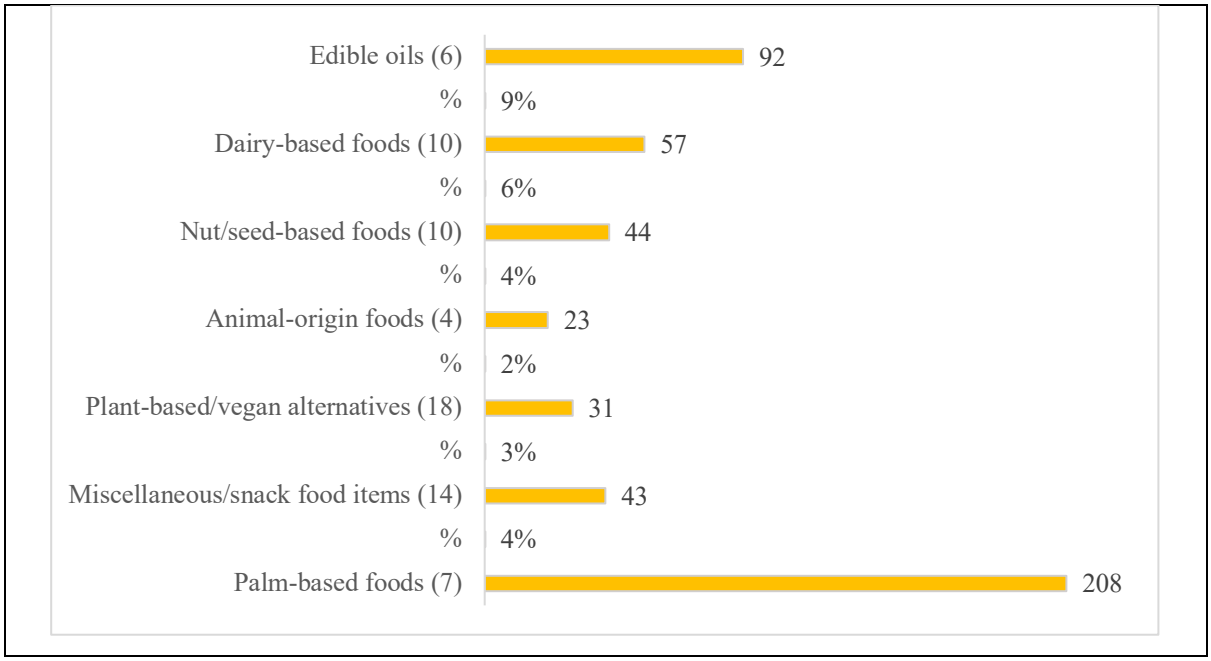


Figure 4.4. The mean (mg/g, %) palmitic acid content per food group category.

Based on the findings of this study, the predominant saturated fatty acid observed within dairy foods is palmitic acid, particularly in margarine, butter, and various cheeses (cheddar, feta, parmesan, and goat cheese). Palmitic acid is also the most abundant fatty acid overall in the majority of analysed dairy foods (excluding double cream and coleslaw). Goat cheese is particularly high (comparatively) in capric acid, associated with anti-neurodegenerative effects (Shekhar et al., 2023). Interestingly, while margarine, typically classed as an ultra-processed food (UPF) (Monteiro et al., 2019), contains the highest content (amongst dairy foods) of palmitic acid (~15%), it also contains oleic acid (~31%). Similarly, butter contains ~8%, and ~15%, respectively. Oleic acid has been associated with various health benefits, such as reductions in heart disease and cancer risk, through blood pressure and inflammatory cytokine regulation, as well as the inhibition of type 2 diabetes via induced insulin production (Sales-Campos et al., 2013; Teres et al., 2008). Margarine and butter also contain (comparatively) high levels of the omega-6 (ω-6) and omega-3 (ω-3) fatty acids, linoleic acid, and α-linoleic acid, respectively. Omega-3 fatty acids are typically considered to possess favourable effects on health (Yashodhara et al., 2009), whereas omega-6 fatty acid have been associated with various adverse health risks (Patterson et al., 2012). The Standard American Diet (SAD), commonly referred to as the 'Western' diet, contains significantly more omega-6 content, when compared to omega-3 (Mercola & D'Adamo, 2023). Recent studies have observed favourable

effects of both omega fatty acids on cardiovascular disease, although an exact ratio is still not widely accepted (Djuricic & Calder, 2021).

Animal-origin foods contained (comparatively) very low contents of palmitic acid, and saturated fat content overall, with beef burger containing the most at ~4%, and ~7%, respectively. Other prominent fatty acids of animal-origin foods include stearic acid, oleic acid, and linoleic acid. Recent studies have investigated the complicated relationship between stearic acid and metabolic health, observing favourable health effects such as protection against atherosclerosis and neurodegenerative diseases, yet unfavourable effects towards type 2 diabetes, as well as tumour growth and metastasis (Shen et al., 2025). Beef burger contains a higher proportion of myristic acid, associated with adverse effects on cholesterol, and insulin sensitivity (Saraswathi et al., 2022; Temme et al., 1997). The outcomes suggest dissimilarities between beef-products and pork-products, more specifically, linoleic acid (omega-6) is higher in both pork sausage and bacon rashers. Beef foods, and beef burgers in particular, contain higher amounts of SFAs and MUFAs, yet a very low proportion of PUFAs. Intriguingly, efforts have been made to investigate the creation of 'healthier' burgers, chiefly through increased PUFA (ω-3) content (Heck et al., 2017; Heck et al., 2019). Red meats, whether unprocessed or processed, are typically classified as carcinogenic, due to naturally-occurring contents of heme iron (Seiwert et al., 2020), or the manufactured addition of nitrites and smoke-flavourings (Iko Afé et al., 2021; Shakil et al., 2022). Conversely, animal-origin foods such as beef and pork are also particularly high in both protein and phospholipid content, nutrients typically associated with beneficial effects on cardiovascular health (Küllenberg et al., 2012; Pederson et al., 2013).

Cooking of meats can also significantly affect lipid profile. Various cooking methods, such as frying or grilling, can degrade (break-down) unsaturated fatty acids in a process named lipid (fat) oxidation (Domínguez et al., 2019). Fat oxidation reduces overall unsaturated fatty acid content, leading to changes in the saturated/unsaturated fatty acid ratio, as well as the formation of hydroperoxides (Geng et al., 2023). Peroxidation of unsaturated fatty acids produces various derivatives such as lipid radicals (a type of free radical, reactive oxygen species); elevated circulating levels of reactive oxygen species (ROS) can induce imbalances in pro- and anti-oxidant homeostasis (oxidative stress), implicated in inflammation, neurodegeneration, obesity, diabetes, cardiovascular disease, and certain cancers (Huang et al., 2019). Peroxidation can also produce 4-hydroxynonenal, an aldehyde associated with cytopathogenesis and

carcinogenesis (Csala et al., 2015). Foods rich in monounsaturated and polyunsaturated fatty acids are most vulnerable to cooking-related deterioration, due to unsaturated fat's instability at high temperatures, a characteristic not shared in saturated fats (Szabo et al., 2022).

Edible oils are particularly high in monounsaturated and polyunsaturated fats. Atypically consumed in isolation (mostly used as an addition to, or as part of, a meal; i.e., frying meats, sautéing vegetables, drizzling over salads), edible oils can also significantly increase the fatty acid content of consumed foods. Rapeseed oil, arguably the most commonly consumed cooking oil in the UK (mainly due to generic 'vegetable oil' products being canola/rapeseed), contains a relatively low content of palmitic acid (~4%), yet high oleic acid (~56%). Similarly, olive oil also contains particularly high content of oleic acid (~84%), although also contains the highest content of palmitic acid within edible oils (excluding red palm oil & rapeseed oil), at ~12%. Edible oils are predominantly high in MUFA (rapeseed oil, olive oil) or PUFA (sunflower oil, sesame oil) content, chiefly oleic acid or linoleic acids. Inversely, coconut oil contains a wide range of various saturated fatty acids, lauric acid (~47%), myristic acid (~19%), palmitic acid (~10%), capric acid (~6%), and stearic acid (~3%). Despite being intensely high in saturated fat content (~86%), coconut oil has generally been linked to favourable cardiometabolic health effects over the past two decades (Boateng et al., 2016; Kappally et al., 2015). Intriguingly, many of these studies circulated during the peak of its' popularity in the early-to-mid 2010s (Lockyer & Stanner, 2016), perhaps concurrent with the rise in plant-based diets/veganism. Recent studies re-examining coconut oil have challenged these perceived health benefits, as well as suggested or reported adverse health effects on cardiovascular health (Jayawardena et al., 2021; Spiazzi et al., 2023). Many of the favourable associations of coconut oil have been attributed to lauric acid, although more extensive and applicable research is needed (Ameena et al., 2024).

For nut/seed-based foods, Brazil nuts and tahini contain the highest contents of palmitic acid, at ~9% and ~8%, respectively. Most nut/seed-based foods are rich sources of linoleic acid, while walnuts contain considerably more α-linoleic acid. Interestingly, sunflower seeds contain negligible omega-3 or omega-6 fatty acids. Due to their overall fatty acid composition (high MUFA, high PUFA, low SFA); the majority of nut/seed-based foods are optimal food choices to adhere to the clinical recommendations for fat intake, generally advocating the reduction of saturated fat, alongside increased polyunsaturated and monounsaturated fat intake (American Heart Associations, 2017). Nuts are also a rich source of phytosterols, particularly walnuts,

which exhibit anti-oxidant, anti-inflammatory and hypocholesterolaemic properties (Nguyen & Vu, 2023).

Interestingly, high-quality, or high percentage cocoa (in this case, 85%), dark chocolate contains particularly high palmitic acid (~16%), as well as stearic acid content (~23%). There appears to be no data on low-quality (low percentage cocoa) dark chocolate's palmitic acid content within the literature. Despite high content of saturated fat (~41%), many studies over the past few decades have associated dark chocolate with various beneficial effects within cardiometabolic-related disorder/disease, chiefly due to its high polyphenol (flavonoid) content (García & Hernández, 2020; Haritha et al., 2014; Shin et al., 2022). Consumption of flavanols, potent anti-oxidant flavonoids, reportedly induces the lowering of blood pressure (Amoah et al., 2022), improved cognitive function (Zhu et al., 2021), improved insulin sensitivity and secretion (Shah et al., 2017), as well as protection against cardiovascular disease (Kozłowska & Szostak-Węgierek, 2022). Dark chocolate also contains ~20% oleic acid. Both crisp food items (ready salted, vegetable straw) also contain high content of oleic acid (~24% and ~19%, respectively) most likely due to olive oil being used as an ingredient. Comparatively, milk chocolate (~9% palmitic acid, ~19% saturated fatty acid content), does not share the same health benefits as dark chocolate (Liu et al., 2024), perhaps due to considerably less flavonoid and cocoa content (Latif, 2013), as well as higher milk-fat (Shukla, 1994) and sugar content (56g in the milk chocolate sample used within this study, versus 15g in dark chocolate; tesco.com).

The NOVA classification system, which categorises processed foods based on various criteria, classifies 'chocolate' as an ultra-processed food (Dinu & Martini, 2023). The classification has been challenged (Ditchfield et al., 2023), however there appears to be no clear distinction (in either source) between dark chocolate or milk chocolate, instead opting to use the much broader, all-encompassing term. The ingredients (label list) of both products reveal distinct differences, indicating dark chocolate to be minimally processed, and milk chocolate as an ultra-processed food. Consequently, these two food items alone provide evidence that high saturated fat, or palmitic acid content alone does not indicate risk to health, as there are various other factors.

Comparison of plant-based alternatives to prevalent animal-based and dairy food items, highlighted various differences. Plant-based butter for example, while containing comparable levels of palmitic acid (~8% and ~10%, respectively) contains considerably more saturated fats

than dairy butter, principally due to lauric acid content. Similarly, plant-based chocolate contains comparative contents of palmitic acid (~9%), yet more stearic acid and oleic acid content than milk chocolate. High palmitic acid levels within both of these foods (both the dairy and plant-based versions) can most likely be attributed to palm oil, or its derivatives palm olein and palm stearin (Sulaiman et al., 2022). Dairy cheese (cheddar) contains significantly more palmitic acid than plant-based cheese, at ~8% and ~1%, respectively, dairy cheddar contains significantly more oleic acid, yet neither contain any (detected) lauric acid. Dairy ice cream contains comparable palmitic acid, myristic acid, stearic acid and oleic acid content to plant-based ice cream, yet plant-based ice cream contains more lauric acid (none detected in dairy ice cream). Similarly, in dairy double cream, lauric acid is considerably higher in the plant-based version, alongside higher palmitic acid and oleic acid. Animal-origin beef contains higher oleic acid than plant-based beef, however plant-based pork contains more oleic acid than animal-origin pork. Plant-based pork also contains higher levels of both α-linoleic acid and linoleic acid. A visualisation of the palmitic acid content in animal-origin foods and their plant-based alternatives is available to view in Figure 4.5.

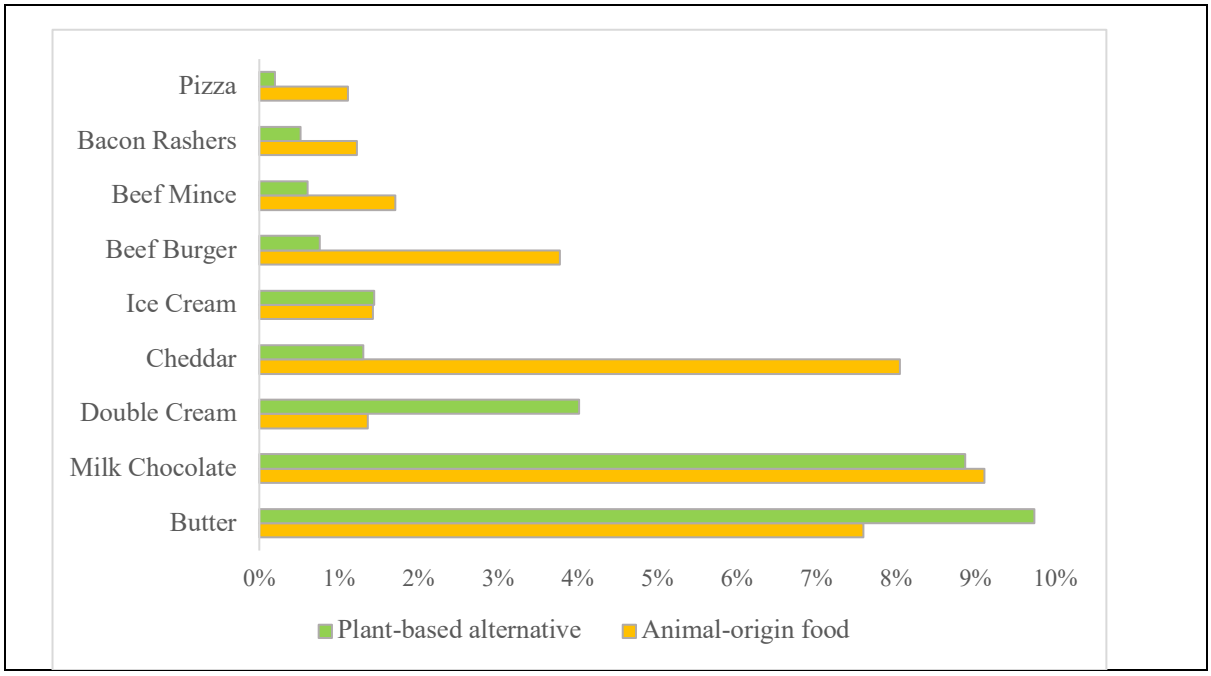


Figure 4.5. Palmitic acid content of animal-origin foods and their plant-based alternative.

In the United Kingdom, the Scientific Advisory Committee on Nutrition (SACN) advises total fats and saturated fats to be consist of approximately 33%, and 10-11% of total energy intake,

respectively (Public Health England, 2019). In the years 2019-2023, total fat and saturated fat consumption of United Kingdom consumers (aged 19-64 years old) accounted for ~33% (62.5g) and ~12% (22.6g) of total energy intake (per day), respectively (Office for Health Improvement and Disparities, 2025). The largest contributors to total fat consumption were cereal or cereal-products, milk or milk-products (yoghurt, cheese), as well as meat or meat-products (including processed meats), at 30%, 12%, and 19%, respectively. The largest contributors to saturated fat consumption were consistent with total fats', at 31%, 20%, and 18%, respectively. Although, cereals are typically low in both total fat and saturated fat content. In an analysis of common UK breakfast cereals, the total fat content and saturated fat content were between 0.6-2.9%, and 0.2-1.6%, respectively (Khehra et al., 2018). Additionally, the OHID survey includes wheat or grain food products such as pizza, pasta-based dishes, sandwiches, and buns, cakes, pastries in their cereal and cereal-products category. Nevertheless, approximately 1/5 of a consumers total saturated fat intake per day is reportedly sourced from dairy-foods alone, with around the same coming from meats, or processed meats.

The diverse fatty acid profiles of foods, including palmitic acid, may contribute to various aspects of metabolic health in many different ways. Therefore, perhaps classification of fatty acids by saturation, or as saturated versus unsaturated fats, requires re-examination. Instead, perhaps a greater focus on the source, i.e., dairy, meat, versus plant-origin, processed versus unprocessed, including how their unique fatty acid arrangement, as well as their non-fatty acid (protein, polyphenol, iron) content, affects lipid bioavailability and function, could be areas of future research. Regardless, continued and copious epidemiological study is essential to understanding the role of fatty acids, including palmitic acid, within metabolic health and chronic disease development.

Analysis of a large number of varied and prevalent Westernised diet food items, across multiple different food categories, provides evidence for the continued innovations in lipidomics, as well as contributions to the field of food science and human nutrition. In addition, the first-time analysis of a wide range of plant-based/vegan alternatives provide a novel insight into the diverse health effects of a plant-based/vegan diet, as well as an in-depth look at their palmitic acid content, when compared to their animal-origin equivalent. Palmitic acid differences between the findings of this study, and the CoFID study, are available to view in Appendix 4.11.

This research has several limitations. Firstly, while this study targeted the fatty acid composition data of 79 food products, this is still a very small proportion of food products available to consumers within the UK. Further comprehensive study is both needed and recommended to better represent the vast and increasing body of food products available. Secondly, it is difficult to discern whether the presence of high palmitic acid content alone indicates metabolic health concerns for the consumer. Therefore, further research is needed to understand if low, or high dietary intake of palmitic acid is associated with any beneficial, or adverse metabolic health effects (Chapter 5 of this thesis).

Additionally, further research is essential in addressing the wider consumption of total fatty acids, as well as palmitic acid, for the average UK consumer, as literature at the current time is applying estimations. Third, this study is based on the collection of food samples at one time point during the year, where possible batch-to-batch variations of fatty acid composition, including palmitic acid, could provide (even slight) differences in the representation of fatty acid content, based on factors such as seasonality, weather, or harvest/yield availability restraints. Similarly, many foods available to consumers consisting of the same or similar description i.e., milk chocolate, have numerous variations in ingredients, as well as nutritional compositions, chiefly due to differences in supplying county of origin, brand, or cost. Lastly, the study resulted in 9 failed food samples (at the author's discretion), due to inaccuracies or implausibilities' in their outcome data (Appendix 4.12a, 4.12b). If the research methodology was to be replicated or expanded upon within future study, perhaps a composite sampling (at different time points) approach could be adopted, alongside multiple (more than 2) sampling for each food sample, to increase accuracies and alleviate failure rate.

4.7 Conclusions

Various foods within Westernised diets containing palmitic acid, from multiple distinct food groups, are also sources of numerous other fatty acids, Additionally, other nutrients (carbohydrates, proteins) and bioactive compounds (phytochemicals), as well as non-nutritional ingredients such as preservatives, colourings and sweeteners can further contribute to metabolic health status, and chronic disease development. Plant-based/vegan food alternatives to animal-origin and dairy foods, typically contain less palmitic acid than their animal-origin counterparts. The effects of different fatty acid compositions of foods, more specifically, their unique saturated and unsaturated fat profile, significantly influence the food

matrix in a myriad of ways, although their microstructure, the extent of their impact, as well as their interactions with one another, are not fully understood.

4.7.1 Acknowledgements

This study was funded by the School of Agriculture and Food Science at University College Dublin (UCD). Thank you to Dr Marco Garcia-Vaquero and Dr Raquel Cama-Moncunill of UCD for their invaluable guidance and support.

Chapter 5. An assessment of gene expression biomarkers linked to metabolic health in participants with different levels of dietary palmitic acid consumption

5.1 Chapter Overview

Some of the leading causes of death worldwide are cardiovascular disease, diabetes and cancer - all closely linked to dietary fat. The most prevalent saturated fatty acid in most dietary habits is palmitic acid (16:0). Palmitic acid promotes adversely effects on metabolic health, mainly through inflammation, oxidative stress and insulin resistance. The main objectives of this study were to: screen a panel of gene expression biomarkers in participants with different levels of dietary palmitic acid intake and assess their potential role in metabolic health. A total of 18 participants included in this study. Anthropometry and food diary (1-week) were collected. Intravenous blood sample (on one occasion) was collected from each participant. Participant's palmitic acid intake data was calculated using a total of three resources: Nutritics, Widdowson's the Composition of Foods and Integrated Dataset (CoFID) 2021, in addition to fatty acid composition data obtained from Chapter 4 of this thesis. Participants were allocated into one of the palmitic acid intake groups (low, medium and high) based on their daily palmitic acid intake. Individuals with 'Low' palmitic acid content consumed fewer calories (P <0.001), total carbohydrates (P = 0.002), total fats (P <0.001), saturated fat (P = 0.003), and total protein (P <0.001), per day, compared to the 'High' intake group = 0.050). Concerning mRNA expression, the 'Low' dietary palmitic acid intake group exhibited several health benefits, when compared to 'High', implicating dietary palmitic acid intake in the progression of several cardiometabolic diseases. Interestingly, the 'Medium' palmitic acid group may induce a more unfavourable gene expression profile, than contents of high palmitic acid. There is evidence to suggest that palmitic acid intake is positively associated with an undesirable gene expression profile, although metabolic adaptation(s) may occur above a certain threshold or due to long-term exposure of palmitic acid. Prospective randomized control trials are critical in investigating these suppositions further.

5.2 Introduction

Chronic diseases are rapidly increasing worldwide, with heart disease, diabetes, and cancer being some of the leading causes of death (Hacker, 2024). Dietary fat contributes to metabolic health, influencing processes such as energy balance, insulin sensitivity, glucose tolerance, blood pressure and blood lipid profile (Riccardi et al., 2004). There are various forms of dietary fat, more specifically, different types of fatty acids; each possessing distinctive effects on metabolic health. Unsaturated fatty acids (monounsaturated, polyunsaturated) are typically considered healthy, primarily due to their role within cardiovascular disease prevention (Lunn & Theonbald, 2006). Despite this, an excess or imbalance of particular unsaturated fats: polyunsaturated fatty acids omega-6 (ω-6) and omega-3 (ω-3), can produce health complications (Candela et al., 2011). Saturated fatty acids (SFAs) are well-established to possess various adverse health effects including chronic inflammation, insulin resistance, as well as increased oxidative stress and raised LDL-cholesterol levels. Trans fats are similarly believed to possess negative health effects; however, when compared to both unsaturated and saturated fatty acids, they have either been eradicated or appear in particularly small amounts in modern households.

In Westernised dietary habits, saturated fat is consumed at much higher levels than what is recommended (NHS, 2023). Palmitic acid (C16:0), a long-chain saturated fatty acid, is the most abundant saturated fatty acid within the diet, and a key risk factor in most chronic diseases (Carta et al., 2017). In chronic inflammation, by activation of toll-like receptor 4 (TLR4), palmitic acid stimulates the body's inflammatory response, via transcription factor proteins such as nuclear kappa-factor-light-chain-enhancer of activated B cells (NF-κB) that induces the expression of pro-inflammatory cytokines (Korbecki & Bajdak-Rusinek, 2019). For example, interleukin-1, 6 (IL-1, IL-6) and tumour necrosis factor alpha (TNF-α) are important pro-inflammatory cytokines induced by NF-kB (Zhou et al., 2013). Palmitic acid affects insulin resistance through influence or disruption of various processes/pathways; including the aforementioned effects of inflammation, pancreatic/endoplasmic reticulum (ER) stress, as well as the inhibition of the insulin signalling pathway. Excessive activities of pro-inflammatory cytokines IL-1, IL-6, IL-1β and TNF-α can induce insulin resistance (Shoelson et al., 2006). An increase in the expression of endoplasmic reticulum stress markers (unfolded protein response) has been observed in pancreas cells exposed to palmitic acid, including glucose-related protein 78 (GRP78) and UPR X-box binding protein (XBP1) (Ben-Dror & Birk, 2019).

High levels of palmitic acid reportedly impair the insulin signalling pathway by affecting the phosphorylation of insulin receptor substrate-1 (IRS-1) (Reynoso et al., 2003). While oxidative stress is an expected, naturally occurring and healthy physiological mechanism, palmitic acid induces (increased) oxidative stress through an uptick in reactive oxygen species (ROS) production. Reactive oxygen species cause cellular damage through oxidation of DNA, proteins and lipids, often inducing cell stress response and ultimately, apoptosis (cell death) (Checa & Aran, 2020).

The harmful effects of palmitic acid on health could be strongly linked to its increased presence in foods/food ingredients, resulting in chronic exposure. However, as to what denotes 'increase/high' consumption remains unclear; as there appears to be no recommended daily intake/recommended dietary allowance, be that exact gram amount or within-parameter proposals available within clinical, educational and governmental institutions or within the literature. Myristic acid, also a saturated fatty acid, can exhibit similar adverse health effects as palmitic acid (Muñoz-Alvarez et al., 2024; Zock et al., 1994). However, it is a minor fatty acid and is present at a much lower abundance in most diets compared to palmitic acid. Conversely, other major saturated fatty acids such as stearic acid and lauric acid, have reported positive health effects (Dayrit, 2015; Hunter et al., 2010).

Following consumption of dietary lipids (fats) the postprandial response to lipids (fats) begins with the digestion of triacylglycerols (triglycerides) and phospholipids, through mastication as well as hydrolysis through the digestive enzyme salivary (or lingual) lipase (Liao et al., 1984). Once reaching the stomach, triacyclglycerols are further broken down by gastric lipase before entering the small intestine (duodenum) (Iqbal & Hussain, 2009). Here, bile emulsifies lipids, allowing for pancreatic lipase to break down the triacylglycerols further still into free fatty acids and monoglycerides, forming micelles. Micelle formation allows for transportation and absorption across microvilli and epithelial enterocytes (cells). Enterocytes then reconstitute the lipid particles into chylomicrons (triglyceride-rich lipoproteins), in order to transport the digested dietary lipids through the lymphatic system, and into the bloodstream (Feingold, 2024). Triglycerides are then distributed to (all) body tissue(s), including muscle cells for energy (Goodpaster & Kelley, 1998) and adipose tissue for fat storage (Ahmadian et a., 2007; Nye et al., 2008).

Gene expression profiling helps locate biomarkers linked to how our body interacts with dietary nutrients and physiological processes associated with metabolic health (Lagoumintzis & Patrinos 2023). Peripheral blood samples allow for comprehensive profiling of diet-induced gene transcripts (messenger ribonucleic acid, mRNA), a powerful technique for identifying novel biomarkers and investigating nutrient-genome interactions (Vanhaverbeke et al., 2022). Dietary stimulation, such as specific nutrient excess or deficiency, is a large factor in epigenetics, the study of environmental factors that influence gene expression and induce both disease prevention and development. Dietary bioactive compounds, more often referred to as vitamins, minerals or phytochemicals (i.e., polyphenols, carotenoids), as well as dietary fatty acids such as omega-3 and omega-6, reportedly alter microRNA (miRNA) levels, affecting chromatin structure, DNA methylation, the modification of histones and the activation of transcription factors and altered expression profile of genes at the system level (Mierziak et al., 2021). Whilst both associated with gene expression, mRNA and miRNA possess different roles/mechanisms: mRNA transports genetic information copied from DNA, in order to build proteins during translation (protein synthesis), whereas miRNA regulates gene expression (post-transcription), primarily through targeted mRNA binding, leading to degradation, or silencing (Cheng et al., 2025).

Saturated fatty acids are widely believed to possess adverse effects on metabolic health. Post-translation (protein synthesis), long-chain fatty acids attach to proteins in a process named palmitoylation. Primarily occurring with palmitic acid, these fatty acids attach to cysteine residues within proteins, aiding in protein function, regulation and transportation (localisation) (Dennis & Heather, 2023). Palmitoylation dysregulation, or excess palmitic acid (hyper-palmitoylation), can induce adverse effects on protein function, stability and localisation, leading to inflammation, insulin resistance, neurological disorders, diabetes, and some cancers (Chen et al., 2025; Qian et al., 2025). However, the cause-effect relationship between dietary palmitic acid and metabolic health has not been extensively evaluated. Principally, key genes associated with inflammation (*TNF-α, NFκB1, CCL2*, *CCL3*, *IL6*, *PTGS2*, *IRAK1*, *IRF1*, *MMP2*, *MMP3*, *TLR2*, *TLR8),* lipid metabolism *(ABCA1, ADIPIQ, APOE, FAM13A, LCN2)*, insulin synthesis *(INS, RETN)*, and neuronal function *(UNC13C, PICALM)* require more in-depth analysis. While saturated fat consumption is at a recommended level of <11% of total daily energy intake (Public Health England, 2019), or ~24-31g per day (ages 19-64), depending on biological sex (Office for Health Improvement and Disparities, 2024) - there is no specific recommended dietary intake/allowance (RDI/RDA) for palmitic acid, a major saturated fat

constituent of Westernised diets. This study is follow-up research following identification of individuals with different levels of dietary palmitic acid consumption, with the research question: In participants with low, medium and high dietary intake of palmitic acid, is the gene expression profile of key biomarkers linked to metabolic health contrarily affected? Thus, the main aims and objectives of this research are:

- To determine which key biomarkers between the three dietary groups are predominantly affected, and to what extent.
- To provide insight on prospective parameters of dietary palmitic acid consumption for intake recommendations/guidelines.

It is hypothesised that metabolic health status - including risks of contribution to chronic disease development - will have an association with dietary palmitic acid levels.

5.3 Methodology

5.3.1 Ethics

This study was conducted in accordance with the Declaration of Helsinki, and the ethics was reviewed and approved by the institutional ethics committee (Reference: HEALTH 01004). All subjects read and understood the study information (Appendix 5.1), then provided signed informed consent (Appendix 5.2) for inclusion before they participated in the study. Safety (COSHH), risk assessment, and data protection forms are available in Appendices 5.3, 5.4, 5.5.

5.3.2 Participants

Participants were recruited following two primary methods, 1: invitations sent to those who participated in the intake study (Chapter 3) and expressed interest to participate in this follow-up study, 2: posters, flyers and social media posts (Appendix 5.6) aiming to recruit participants from the local area, due to the logistics of the participants' vicinity of/opportunity to participate in the blood and faecal sample process. Hence, the participants included in this study came from two sources: a) those who participated in the survey study (Chapter 3) and their dietary palmitic acid consumption was assessed, and b) volunteers who couldn’t participate in the survey study, however based on their own self-perceived palmitic acid intake based on factors such as dietary habit and/or lifestyle, they deemed suitable to be included in this study. Participants were required to meet the following inclusion criteria:

i. No pre-existing disease conditions and should be in good health.
ii. Must be aged 18+, any gender, any ethnicity.
iii. Be on the respective diet for >3 months.
iv. Must not be taking any medication.
v. Able to clearly understand/communicate (reading and writing) in English.
vi. Must read and fully understood the procedure(s) and all information provided on the participant information sheet (PIS).
vii. Must have read, fully understood and signed the participant consent form.

5.3.3 Anthropometry

Participants were instructed to visit the Nutrition Suite (Darwin building, UCLan, Preston) on one occasion, where anthropometric measurements were taken. Participant height was measured using a seca 213 Portable Height Measure (Seca; Hamburg, Germany). Participant weight was measured using an Omron BF511 weighing scale (Omron; Kyoto, Japan). Participant hip/waist circumference was measured using a standard measuring tape. Body composition parameters including skeletal muscle mass, fat free mass, fat mass and water content were measured using a seca mBCA 515 Body Composition Analyzer (Seca; Hamburg, Germany).

5.3.4 Diet diary and assessment of palmitic acid intake

All participants completed a 1-week food diary that records all foods and drink consumed during the week. Dietary intake was analysed using a nutrition analysis software Nutritics Research Edition v5.94 (nutritics.com, 2023). Individual's daily intake of energy (kcal), carbohydrates (g), fibre, sugars, free sugars, total fats, saturated fats, trans-fatty acids, monounsaturated fats, polyunsaturated fats, total protein and vitamins (A, D, E and K) were recorded. To determine the palmitic acid within food(s) consumed by each participant, two resources were used (alongside provided food diaries), 1: The McCance & Widdowson's the Composition of Foods and Integrated Dataset (CoFID) 2021). 2: Fatty acid composition data developed for selected food groups (70 food items), reported in Chapter 4. Both of these resources together provided sufficient palmitic acid content data to calculate individual daily palmitic acid intake. All data was calculated using, and compiled within, a Microsoft Excel datasheet.

5.3.5 Dietary palmitic acid intake categories

A total of 18 participants included in this study. Participants were categorised into one of three groups based on the 33.33rd percentile range of their daily total palmitic acid intake. The low palmitic acid intake group (n=6) had <7g/day (2.75- 6.17g/day), the medium PA intake group (n=6) had 7-10g/day (7.49-9.47g/day) and the high PA intake group (n=6) had >10g/day (10.32-22.20g/day) intake of palmitic acid.

5.3.6 Statistical analysis of dietary data

Statistical analysis was performed using SPSS v29.0.2.0 (IBM, SPSS). Basal demographic frequency data was calculated via two-way cross tabulation analysis. A one-way analysis of variance was then performed to determine mean intake per each diet, external of the three intake groups. Following this, the three intake groups: 'Low' (*n* =6), 'Medium' (*n* =6) and 'High' (*n* =6), were used in the following statistical analyses: two-way cross tabulation analysis was performed on the demographic data, as well as palmitic acid intake per dietary habit, to obtain mean values. A one-way ANOVA was conducted to examine the effect of dietary habit, anthropometric and bioimpedance analysis measurements, as well as on dietary intake of nutrients on palmitic acid intake. Post-hoc analyses used Tukey's HSD Test for multiple comparisons. Correspondingly with Chapter 3 of this thesis, formal tests of normality were not deemed necessary.

5.3.7 Collection of Blood samples and RNA extraction

From each participant, one intravenous blood sample was collected by a trained phlebotomist. Immediately after collection, the blood samples were processed for RNA extraction as described in Bahar et al., (in press). Briefly, the steps are 1: The intravenous blood sample (8 ml) was collected by the phlebotomist into a Becton Dickinson (BD) Vacutainer (8.5ml, ACD-A, yellow) blood collection tube. 2: Using a total RNA isolation (TRI)-reagent, total ribonucleic acid (RNA) was extracted from the peripheral blood mononuclear cells (PBMCs), following manufacturer guidelines. 3: To eliminate genomic DNA, total RNA was then subjected to DNase I (Sigma-Aldrich Corp.) treatment. 4: GenElute mammalian total RNA miniprep kit (Sigma-Aldrich Corp.) was used to perform RNA column purification. 5: Finally, total RNA was dissolved in 50µL 0.1% diethylpyrocarbonate (DEPC)-treated water, to be then stored at -80 °C. Quantification and quality of total RNA were assessed within a NanoDrop-ND1000 Spectrophotometer (Thermo Fisher Scientific Inc. MA, USA). An equal quantity

(1µg) of RNA from each of the six participants representing each of the three palmitic acid intake groups (total of 18 participants) was pooled to constitute a single RNA pool per group - resulting in 3 RNA pools; low, medium and high palmitic acid intake.

5.3.8 Gene expression profiling

As described in Bahar et al., (in press): total RNA (1µg) from pooled RNA was used to evaluate quantification of gene expression, cDNA synthesis, performed using the RevertAid H Minus First Strand cDNA Synthesis Kit (Fermentas GmbH, St. Leon-Rot, Germany) following manufacturer guidelines. Using a customized quantitative PCR (qPCR) array (Applied Biosystems) on a 7300 Real Time PCR System, quantitative expression(s) of a panel of 92 genes were evaluated. Using this pre-customized array in a 96-well format enabled the screening of 192 genes relating to energy homeostasis, immune response, cancer, oxidative stress, neuronal signaling and other multiple disease pathogenesis pathways (Bahar et al., 2016). Quantitative polymerase chain reaction (PCR) was performed in a total volume of 10µl containing 0.5µl cDNA (post-1:5 dilution), 4.5µl water and 5µl SYBR Green qPCR master mix (Applied Biosystems). Cycling conditions were as follows: 94°C for 30s followed by 60 °C for 1 min, for 40 cycles. The mRNA abundances were expressed in cycle threshold (Ct) values, the number of PCR cycles in which the PCR product crosses a threshold. In this experiment, a CT value of 35 was considered as the cut-off limit. Normalization of the expression of the target genes included in the PCR array was performed based on three reference genes (glyceraldehyde phosphate dehydrogenase (*GAPDH*) and beta actin (*ACTB*) and following $2^{-\Delta\Delta Ct}$ method (Δ = Delta). ΔCt value was calculated as the difference of target gene Ct, minus the geometric average of the relative quantities of the three reference genes. Lastly, fold change was calculated as: $2^{(-\text{ average }\Delta Ct\text{ target gene})}/2^{(-\text{ average }\Delta Ct\text{ reference gene})}$.

5.4 Results

5.4.1 Participant characteristics

The characteristics of participants included in this study are presented as total count (n; %) in Table 5.1. The majority (72%) of participants were aged at or above thirty-five years old, with participant gender equally (50:50) divided between males and females. The dominant dietary habit amongst participants was omnivore (50%), with the plant-based/vegan dietary habit being second (16.7%), alongside flexitarian (11.1%), vegetarian (5.67%), and other (16.7%). A two-

way cross-tabulation analysis was performed on each of the participant characteristic variables within the three intake groups, presented in Table 5.2.

Table 5.1. Participant baseline demographic/characteristics parameters.

Variables	n (%)	*P-value*
Age(y)		**0.002**
18-25	1 (5.6)	
25-34	4 (22.2)	
>35	13 (72.2)	
Gender	**n (%)**	**1.000**
Male	9 (50)	
Female	9 (50)	
Dietary Habit	**n (%)**	**0.028**
Omnivore	9 (50.0)	
Flexitarian	2 (11.1)	
Vegetarian	1 (5.6)	
Plant-based/vegan	3 (16.7)	
Other	3 (16.7)	
Actual Values (Expected Values).		

Note: P-values were determined by chi-square (χ^2) tests, measuring the probability that observed results (differences from expected results) occurred by chance.

Table 5.2. Participant baseline demographic/characteristics per PA intake group.

Parameters	Low	Medium	High	*P-value*
Demographic				
Gender				**0.135**
Male	2 (3)	2 (3)	5 (3)	
Female	4 (3)	4 (3)	1 (3)	
Age (y)				**0.539**
18-25	1 (0.3)	0 (0.3)	0 (0.3)	
25-34	2 (1.13)	1 (1.13)	1 (1.13)	
>35	3 (4.3)	5 (4.3)	5 (4.3)	

	Actual Values (Expected Values).	

5.4.2 Palmitic acid content

Mean palmitic acid intake was calculated for the three groups, presented in Table 5.3. There was a significant difference in palmitic acid intake between these groups (P <0.001). A significant difference (P <0.001) between dietary habit and palmitic acid intake was also observed. No significant differences were observed concerning palmitic acid intake between the five dietary habits. A significant difference between mean palmitic acid intake and dietary habit was also not observed when condensed into two groups (1: diets containing meat and/or dairy, 2: plant-based/vegan diets) (Table 5.4).

Table 5.3. Participant palmitic acid intake (g) per dietary habit per PA intake group.

Parameters	Low	Medium	High	P-value
Daily intake				
Palmitic acid (16:0)	3.87 ± 0.52	8.65 ± 0.33	13.97 ± 1.71	**<0.001**
Dietary parameter(s)				
PA intake by dietary habit				**<0.001**
Omnivore	3.41 ± 0.54	8.65 ± 0.76	12.21 ± 0.73	
Flexitarian		8.77	22.20	
Vegetarian		7.49		
Plant-based/vegan	4.33 ± 0.93			
Other		9.18 ± 0.29	12.80	
Mean ± Standard Error of the Mean.				

Note: P-values were determined by two-way ANOVA, measuring the probability that observed variations (between group means) occurred by chance.

Table 5.4. Participant palmitic acid intake (g) per dietary habit.

PA intake by dietary habit	*n*	Mean ± SE	*P-value*
Omnivore	9	8.49 ± 1.41	
Flexitarian	2	15.49 ± 6.72	
Vegetarian	1	7.49	**0.139**
Plant-based/vegan	3	4.33 ± 0.93	
Other	3	10.38 ± 1.15	

PA intake by dietary habit (cont.)	*n*	Mean (SE)	*P-value*
Diet(s) containing meat and/or dairy	15	9.73 ± 1.25	**0.080**
Plant-based/vegan	3	4.33 ± 0.93	
SE = Standard Error of the Mean.			
Note: P-values were determined by ANOVA, measuring the probability that observed variations (between group means) occurred by chance.			

5.4.3 Anthropometry

A significant difference in 'Height' between at least two groups ($F_{(2, 15)} = 3.883$, $P = 0.044$, $\eta^2 = 0.341$, was observed. Post-hoc, Tukey's HSD Test for multiple comparisons found that the mean value of 'Height' was not significantly different between 'Low' and 'Medium' ($P = 0.993$), 'Low' and 'High' ($P = 0.064$), or 'Medium' and 'High' ($P = 0.078$) palmitic acid intake groups. Therefore, while the one-way ANOVA indicated a difference between overall groups, post-hoc testing indicated no significant differences between paired groups. No other significant differences were observed between any of the variables within the three groups (Table 5.5).

Table 5.5. Participant bioimpedance analysis parameters per PA intake group.

Parameters	Low	Medium	High	*P-value*
Anthropometry				
Weight (kg)	70.23 ± 7.25	71.19 ± 6.74	75.83 ± 4.69	**0.802**
Height (m)	1.67 ± 0.03	1.67 ± 0.02	1.74 ± 0.02	**0.044**
BMI (kg/m²)	25.05 ± 1.89	25.42 ± 2.16	24.94 ± 1.26	**0.981**
Waist circumference (m)	0.82 ± 0.04	0.80 ± 0.05	0.87 ± 0.04	**0.568**
Water (l)				
Total body water (TBW)	34.85 ± 4.30	33.68 ± 2.50	40.05 ± 2.30	**0.344**
Extracellular water (ECW)	14.95 ± 1.54	15.07 ± 0.91	16.97 ± 0.63	**0.404**
ECW/TBW %	43.33 ± 1.85	44.94 ± 0.96	42.40 ± 1.05	**0.417**
Visceral Adipose Tissue (l)				
Visceral adipose tissue (VAT)	1.33 ± 0.25	1.47 ± 0.40	1.98 ± 0.48	**0.480**

Skeletal Muscle Mass (kg)				
Whole body (Total)	21.99 ± 3.45	20.71 ± 2.13	26.03 ± 2.02	**0.349**
Left arm	1.26 ± 0.26	1.23 ± 0.26	26.03 ± 2.02	**0.369**
Torso	9.62 ± 1.72	8.70 ± 1.03	11.77 ± 1.18	**0.283**
Right arm	1.29 ± 0.26	1.28 ± 0.14	1.65 ± 0.15	**0.329**
Left leg	4.83 ± 0.59	4.70 ± 0.42	5.50 ± 0.32	**0.439**
Right leg	4.99 ± 0.65	4.78 ± 0.41	5.52 ± 0.27	**0.526**
Fat-Free Mass				
Fat-free mass (FFM) (kg)	47.57 ± 5.79	46.05 ± 3.50	54.91 ± 3.12	**0.325**
Fat-free mass index (FFMI) (kg/m²)	16.90 ± 1.46	16.45 ± 1.01	18.05 ± 0.72	**0.585**
Fat Mass				
Fat mass (FM) (kg)	22.67 ± 3.09	25.14 ± 4.66	20.93 ± 2.465	**0.703**
Fat mass index (FMI) (kg/m²)	8.15 ± 1.12	8.96 ± 1.59	6.92 ± 0.80	**0.503**
Energy				
Total daily energy expenditure (TDEE)	2698.67 ± 282.49	2607.50 ± 114.74	2891.83 ± 251.99	**0.675**
Physical activity level (PAL)	1.77 ± 0.08	1.77 ± 0.03	1.73 ± 0.08	**0.927**
Resting daily energy expenditure (kcal)	1523.83 ± 123.19	1481.17 ± 80.98	1659.33 ± 90.73	**0.440**
Mean ± Standard Error of the Mean.				

Note: P-values were determined by ANOVA, measuring the probability that observed variations (between group means) occurred by chance.

5.4.4 Dietary intake of nutrients

The dietary intake of all assessed macronutrients and micronutrients are presented in Table 5.6. Individuals with 'Low' palmitic acid content consumed less calories per day (P <0.001), when compared with the 'High' intake group. The 'Low' group also consumed fewer total carbohydrates (P = 0.002), total fats (P <0.001), saturated fat (P = 0.003), and total protein (P <0.001), compared to the 'High' intake group, with borderline significance observed for carbohydrates between 'Medium' and 'High' (P = 0.050).

Table 5.6. Participant dietary intake parameters per PA intake group.

Parameters	Low	Medium	High	*P-value*
Energy (kcal)				
Daily intake (kcal)	1205.34 ± 167.24	1690.07 ± 203.55	2379.95 ± 110.73	**<0.001**
Target intake (kcal)	2487.93 ± 225.43	2430.51 ± 120.86	2686.98 ± 198.73	**0.606**
Carbohydrates (g)				
Total	139.32 ± 17.06	192.86 ± 33.86	279.40 ± 15.20	**0.003**
Fibre	19.96 ± 2.96	24.97 ± 4.63	26.53 ± 3.74	**0.467**
Sugars	41.26 ± 7.56	68.75 ± 21.62	87.80 ± 8.64	**0.097**
Free sugars	10.08 ± 4.83	0.88 ± 2.29	18.17 ± 3.64	**0.129**
Calories	557.67 ± 68.22	771.45 ± 135.44	1117.60 ± 60.80	**0.003**
Macronutrient (%)	46.92 ± 1.56	44.52 ± 2.26	46.89 ± 0.83	**0.515**
Fats (g)				
Total	51.05 ± 8.00	70.10 ± 6.66	91.73 ± 4.52	**0.002**
Saturated	14.02 ± 2.44	24.24 ± 2.38	35.89 ± 5.76	**0.004**
Saturated (% of Total)	28.78 ± 3.22	35.22 ± 2.83	38.08 ± 4.90	**0.234**
Saturated (kcal)	126.13 ± 21.99	218.14 ± 21.47	322.97 ± 51.83	**0.004**
Trans-fats	0.52 ± 0.16	0.73 ± 0.23	0.42 ± 0.18	**0.508**
Monounsaturated	11.24 ± 3.06	20.75 ± 3.20	12.97 ± 3.20	**0.080**
Polyunsaturated	6.17 ± 1.63	8.90 ± 2.27	6.47 ± 1.15	**0.495**
Calories	459.49 ± 71.99	630.91 ± 59.89	825.52 ± 40.72	**0.002**
Macronutrient (%)	37.67 ± 1.21	38.02 ± 2.14	34.73 ± 1.06	**0.284**
Protein (g)				
Total	44.03 ± 7.27	62.87 ± 7.81	98.01 ± 8.71	**<0.001**
Calories	176.14 ± 29.10	251.49 ± 31.25	392.03 ± 34.85	**<0.001**

Macronutrient (%)	14.50 ± 3.27	15.00 ± 3.23	16.83 ± 4.17	**0.509**
Vitamin(s)				
Vitamin A (ug)	587.96 ± 209.94	636.14 ± 214.74	431.53 ± 158.29	**0.747**
Vitamin D (ug)	1.09 ± 0.40	2.10 ± 0.55	2.00 ± 0.38	**0.251**
Vitamin E (mg)	4.79 ± 0.73	6.24 ± 0.99	5.17 ± 0.88	**0.497**
Vitamin K (ug)	42.74 ± 16.79	56.59 ± 27.52	35.21 ± 16.26	**0.767**
Mean ± Standard Error of the Mean.				

5.5.5 Gene expression

In this study, a total of 184 gene expression markers (mRNA) and 8 internal references were screened and the mRNA of 155 genes were detected in the three groups of palmitic acid intake. A total of 57 differentially expressed genes (mRNA abundances exceeded the cut off range of ±2 folds) were identified and used for further evaluation: energy homeostasis (11), immune response (21), cancer (9), oxidative stress (2), neuronal signalling (4), multiple disease pathogenesis (10). Gene abbreviations and their full terms are presented in Table 5.7. The full list of differentially expressed genes (fold changes) across the three palmitic acid intake groups is presented in Table 5.8.

Table 5.7. Gene identifiers (abbreviations) and their respective gene name.

Identifier	Gene name
ABCA1	Adenosine triphosphate (ATP) binding cassette subfamily A member 1
ADIPOQ	Adiponectin (complement component 1q and collagen domain containing)
ADRA1B	Adrenoceptor alpha 1B
APOE	Apolipoprotein E
BCAM	Basal cell adhesion molecule
BCL2	B-cell lymphoma 2 apoptosis regulator
CCL2	C-C motif chemokine ligand 2
CCL3	C-C motif chemokine ligand 3
CCR1	C-C chemokine receptor type 1
COBL	Cordon-bleu Wiskott-Aldrich Homology (WH2) repeat protein
CREB1	Cyclic adenosine monophosphate (cAMP) responsive element binding protein 1
CSF2	Colony stimulating factor 2
CSPG5	Chondroitin sulphate proteoglycan 5
CYBB	Cytochrome b-245 beta chain
DPP4	Dipeptidyl peptidase 4
EGF	Epidermal growth factor
EGR2	Early growth response protein 2
FAM13A	Family with sequence similarity 13 member A
FRMD4A	Four.1 protein, ezrin, radixin, moesin (FERM) domain containing 4A
GEM	Guanosine triphosphate (GTP) binding protein overexpressed in skeletal muscle
GSK3β	Glycogen synthase kinase 3 beta
HRH3	Histamine receptor 3
IL10	Interleukin 10
IL17A	Interleukin 17A

	IL6	Interleukin 6	
	INS	Insulin	
	IRAK1	Interleukin 1 associated receptor kinase 1	
	IRF1	Interferon regulatory factor 1	
	IRS1	Insulin receptor substrate 1	
	JUN	Jun proto-oncogene	
	LCN2	Lipocalin 2	
	LEPR	Leptin receptor	
	MMP2	Matrix metalopeptidase 2	
	MMP3	Matrix metalopeptidase 3	
	MS4A6E	Membrane spanning 4-domains A6E	
	MUC5AC	Mucin 5AC, oligomeric mucus/gel-forming	
	NFκB1	Nuclear factor kappa B subunit 1	
	PCDH11X	Protocadherin 11 X-linked	
	PCK1	Phosphoenolpyruvate carboxykinase 1	
	PICALM	Phosphatidylinositol binding clathrin assembly protein	
	PIK3R1	Phosphoinositide-3-kinase regulatory subunit 1	
	PLAT	Plasminogen activator, tissue type	
	PPP2CA	Protein phosphatase 2 catalytic subunit alpha	
	PRKACG	Protein kinase cAMP-activated catalytic subunit gamma	
	PTGER1	Prostaglandin E receptor 1	
	PTGS2	Prostaglandin endoperoxidase 2	
	RETN	Resistin	
	SLC10A2	Solute carrier family 10-member 2	
	SOD1	Superoxide dismutase 1	
	SOX2	Sex-determining region Y (SRY)-box transcription factor 2	
	TERT	Telomerase reverse transcriptase	
	TLR2	Toll-like receptor 2	

	TLR8	Toll-like receptor 8	
	TNF-α	Tumour necrosis factor-alpha	
	TREM2	Triggering receptor expressed on myeloid cells 2	
	UNC13C	Unc-13 homolog C	
	VCAM1	Vascular cell adhesion molecule 1	

Table 5.8. Differential expression of genes (by fold change) in cardiometabolic-related pathways between the three palmitic acid intake groups.

Gene	Mechanism	Pathway	Role	[1]Low/High	[1]Low/Medium	[1]Medium/High
ABCA1	Lipid metabolism, transportation, β-cell function	Energy homeostasis	Promoter	+63.16	-1.36	+86.14
ADIPOQ	Glucose, fatty acid metabolism	Energy homeostasis	Promoter	+2.38	+0.57	+4.14
APOE	Lipid metabolism	Energy homeostasis	Promoter	-8.03	-15.81	+1.97
DPP4	Glucose and insulin metabolism, homeostasis	Energy homeostasis	Regulator	+6.54	+0.66	+9.97
FAM13A	Glycaemic control, lipid metabolism	Energy homeostasis	Inhibitor	+2.47	+1.20	+2.05
INS	Insulin synthesis	Energy homeostasis	Regulator	+2.92	-2.11	+6.16
LEPR	Leptin signalling	Energy homeostasis	Promoter	-0.69	+0.70	+2.05
PCK1	Control point of gluconeogenesis	Energy homeostasis	Promoter	-0.96	+2.74	-2.63
PIK3R1	Gene expression, cellular metabolism	Energy homeostasis	Regulator	+2.88	-1.95	+5.61
RETN	Impair glucose homeostasis	Energy homeostasis	Promoter	+1.93	-2.65	+5.11
SLC10A2	Cholesterol metabolism/homeostasis	Energy homeostasis	Promoter	+2.64	-1.82	+4.81
CCL2	Pro-inflammatory chemokine, leukocyte migration	Immune response	Promoter	-0.80	-5.91	+7.37
CCL3	Pro-inflammatory chemokine, cellular migration	Immune response	Promoter	-1.39	-4.47	+3.21
CCR1	Chemokine receptor, recruitment of immune cells	Immune response	Promoter	-5.35	-3.06	-1.75

CREB1	Transcription factor	Immune response	Regulator	-1.07	+7.11	-7.63
EGR2	Transcription factor, Th17 cell pathogenicity	Immune response	Regulator	-1.70	-8.58	+5.05
HRH3	Histamine release	Immune response	Regulator	-0.97	-7.28	+7.53
IL10	Anti-inflammatory cytokine	Immune response	Inhibitor	+3.06	+1.96	-0.64
IL17A	Pro-inflammatory cytokine	Immune response	Promoter	-0.70	-6.45	+9.28
IL6	Pro-inflammatory cytokine	Immune response	Promoter	-1.54	+1.74	-2.68
IRAK1	Protein kinase, inflammatory gene expression	Immune response	Regulator	-0.81	+6.66	-5.41
IRF1	Regulation of the anti-viral immune response	Immune response	Regulator	-0.81	+4.76	-3.85
MMP2	Extracellular matrix component degradation	Immune response	Regulator	-1.39	-7.00	+5.03
MMP3	Extracellular matrix component degradation	Immune response	Promoter	-1.39	-7.89	+5.68
MS4A6E	Stem cell proliferation, cell-to-cell interaction	Immune response	Regulator	+7.63	-1.24	+9.45
NFκB1	Transcription factor, immune response	Immune response	Regulator	-0.88	+2.37	-2.10
PTGS2	Enzyme, prostaglandin synthesis	Immune response	Promoter	-1.92	+0.71	-1.36
TLR2	Mediates pro- and anti-inflammatory processes	Immune response	Regulator	-0.91	+6.55	-5.95
TLR8	Response to viral infection	Immune response	Promoter	-10.60	-6.42	-1.65
TNF-α	Pro-inflammatory cytokine	Immune response	Promoter	-1.31	-10.19	+7.78
TREM2	Phagocytosis & cytokine production	Immune response	Promoter	+1.41	-1.42	+2.00
VCAM1	Leukocyte adhesion, trans-endothelial migration	Immune response	Regulator	-1.31	-2.40	+1.83
BCAM	Plasma membrane glycoprotein	Cancer	Promoter	-1.39	-7.04	+5.06
BCL2	Anti-apoptosis	Cancer	Inhibitor	-1.14	+34.45	-39.16
CSF2	Intracellular signalling, myeloid cell proliferation	Cancer	Promoter	+1.64	+0.63	+2.59
EGF	Growth factor, cell proliferation	Cancer	Inhibitor	+6.08	+0.63	+9.72
GEM	Hepatocellular carcinoma	Cancer	Regulator	-4.53	+1.70	-7.71
JUN	Transcription factor	Cancer	Promoter	-5.30	-10.42	+1.97
LCN2	Lipid metabolism, anti-metastasis	Cancer	Regulator	-1.18	+2.95	-3.48
SOX2	Gene expression, stem cell maintenance	Cancer	Regulator	-6.20	-4.46	+0.72

TERT	Telomerase function	Cancer	Regulator	-1.61	-4.46	+2.76
CYBB	Phagocytosis	Oxidative stress	Promoter	-1.89	-2.04	+1.08
SOD1	Anti-oxidant, cellular protection from ROS toxicity	Oxidative stress	Inhibitor	+2.44	+0.72	+3.36
CSPG5	Neuronal growth, function and differentiation	Neuronal signalling	Promoter	+1.01	-3.94	+3.98
PCDH11X	Central nervous system cell-to-cell communication	Neuronal signalling	Promoter	-1.14	-3.95	+3.47
PICALM	Neuronal function, endocytosis	Neuronal signalling	Promoter	+18.92	+17.68	+1.07
UNC13C	Synaptic transmission, exocytosis	Neuronal signalling	Promoter	-11.44	-13.20	+1.15
ADRA1B	Activates cell growth and proliferation	Multiple disease pathogenesis	Promoter	-1.02	-2.78	+2.73
COBL	Intestinal homeostasis, neuronal regulation	Multiple disease pathogenesis	Regulator	-6.59	-13.21	+2.01
FRMD4A	Epithelial cell polarity, integrity	Multiple disease pathogenesis	Regulator	-4.54	-9.07	+2.00
GSK3β	Glucose metabolism, phosphorylation	Multiple disease pathogenesis	Regulator	-2.13	-1.90	-1.13
IRS1	Adaptor protein, insulin signalling pathways	Multiple disease pathogenesis	Promoter	-0.70	-2.48	+3.56
MUC5AC	Glycoprotein, epithelium protection	Multiple disease pathogenesis	Promoter	-1.60	-4.60	+2.88
PLAT	Blood clotting, fibrinolysis	Multiple disease pathogenesis	Inhibitor	-1.49	-6.53	+4.40
PPP2CA	Dephosphorylation of proteins	Multiple disease pathogenesis	Inhibitor	+1.07	-23.35	+25.05
PRKACG	Intracellular signalling	Multiple disease pathogenesis	Promoter	-2.36	-4.12	+1.75
PTGER1	G-protein coupled receptor, blood pressure	Multiple disease pathogenesis	Promoter	-1.39	-5.71	+4.10
	[1]Fold change values.					

5.5.6 Comparison of gene expression profiles in low vs high palmitic acid intake

The ‘Low’ palmitic acid intake group had a higher mRNA abundance of 8 genes concerning energy homeostasis pathway(s), including: *ABCA1*, *ADIPOQ*, *DPP4*, *FAM13A*, *INS*, *PIK3R1*, *RETN*, and *SLC10A2*. The mRNA of 3 genes had lower abundance: *APOE*, *LEPR*, *PCK1*. For the immune response pathway(s), the ‘Low’ palmitic acid intake group had a lower mRNA abundance of 19 pro-inflammatory genes, including: *CCL2*, *CCL3*, *CCR1*, *CREB1*, *EGR2*, *HRH3*, *IL6*, *IRAK1*, *IRF1*, *MMP2*, *MMP3*, *NFκB1*, *PTGS2*, *TLR2*, *TLR8*, *TNF-α,* and *VCAM1*. The 3 genes had higher mRNA abundance include: *IL10*, *MS4A6E*, and *TREM2*.

For the markers involved in cancer pathway(s), the ‘Low’ palmitic acid intake group had a lower mRNA abundance of 7 genes, including: *BCAM*, *BCL2*, *GEM*, *JUN*, *LCN2*, *SOX2*, *TERT*. Among the genes that has higher mRNA abundance in the low palmitic acid intake group include *CSF2* and *EGF*. For the oxidative stress pathway(s), the ‘Low’ palmitic acid intake group, had a higher mRNA abundance of *SOD1*, while lower abundance of *CYBB*. Concerning the neuronal signalling pathway(s), the ‘Low’ palmitic acid intake group had a higher mRNA abundance of *CSPG5* and *PICALM* genes while lower abundance of *PCDH11X* and *UNC13C* mRNA. In addition, the mRNA of genes involved in multiple disease pathogenesis including *ADRA1B*, *COBL*, *FRMD4A*, *GSK3B*, *IRS1*, *MUC5AC*, *PLAT*, *PRKACG*, and *PTGER1* were lower in the ‘Low’ palmitic acid intake group. The mRNA of only one gene concerning multiple disease pathogenesis pathway(s) was in higher abundance: *PPP2CA.*

5.5.7 Comparison of gene expression profiles in low vs medium palmitic acid intake

The ‘Low’ palmitic acid intake group had a lower mRNA abundance of 6 genes concerning energy homeostasis pathway(s), including: *ABCA1*, *APOE*, *INS*, *PIK3R1*, *RETN*, and *SLC10A2*. The mRNA of 5 genes had lower abundance: *ADIPOQ*, *DPP4*, *FAM13A*, *LEPR*, and *PCK1*. For the immune response pathway(s), the ‘Low’ palmitic acid intake group had a lower mRNA abundance of 13 pro-inflammatory genes, including: *CCL2*, *CCL3*, *CCR1*, *EGR2*, *HRH3*, *IL17A*, *MMP2*, *MMP3*, *MS4A6E*, *TLR8*, *TNF-α*, *TREM2*, and *VCAM1*. The mRNA of 8 genes had higher abundance: *CREB1*, *IL10*, *IL6*, *IRAK1*, *IRF1*, *NFκB1*, *PTGS2*, and *TLR2*.

For the markers involved in cancer pathway(s), the 'Low' palmitic acid intake group had a higher mRNA abundance of 5 genes, including: *BCL2*, *CSF2*, *EGF*, *GEM*, *LCN2*. The mRNA of 4 genes had lower abundance: *BCAM*, *JUN*, *SOX2*, and *TERT*. For the oxidative stress pathway(s), the 'Low' palmitic acid intake group, had a higher mRNA abundance of *SOD1*, while lower abundance of *CYBB*. Concerning neuronal signalling pathway(s), the 'Low' palmitic acid intake group had a lower mRNA abundance of 3 genes, including: *CSPG5*, *PCDH11X*, and *UNC13C*. The mRNA of 1 gene had higher abundance: *PICALM*. In addition, the mRNA of genes involved in multiple disease pathogenesis including *ADRA1B*, *COBL*, *FRMD4A*, *GSK3B*, *IRS1*, *MUC5AC*, *PLAT*, *PPP2CA*, *PRKACG*, and *PTGER1* were lower in the 'Low' palmitic acid group. No genes involved in multiple disease pathogenesis pathway(s) were observed in higher abundance.

5.5.8 Comparison of gene expression profiles in medium vs high palmitic acid intake

The 'Medium' palmitic acid intake group had a lower mRNA abundance of 10 genes concerning energy homeostasis pathway(s), including: *ABCA1*, *ADIPOQ*, *APOE*, *DPP4*, *FAM13A*, *INS*, *LEPR*, *PIK3R1*, *RETN*, and *SLC10A2*. The mRNA of 1 gene concerning energy homeostasis pathway(s) had lower mRNA abundance: *PCK1*. For the immune response pathway(s), the 'Medium' palmitic acid intake group had a higher mRNA abundance of 11 genes, including: *CCL2*, *CCL3*, *EGR2*, *HRH3*, *IL17A*, *MMP2*, *MMP3*, *MS4A6E*, *TNF-α*, *TREM2*, and *VCAM1*. The mRNA abundance of 10 genes had lower abundance: *CCR1*, *CREB1*, *IL10*, *IL6*, *IRAK1*, *IRF1*, *NFκB1*, *PTGS2*, *TLR2*, and *TLR8*.

For the markers involved in cancer pathway(s), the 'Medium' palmitic acid intake group (7-10g/day), compared to the 'High' palmitic acid intake group (>10g/day), had a higher mRNA abundance of 6 genes, including: *BCAM*, *CSF2*, *EGF*, *JUN*, *SOX2*, and *TERT*. The mRNA of 3 genes had lower mRNA abundance: *BCL2*, *GEM*, and *LCN2*. For the oxidative stress pathway(s), the 'Medium' palmitic acid intake group had a higher mRNA abundance of 2 genes, including: *CYBB* and *SOD1*. No genes concerning oxidative stress pathway(s) were observed in lower mRNA abundance. Concerning neuronal signalling pathway(s), the 'Medium' palmitic acid intake group had a higher mRNA abundance of 4 genes, including: *CSPG5*), *PCDH11X*, *PICALM*, and *UNC13C*. No genes concerning neuronal signalling pathway(s) were

observed in lower mRNA abundance. In addition, the mRNA of genes involved in multiple disease pathogenesis had a higher mRNA abundance of 9 genes, including: *ADRA1B*, *COBL*, *FRMD4A*, *IRS1*, *MUC5AC*, *PLAT*, *PPP2CA*, *PRKACG*, and *PTGER1* were lower in the 'Medium' palmitic acid group. The mRNA of 1 gene had higher abundance: *GSK3B*.

5.6 Discussion

The objectives of this study were to compare anthropometry, dietary intake and metabolic health biomarkers in 3 groups of healthy adults with different levels of palmitic acid intake ('Low' <7g/day, 'Medium' 7-10g/day, 'High' >10 g/day). Participant body mass index (BMI) was found to be moderately analogous (*25.05 ± 1.89, 25.42 ± 2.16, 24.94 ± 1.26*), whereas there were significant (P<0.001) differences in caloric intake (1205.34 ± 167.24, 1690.07 ± 203.55, 2379.95 ± 110.73). Perhaps due to under/over-reporting, although additional areas of consideration include physical activity level, or genetic-influences such as body type/composition (musculature, fat distribution), and metabolism. Blood samples from a total of 18 participants were collected and equal amount of cDNA were pooled and the gene expression profiling was performed on these pooled samples. The mRNA of 57 genes were differentially expressed and these related to energy homeostasis, immune response, cancer, oxidative stress, neuronal signalling, as well as multiple disease pathogenesis. Unless stated otherwise, all genes listed are eponymous with the proteins they encode, i.e., the Interleukin-6 (*IL-6)* gene encodes (produces) the IL-6 protein. The participants with 'Low' palmitic acid intake in comparison to 'Medium' and 'High' palmitic acid intake groups had a favourable mRNA expression profile as far as metabolic health is concerned. Interestingly, the 'High' intake group also exhibited a healthier mRNA expression profile, when compared with the 'Medium' intake group. The three intake groups showed no significant differences in anthropometry, where there were significant differences in caloric, carbohydrate, fat, as well as protein intake.

As a comparison of 'Low' and 'High' palmitic acid intake groups, several mRNA markers suggested that 'Low' palmitic acid intake may be associated with a better lipid profile and efficient energy homeostasis. For instance, the *ABCA1* gene, that codes the *ABCA1* transporter protein, is upregulated (by 63.2 folds) in the 'Low' palmitic acid intake group; reportedly linked to beneficial effects on cardiovascular health. By removal of excess cholesterol from

cells, higher level of *ABCA1* expression can contribute to maintaining cholesterol homeostasis (Chen et al., 2022). Excess cholesterol is transported, alongside phospholipids, to then combine with apolipoprotein A-I protein (ApoA-I) to form high-density lipoprotein(s) (HDL). High levels of HDL cholesterol (>2.2 mmol/L, or >40 mg/dL) are generally considered to be inversely associated with risks of cardiovascular diseases such as coronary heart disease and atherosclerosis (Rader & Hovingh, 2014; Nicholls & Nelson, 2019). *ADIPOQ*, a gene that encodes the protein adiponectin, expressed primarily within adipose tissue, also had a 2.4-fold abundance in the low palmitic acid intake group. *ADIPOQ* contributes to increased insulin sensitivity and reduced inflammation (Hivert et al., 2008). In addition, *ADIPOQ* also regulates glucose concentrations and aids in lipid breakdown and metabolism, with low levels (hypoadiponectinemia) observed in obesity, type 2 diabetes and coronary heart disease (Ouchi & Walsh, 2007). Interestingly, adiponectin can attenuate various adverse health effects associated with palmitic acid intake such as inflammation, insulin resistance and oxidative stress, via the promotion of fatty acid oxidation, the inhibition of inflammatory cytokines, as well as reduction of reactive oxygen species (Lee at al., 2019; Zhao et al., 2016). Hence, a higher abundance of *ADIPOQ* mRNA in the low palmitic acid intake group potentially suggests better insulin sensitivity and metabolism of glucose and lipids.

It was also evident that the mRNA level of *APOE* was reduced by 8 folds. The *APOE* gene has several variants: *APOE ε2*, *APOE ε3* and *APOE ε4*. *APOE ε2* is generally considered as protective against, *APOE ε3* is neutral, whereas *APOE ε4* has strong links to increased risks of, Alzheimer's disease (Liu et al., 2013; Raulin et al., 2022). *APOE ε4* impairs energy expenditure and affects glucose utilization and thereby affecting overall energy homeostasis of the body (Farmer et al., 2021). Hence, a lower mRNA abundance of *APOE* in the low palmitic acid intake group may contribute to overall energy homeostasis favourably.

Another interesting marker of growth, metabolism and survival of cells is *PIK3R1* (a gene that primarily encodes the p85-alpha, or p85α protein), whose abundance was 2.9-fold higher in the 'Low' palmitic acid intake group. Through negative regulation of the *PI3K* signalling pathway, *PIK3R1* is often considered a tumour suppressant, with reduced expression being observed in various cancers (Chakraborty et al., 2022; Gupta & Gaykalova, 2024). The mRNA abundance of *SLC10A2* was also increased in this group by 2.65 folds. The *SLC10A2* gene, which encodes for the apical sodium/bile acid transporter (ABST) protein, plays vital roles in

glucose and lipid homeostasis by regulating the bile acid reabsorption within the distal ileum (Chothe et al., 2018). Reabsorbed bile acid is recycled via the blood stream, aiding in both lipid digestion and cholesterol regulation. Deficiencies in *SLC10A2* can lead to cholesterol dysregulation, in addition to absorption inefficiency or primary bile acid malabsorption (PMAD), a common cause of chronic diarrhoea (Barkun et al., 2013). The *INS* gene (which encodes the protein pre-proinsulin, a precursor to insulin), predominantly responsible in regulation of insulin production within β-cells to aid in blood glucose and lipid metabolism, was upregulated by 2.9 folds. Mutations in, or deficiencies of *INS* leads to the development of diabetes (Liu et al., 2015; Raile et al., 2011).

'Low' palmitic acid intake may also be associated with a healthy immune response. Most notably observed by a negative fold change of mRNA biomarkers in pro-inflammatory chemokines and cytokines, compared to 'Medium' palmitic acid intake: *CCL2* (by -5.9 folds), *CCL3* (by -4.5 folds), as well as *IL17A* (by -6.5 folds) and *TNF-α* (by -10.2 folds). *CCL2* acts as a mediator of the immune response, guiding immune cells to sites of inflammation. Higher expressions of *CCL2* have been associated with the promotion of cancer (pancreatic, prostate) cell function, in addition to poor host survival (Zhang et al., 2010; Yan et al., 2007). Similarly, *CCL3* aids in the recruitment of immune cells during the inflammatory response. *CCL3* expression level positively correlates with the expression of tumour necrosis factor receptor associated factor 6 (TRAF6) and nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB), both transcription factors associated with various forms of cancer (Ma et al., 2022; Zhu et al., 2019; Inoue et al., 2007). *CCL3* can also regulate *CCR1* expression (-5.35-fold change), a gene that also directs immune cells to sites of inflammation. Elevated *CCR1* expression has been linked to tumour progression and increased metastasis (Zhu et al., 2018).

TNF-α, a pro-inflammatory cytokine, is released upon the recruitment and activation of immune cells such as macrophages, lymphocytes (T, B cells) monocytes, triggered by signalling pathways involving transcription factors such as *TLR2* and NFκB. *TNF-α* upregulation is strongly associated with various insulin resistance-affected metabolic diseases: including obesity and type 2 diabetes, as well as colorectal cancer (Alzamil et al., 2020; Swaroop et al., 2012; Al Obeed et al., 2014). *VCAM1,* downregulated by -2.4-folds in the 'Low' group compared to 'Medium', is a protein which primarily functions in leukocyte recruitment to the endothelium during the inflammatory response, is induced by *TNF-α* (Cook-Mills et al., 2011), and has been linked to numerous immunological disorders, including asthma,

rheumatoid arthritis, inflammatory bowel disease (IBD), atherosclerosis, in addition to cancer growth, spread and development (angiogenesis, metastasis) (Pickett et al., 2015; Kong et al., 2018). *IL17A,* also downregulated in the 'Low' group compared to 'Medium' by -6.5-folds, a pro-inflammatory cytokine that recruits neutrophils (immune cells) as a response to immune system stimulation (McGeachy et al., 2019), positively correlates with the induction of other pro-inflammatory cytokines, including IL-6 and TNF-α (Starnes et al., 2002), further exacerbating their inflammatory consequences. Based on the comparison of the mRNA profile of these pro-inflammatory markers, it was evident that low palmitic acid intake group compared to the medium palmitic acid intake group had a clearly favourable state of innate immune response.

A higher mRNA abundance was observed for *IL10*, by 3.1 folds, for the 'Low' palmitic acid intake group compared to 'High', which further supports this. *IL-10* is generally considered a potent anti-inflammatory cytokine and vital to the stability of immune system (Iyer & Cheng, 2012), where aberrant expression, or deficiency can lead to disruptions in homeostasis (enhanced inflammation), and the development of various autoimmune diseases including inflammatory bowel disease (IBD) (O'Garra et al., 2008).

Another important marker of inflammation; *TLR8*, had a lower mRNA abundance, by 10.6 folds, in the 'Low' palmitic acid intake group compared to 'Medium'. While *TLR2* is primarily located on the surface of cells, *TLR8* is an intra-cellular pattern recognition receptor (PRR). Both TLRs trigger the beginning steps of the innate immune (inflammatory) response; however, *TLR8* focuses on viral and bacterial single stranded RNA (ssRNA) recognition. Upregulation of *TLR8* expression has been associated with cancer (lung, pancreatic) cell growth and survival (Cherfils-Vicini et al., 2010; Grimmig et al., 2015). Lastly, *PTGS2*, which aids in prostaglandin synthesis during the inflammatory response, had a negative 1.9-fold change in the 'Low' palmitic acid intake group, when compared to 'High'. Under homeostatic conditions, *PTGS2* is not typically expressed; however, elevated expression or increased activity has been suggested to induce various neurological disorders, and forms of cancer (Kunzmann et al., 2013; Lin et al., 2020; Semenova et al., 2023; Xie et al., 2022).

There was a negative fold change of mRNA biomarkers in several genes relating to various types of cancer cell proliferation, survival, invasion and metastases, within 'Low' palmitic acid intake, compared to 'Medium'. *BCAM,* primarily responsible for cell adhesion to other cells,

or the extracellular matrix (ECM) (Sivakumar et al., 2023), had a negative fold change of -7.0. Overexpression has been associated with both ovarian and lung cancers (Burela et al., 2024). Similarly, *JUN* was expressed at a negative 10.4-fold change. *JUN* (a gene that encodes the c-JUN protein) plays a role in cellular apoptosis mediation, as well as cell proliferation and differentiation (Wisdom et al., 1999). *JUN* overexpression is typically associated with skin cancers (Cai et al., 2012; Jafri et al., 2025), suggesting a correlation between this gene and apoptosis circumvention, leading to malignant cell survival.

The *BCL2* gene was upregulated by 34.5 folds in the 'Low' palmitic acid intake group compared to the 'Medium'. *BCL2*'s primary role is the regulation of programmed cell death, via the prevention/blocking of cytochrome c protein release from mitochondria, an initiator of apoptosis (Vogler et al., 2025). High expression of *BCL2* has been associated with beneficial effects on cancer, such as increased overall survival in patients with cancer, including colorectal cancer (Huang et al., 2017; Qian et al., 2022). Also, in the 'Low' palmitic acid intake group, compared to 'Medium'; *TERT* had a negative 4.5-fold change. *TERT* aids in chromosomal protection and stability, via the regulation of telomeres (protective structures based at the ends of chromosomes) function (Yuan & Xu et al., 2019). Expression of *TERT* plays a role in cancer progression chiefly due to this stabilisation of telomeres in oncogenic cells, allowing them to avoid degradation, or senescence (Dratwa et al., 2020). *FRMD4A* was observed in lower expression, by negative 9.1 folds in the 'Low' palmitic acid intake group, compared to 'Medium'. *FRMD4A* plays a role in the regulation of epithelial cell polarity (asymmetry), aiding in the function of the cell, and integrity of the cell barrier (Ikenouchi & Umeda, 2010). Increased expression of *FRMD4A* is closely associated with colorectal cancer development and metastasis (Fan et al., 2016).

SOX2, a transcription factor, had a negative 6.2-fold change in the 'Low' palmitic acid intake group, compared to 'High'. *SOX2* is predominantly responsible for regulating the pluripotency (differentiation ability) of stem cells (Zhang & Cui, 2014), although recent studies have implicated high *SOX2* expression with the promotion of tumour growth and metastasis, as well as medication/drug resistance(s) in several cancers (Mirzaei et al., 2022; Wuebben & Rizzino, 2017).

For oxidative stress-related biomarkers, *CYBB* (which encodes the protein NADPH oxidase 2, or NOX2 protein) was downregulated by 2.0 folds in the 'Low' palmitic acid intake group

compared to ‘Medium’, whereas *SOD1* was upregulated by 2.4 folds in the ‘Low’ group compared to ‘High’. *CYBB* plays a key role in phagocytosis, the engulfment and destruction of various harmful pathogens, or bacteria, while also inadvertently producing (as a by-product) reactive oxygen species (ROS) (Frazão et al., 2015). High expression of *CYBB* is positively associated with gastric cancer, whereas low expression has been observed in lung cancer (Chen et al., 2021; Lang & Meng, 2025). Conversely, *SOD1* acts as an anti-oxidant, converting superoxide (ROS) into hydrogen peroxide (Afonso et al., 2007). ROS levels are directly linked to oxidative stress; associated with various chronic diseases including diabetes, neurodegenerative diseases, kidney and lung diseases, as well as cardiovascular disease (Jomova et al., 2023).

PICALM, involved in neural development, was upregulated in ‘Low’ palmitic acid intake compared to ‘High’, by 18.9 folds. *PICALM* is principally responsible for cellular transport (endocytosis) of proteins (Baig et al., 2010). Interestingly, higher *PICALM* expression has been associated with Alzheimer’s disease, and conversely, lower expression in Parkinson’s disease (Kumon et al., 2021). Relatedly, *UNC13C* was downregulated by 11.44 folds. *UNC13C* regulates cellular transport (exocytosis) of synaptic neurotransmitters (Brose et al., 2000). High *UNC13C* expression has been associated with hepatocellular carcinoma (Kumar et al., 2023). *GSK3β* had a negative 2.1-fold change in the ‘Low’ palmitic acid group compared to ‘Medium’. *GSK3β* regulates various cellular processes, including cell proliferation, function, and apoptosis, via the targeted phosphorylation of glycogen (Jacobs et al., 2012). Upregulation of *GSK3β* has been linked to neurological disorder development (Lv et al., 2016).

Interestingly, a total of 2 genes: *TLR2* and *NFκB1,* typically observed as major mechanisms within much of the literature investigating palmitic acid’s role within the immune system, did not follow similar patterns to those detailed above. This may suggest potent response(s) to palmitic acid, beginning at low (~2.75 - 6.17g/day) intake. As a membrane protein receptor, *TLR2* acts as a mediator of both pro- and anti-inflammatory processes. *TLR2*’s primary function is to recognise extraneous substances including bacteria, viruses, fungi and parasites; referred to as pathogen-associated molecular patterns (PAMPs), or damage-associated molecular patterns (DAMPs). In response to these harmful stimuli, *TLR2* will initiate signalling via the myeloid differentiation primary response 88 (MyD88) intracellular signalling pathway, leading to the production and release of pro-inflammatory cytokines (including *IL-6* and *TNF-α*) via activation of *NFκB1* (Colleselli et al., 2023). Increased *TLR2* expression has frequently been

associated with type 2 diabetes (Dasu et al., 2010; Sepehri et al., 2016; Chen et al., 2025), in addition to various pulmonary diseases (Sidletskaya et al., 2020; Arora et al., 2019). *NFκB1* (a gene which encodes the p105 protein, a precursor to p50) is a vital transcription factor responsible for inflammation and immunity via the NF-κB signalling pathway (Guo et al., 2024). Activation of the NF-κB pathway induces the expression of various pro-inflammatory cytokines, including *IL-6* and *TNF-α* (Liu et al., 2017). Increased *NFκB1* activation has been associated with various diseases, including inflammatory bowel disease (IBD), (late onset) Alzheimer's disease, Parkinson's disease, coronary artery disease/atherosclerosis, type 2 diabetes and certain cancers (Concetti & Wilson, 2018; Coto et al., 2018; Coto et al., 2019; Perez-Oliveira et al., 2024; Schreiber et al., 2008; Vazquez-Coto et al., 2025).

Another major finding of this study is that the participants with 'Medium' palmitic acid intake, exhibited a more unfavourable mRNA expression profile (typically via higher expression) when compared to the 'High' palmitic acid intake group. For instance, in energy homeostasis, *DPP4* is generally considered harmful to metabolic health, damaging hormones such as glucagon-like peptide-1 (GLP-1), which aids in glucose regulation. Gliptins, or *DPP4* inhibitors, are often used as a medication to manage (lower) blood sugar levels in individuals with type-2 diabetes (Tomkin et al., 2014). *FAM13A* is typically associated with hepatic glucose and lipid metabolism regulation (Lin et al., 2020). High expression of *FAM13A* has been observed within various diseases, including chronic obstructive pulmonary disorder (COPD), pulmonary fibrosis, in addition to liver cirrhosis (Zhang et al., 2019). *RETN*, a protein synthesised within macrophages, stimulates the expression of various pro-inflammatory cytokines, including interleukin-1 (IL1), *IL6* and *TNF-α* (Zhang et al., 2010). Elevated *RETN* expression has been positively associated with insulin resistance (Benomar et al., 2013), increased metabolic risk factors such as waist circumference, blood pressure, serum cholesterol, serum (Gupta et al., 2011) and mortality rate (Deb et al., 2021).

The *EGR2* gene aids in the protection of the peripheral nervous system (PNS), predominantly through myelination regulation, as well as the directive of T helper 17 (T_h17) cells; producing inflammatory cytokines. Increased *EGR2* expression can cause T-cell exhaustion, associated with various autoimmune and inflammatory diseases (Maddur et al., 2021). *HRH3* primarily protects the central nervous system (CNS) via regulation of histamine synthesis and metabolism (Schlicker & Kathmann, 2016). *HRH3* upregulation has been associated with tumour growth and metastasis in various forms of cancer (Medina & Rivera, 2010; Yu et al.,

2019). Anti-histamines have been positively correlated with cancer growth in mice, although there appears to be no correlation with humans (Brandes et al., 1994; Nadalin et al., 2003; Chai et al., 2025). Both *MMP2* and *MMP3*'s primary function(s) are the degradation and remodelling of the extracellular matrix (ECM). Although targeting different components, both genes are essential for the healing of damaged tissue (Page-McCaw et al., 2007). Elevated expression of both *MMP2* and *MMP3* has been associated with increased risks of cancer and cardiovascular disease (Argote et al., 2021; Bräuninger et al., 2023; Han et al., 2020), in addition to exacerbation of diabetes-related disorders including nephropathy and retinopathy (Van Hove et al., 2016; Xu et al., 2014). The *MS4A6E* gene encodes a protein within the cell membrane, and is predominantly involved in inflammatory signalling pathways (Silva-Gomes et al., 2022). Elevated levels of *MS4A6E* have been associated with (late onset) Alzheimer's disease (Naj et al., 2011).

PPP2CA aids in cellular signalling via the dephosphorylation of proteins (Wlodarchak & Xing, 2016). Expression of *PPP2CA* is positively associated with hepatocellular carcinoma (Liang et al., 2021; Yang et al., 2021). *IRS1* is an intracellular adaptor protein within chromosome 2, and is primarily involved in glucose metabolism via the insulin signalling pathway (Stoffel et al., 1993). High expressions of *IRS1* expression have been positively associated with thyroid cancer metastasis (Yu et al., 2024). *MUC5AC* protects epithelial cells from pathogen/irritant-related damage, primarily in the respiratory and gastrointestinal tracts, via formation of a mucus layer/barrier (Bonser & Erle, 2017; Olli et al., 2020). *MUC5AC* overexpression has been associated with the progression of colorectal cancer (Pothuraju et al., 2020). *PLAT* has a primary role in the breaking down of blood clots, or fibrinolysis (Cesari et al., 2010). Increased expression of *PLAT* has been associated with pancreatic adenocarcinoma (Laurell et al., 2006).

The three foremost upregulated and downregulated genes across the three palmitic acid intake groups are available to view in Table 5.9.

Table 5.9. The three foremost differentially expressed genes (by fold change) in the three palmitic acid intake groups.

		Upregulated			**Downregulated**			
	Low/High	***ABCA1***	***MS4A6E***	***PICALM***	***UNC13C***	***TLR8***	***APOE***	
		63.16	18.92	7.63	-11.44	-10.60	-8.03	

	Low/Medium	***BCL2***	***PICALM***	***CREB1***	***PLAT***	***APOE***	***UNC13C***	
		34.45	17.68	7.11	-23.35	-15.81	-13.20	
	Medium/High	***ABCA1***	***MS4A6E***	***PPP2CA***	***BCL2***	***GEM***	***CREB1***	
		86.14	25.05	9.28	-39.16	-7.71	-7.63	

BCL2 and *PLAT* expression, positively associated with cancer risk (both favourable and unfavourable effects, respectively) are upregulated (*BCL2)* and downregulated (*PLAT*) in the 'Low' dietary palmitic acid intake group, when compared with 'Medium'. Similarly, *UNC13C* and *TLR8* expression, both also associated with increased cancer risk (as well as growth and survival), are downregulated in the 'Low' palmitic acid intake group, when compared with 'Medium' and/or 'High'. Conversely, *MS4A6E* and *PICALM*, two genes associated with detrimental effects on Alzheimer's disease, are foremost upregulated in the 'Low' palmitic acid intake group.

In summary, factors such as age, gender/sex, dietary habit, as well as several anthropometrical measurements, appear to have no significant effects on palmitic acid intake. Though expectedly, differences were observed in calorie, carbohydrate, fat, as well as protein consumption, factors which understandably would differ amongst the three groups. Concerning mRNA expression, the 'Low' dietary palmitic acid intake group demonstrated several metabolic health benefits, when compared to the 'Medium' and/or 'High' dietary palmitic acid intake groups, including a favourable lipid profile, a healthy immune response, lower incidences of cancer risk, oxidative stress homeostasis, and strong neural development. Therefore, high dietary palmitic acid is implicated in higher abundances of harmful cholesterol, impaired insulin sensitivity, various inflammatory disorders and type of cancer (progression, metastases), as well as neurodegeneration.

Fascinatingly, and contrasting much of the current literature, some beneficial effects within the 'High' group were also observed, when compared to the 'Medium' group. The majority of available studies report unfavourable health outcomes in response to excessive dietary palmitic acid intake (Ceja-Galicia et al., 2025; Mei et al., 2024; Seufert et al., 2023; Zhao et al., 2024), whereas this study suggests that some form of metabolic adaptation may occur above a certain threshold (in this case, ~10g/day). It is reasonable to assume that dietary intake levels beyond the upper limit of this study (~22.2g/day) may still exacerbate adverse effects of palmitic acid. However, it is also unclear as to what value(s) of dietary palmitic acid are widely-accepted as

‘increased’, ‘elevated, or ‘excessive’, presenting difficulties in recommendations for guidelines of daily intake.

This study provides a comprehensive transcriptomic mRNA analysis of different levels of palmitic acid intake within human participants. Prosperous screening and identification of a large number of biomarkers validates the methodological approach, in reliability and reproducibility. Utilising a simple intravenous blood sample for a large-scale analysis provides the basis for future research on palmitic acid’s dietary cause-and-effect relationship, specifically within established intake parameters.

Despite the study’s strengths, there are a few evident limitations. Firstly, dietary intake data was self-reported, leading to potential inaccuracies or information biases, such as underreporting, or lapses in memory (recall bias). Secondly, there was a relatively low total number of samples (18), including a larger sample size could be more representative of the United Kingdom as a whole. Consequently, data should be interpreted as initial observations, rather than definitive findings; confirmation of results (i.e., validity, reliability) is needed via future study. Relatedly, there was an imbalance in the number of dietary habits amongst the participants, perhaps a balanced ratio should also be considered hereafter. Lastly, total RNA samples were pooled to obtain the gene expression data, leading to the loss of individual transcriptome identifiers: including unique nutrient intake, as well as genetics and lifestyle.

5.7 Conclusions

There is evidence to suggest that palmitic acid intake is positively associated with an undesirable gene expression profile, based on the mRNA profiles of three palmitic acid intake groups. Low dietary palmitic acid intake exhibited several health benefits, or homeostatic conditions, when compared to high dietary palmitic acid intake. Interestingly, high dietary palmitic acid content shared some of these beneficial characteristics, when compared with medium dietary palmitic acid intake, suggesting that metabolic adaptation may occur above a certain threshold or due to long-term exposure of palmitic acid. Further research particularly randomized control trials investigating the cause-and-effect relationship between the dietary palmitic acid and health markers are warranted.

Chapter 6. General Discussion

6.1 Research Overview

This thesis investigated the potential health effect of palm oil and palmitic acid on metabolic health and chronic disease development, with particular focus on the United Kingdom consumer, as well as plant-based/vegan diets. In **Chapter 1. Introduction and Review of Literature**, a comprehensive overview of palm oil, palmitic acid, and human metabolic health identified gaps within the literature, creating opportunities and propositioning novel areas of research for this thesis to explore.

The main aims and objectives of the thesis included:

- Further investigation of palm oil and palmitic acids' metabolic health effects.
- An evaluation of United Kingdom consumers' palm oil and palmitic acid awareness.
- Analysis of palmitic acid content in various foods, including plant-based/vegan.
- Physiological assessment of palmitic acid intake and chronic disease risk.
- Discussion of the significance of the results, in context of the available literature.

Following Chapter 1, below is a quick overview of how these objectives were addressed in the subsequent chapters:

Chapter 2. **Effects of palm oil, palmitic acid and their derivatives on metabolic health: a systematic review**

This chapter systematically examined the current literature concerning palm oil and palmitic acid, as well as any variations or derivatives, and their effects on cardiometabolic-related disorders, or chronic disease. A total of 132 interventional studies were reviewed across human, animal and in vitro populations, chiefly investigating their roles within the immune response, lipid profile, the gut microbiome, endocrine regulation, hepatic health, neurological health and carcinogenesis.

Chapter 3. Awareness and perceptions of the health effects of palm oil and palmitic acid

This chapter investigated the perceptions, behaviours and beliefs of United Kingdom consumers concerning palm oil and palmitic acid's health effects, as well as presence within the diet. Utilising a questionnaire, 251 members of the public provided information based on their anthropometry, physical activity, sleeping habits, dietary patterns, and awareness of palm oil and palmitic acid. A total of 62 participants also provided food diaries, which was utilised for the estimations of palmitic acid intake (also re-evaluated following Chapter 4).

Chapter 4. Fatty acid composition of common foods consumed in Westernised diets

This chapter analysed the fatty acid profile of various food items, based on their prevalence within Westernised diets (food diary data from Chapter 3), their relevance to the thesis, or their absence from nutritional information databases (predominantly plant-based/vegan food alternatives). Using gas chromatography, 70 foods across various food categories including edible oils, dairy foods, nut/seed-based foods, animal-origin foods, plant-based/vegan food alternatives, miscellaneous/snack food items, and palm-based foods, were analysed and their fatty acid profiles in fresh weight (as consumed) were developed. In addition, the dry matter analysis of foods was also performed, to include standardised fatty acid content information across all food categories, excluding water/moisture content and its dilution effect.

Chapter 5. An assessment of gene expression biomarkers linked to metabolic health in participants with different levels of dietary palmitic acid consumption

This chapter assessed how distinct parameters of palmitic acid intake (low, medium, high) influenced various biomarkers of cardiometabolic health and disease. In a follow-up study to Chapter 3, a total of 18 participants provided anthropometric data via manual measurement and bioelectrical impedance analysis, as well as (human) peripheral blood samples via intravenous extraction. The mRNA profile of a total 192 different gene expression markers were assessed using quantitative real-time polymerase chain reaction technology and 57 differentially expressed genes relating to energy homeostasis, immune response, cancer, oxidative stress, neuronal signalling, as well as multiple disease pathogenesis were identified.

6.2 Synthesis of Findings

The major findings from each study (chapter) are presented in Figure 6.1.

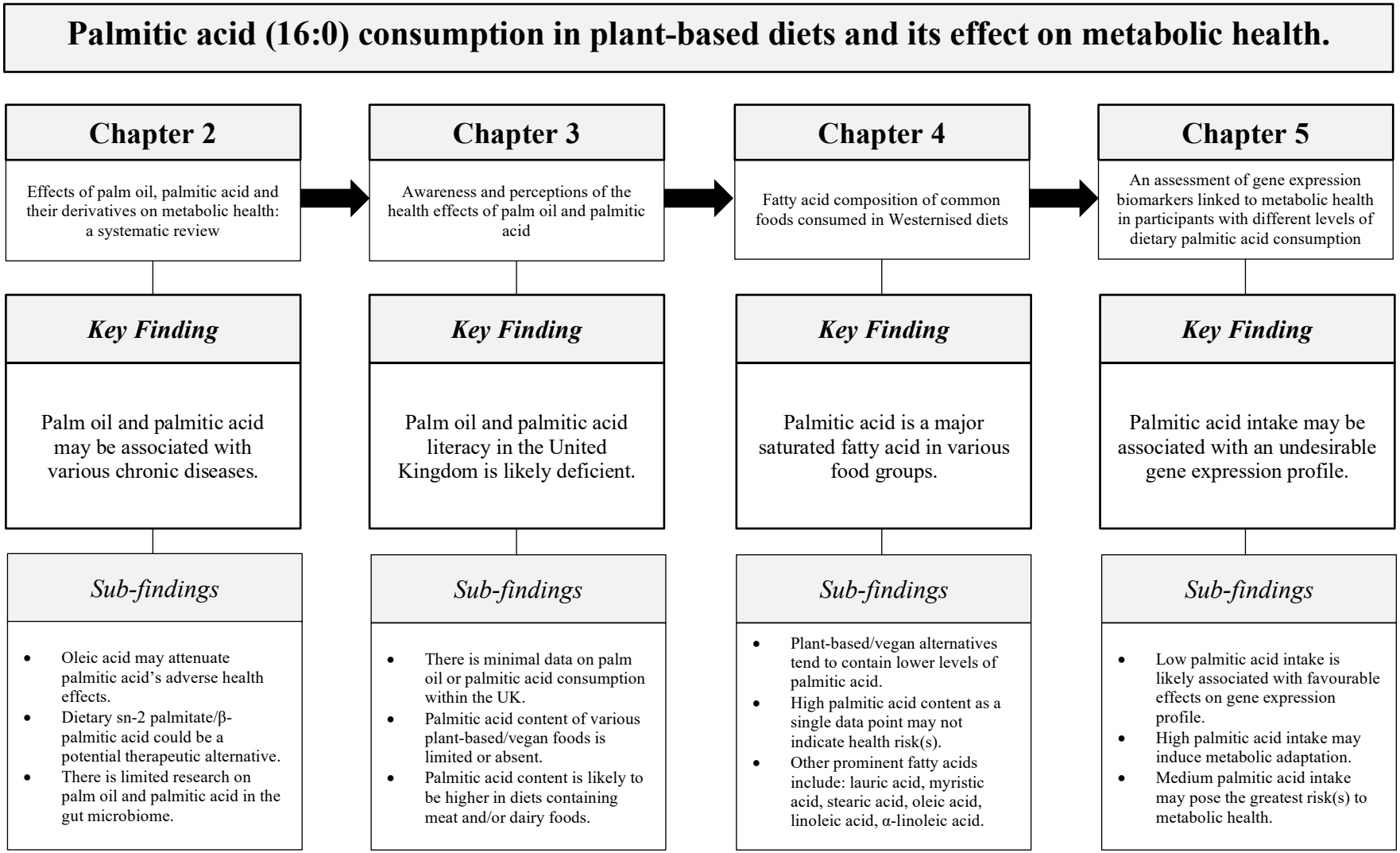


Figure 6.1. An overview of the main findings from each chapter within the thesis.

In Chapter 2, the systematic review provided evidence to suggest palmitic in particular to be associated with various chronic diseases, aligning with observations from Chapter 5, where palmitic acid content was associated with an unfavourable gene expression profile. Chapter 3 also highlighted the general public's perception on palm oil and palmitic acid. An overview of Chapter 2 and Chapter 3 outcomes is presented in Figure 6.2.

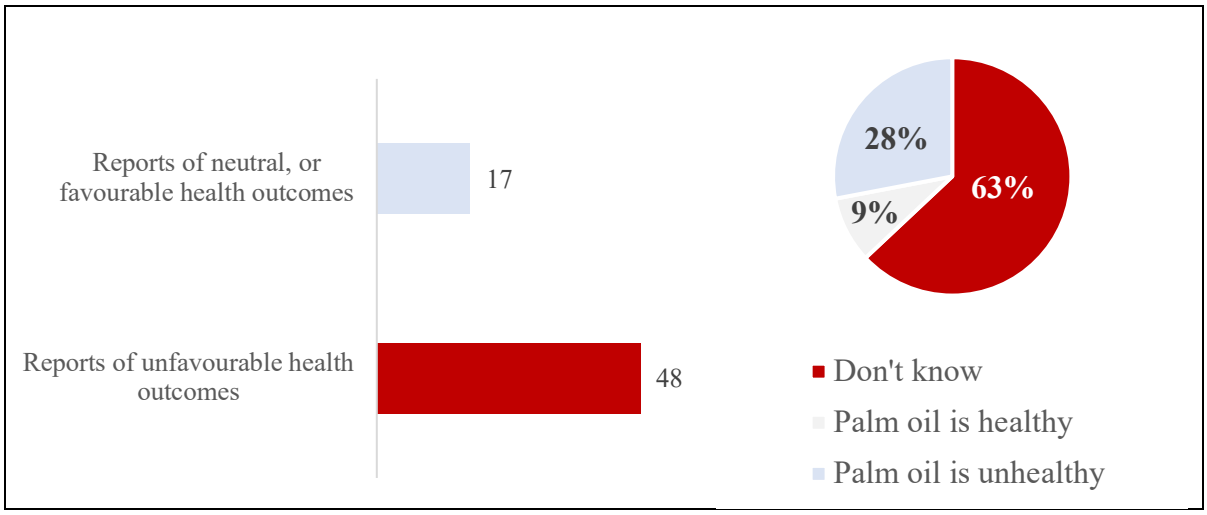


Figure 6.2. Reported health outcomes of palmitic acid (systematic review), alongside the general public's perceptions of palm oil/palmitic acid health outcomes.

Similarly, Chapter 3 and Chapter 5 provided insight into palmitic acid intake via their collective dietary data. The extent of palmitic acid present in the common dietary habits is as follows: omnivore (~8.5-9.2g per day), flexitarian (~9.7-15.5g/d), vegetarian (~6.4-7.5g/d), plant-based/vegan (~4.3-5.6g/d), and other (~6.3-10.4g/d) (Figure 6.3). These suggested that palmitic acid intake to be (comparatively) very low amongst most plant-based/vegan diets, corroborated in Chapter 4's fatty acid analysis. Moreover, the majority of plant-based/vegan alternatives, even those considered to be processed, or ultra-processed food items (faux beef burgers, bacon or pizza, as well as cheese) contained lower proportions of palmitic acid than their animal or dairy-based counterpart.

Plant-based/vegan diets may possess favourable effects on metabolic health, perhaps principally due to lower contents of palmitic acid. Undesirably, plant-based diets/veganism is still a relatively small proportion of the United Kingdom, estimated to be approximately 6.4% of the population in 2025 (Barber, 2025).

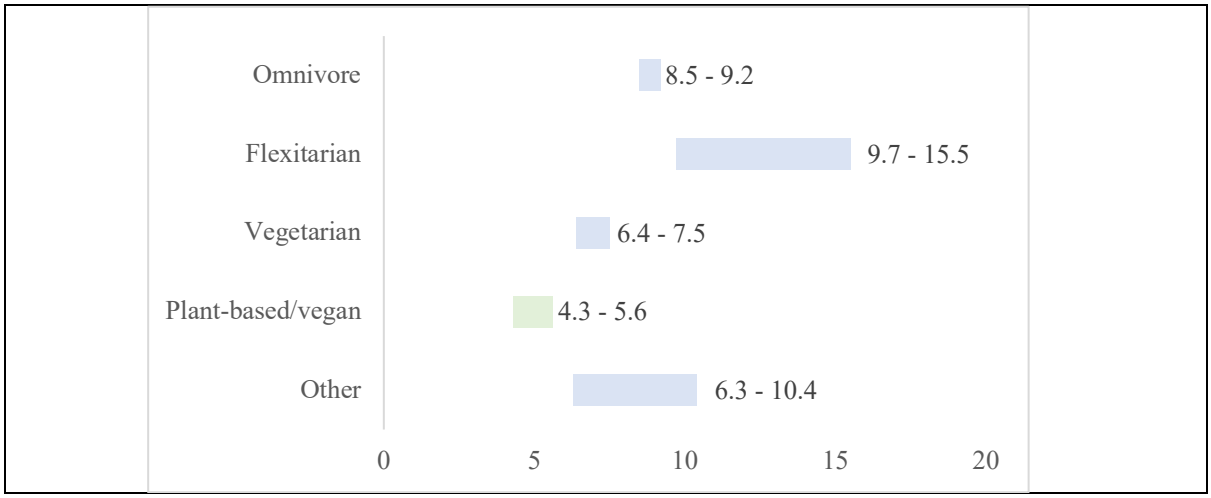


Figure 6.3. Approximate dietary palmitic acid content (grams per day) of common UK dietary habits.

From a metabolic health perspective, the outcomes of this thesis have several implications, both in isolation, and most critically due to the synergy between them. For instance, a hypothetical, but entirely conceivable scenario based on the research; the average United Kingdom consumer typically has limited knowledge on palm oil and palmitic acid's health effects, as well as their overall presence within the diet. Compounding this with the majority of health outcomes in dietary palmitic acid intake to be unfavourable, the ever-growing abundances of palmitic acid in the diet, alongside several adverse effects of palmitic acid on unfavourable biomarker expression, it would be more than reasonable to assume that alterations are necessary in order to avoid a national health epidemic trajectory.

6.3 Original contributions to the research field

Although palm oil and palmitic acid's association with metabolic health status and chronic disease development is both complex and multifaceted, the research conducted in this thesis has made several novel advancements, and crucial findings. First, it was evident that palm oil and palmitic acid are in high abundance in foods commonly associated with Westernised diets. Despite this, there are very few investigations into, nor a synthesis of data concerning, several cardiometabolic-related health effects, including the risks of chronic disease associated with specific dietary habits. Second, there is no widely-accepted data concerning dietary palm oil or palmitic acid consumption within the United Kingdom, nor is there any information on the

knowledge or awareness of either their health effect, or abundance within the diet, per the average United Kingdom consumer. This thesis was the first to provide a comprehensive, systematic overview of the metabolic health effects of palm oil, palmitic acid and their derivates, as well as the first to survey the general public of the United Kingdom regarding their perceptions of palm oil and palmitic acid. Third, palmitic acid content of foods was also limited within the literature and publicly available databases, particularly in plant-based/vegan foods. This thesis was the first to investigate fatty acid composition (including palmitic acid content) in a wide-range of common everyday foods and assessed different parameters of palmitic acid intake. Fourth, the role of dietary palm oil and palmitic acid in human body is mostly unknown. This thesis reported identification of several novel metabolic health-related biomarkers that provides the basis for future investigation on the cause-and-effect relationship.

6.4 Future recommendations

Following are some of the important areas relating to consumption of palm oil/palmitic acid and future research:

- Controlled intervention studies should investigate the role of dietary palmitic acid to oleic acid ratio, or oleic acids' potential ability to attenuate palmitic acid's adverse health effects.
- Investigations of the health effect of different derivatives of palm oil including crude palm olein, oleic acid derivatives and palm oil blends.
- Further investigations into bioactive compounds of crude/red palm oil: i.e., comparison of isolated/extracted tocopherols/carotenoids, with crude/red, and processed palm oil(s).
- Outreaching initiatives informing the general public of palm oil and palmitic acid, as well as the potential health benefits of a meat-free, dairy-free, plant-based/vegan diet.
- More comprehensive research (large-scale studies) into the extent of palm oil and palmitic acid consumption throughout the United Kingdom.
- Development of a comprehensive database for the fatty acid composition of all common everyday foods, with a focus on the contemporary inclusions such as the continuously expanding selection of plant-based/vegan alternatives.
- The exploration of palm oil and palmitic acid health effects within the gut microbiome.
- Further research (Randomized controlled trials) on the health effects of distinct dietary palmitic acid parameters, with the goal of specific daily recommendation guidelines.

References

Abdul Basith Khan, M., Hashim, M. J., King, J. K., Govender, R. D., Mustafa, H., & Al Kaabi, J. (2020). Epidemiology of type 2 diabetes—Global burden of disease and forecasted trends. *Journal of Epidemiology and Global Health*, *10*(1), 107–111.

Aboua, Y. G., Brooks, N., Mahfouz, R. Z., Agarwal, A., & Plessis, S. S. (2012). A red palm oil diet can reduce the effects of oxidative stress on rat spermatozoa. *Andrologia*, *44*, 32–40.

Abumrad, N.A., Nassi, F. and Marcus, A.K.I.V.A. (2016). *Digestion and absorption of dietary fat, carbohydrate, and protein.* Available at: https://www.academia.edu/download/43929086/Digestion_and_Absorption_of_Dietary_Fat_.pdf

Aceves-Martins, M., Bates, R. L., Craig, L. C., Chalmers, N., Horgan, G., Boskamp, B., & Roos, B. (2022). Nutritional quality, environmental impact and cost of ultra-processed foods: A UK food-based analysis. *International Journal of Environmental Research and Public Health*, *19*(6), 3191.

Afonso, V., Champy, R., Mitrovic, D., Collin, P., & Lomri, A. (2007). Reactive oxygen species and superoxide dismutases: Role in joint diseases. *Joint Bone Spine*, *74*(4), 324–329.

Ahmad, R., Al-Roub, A., Kochumon, S., Akther, N., Thomas, R., Kumari, M., Koshy, M. S., Tiss, A., Hannun, Y. A., Tuomilehto, J., & Sindhu, S. (2018). The synergy between palmitate and TNF-α for CCL2 production is dependent on the TRIF/IRF3 pathway: Implications for metabolic inflammation. *The Journal of Immunology*, *200*(10), 3599–3611.

Ahmadian, M., Duncan, R. E., Jaworski, K., Sarkadi-Nagy, E., & Sook Sul, H. (2007). Triacylglycerol metabolism in adipose tissue. *Future Lipidology*, *2*(2), 229–237.

Ahn, J. H., Kim, M. H., Kwon, H. J., Choi, S. Y., & Kwon, H. Y. (2013). Protective effects of oleic acid against palmitic acid-induced apoptosis in pancreatic AR42J cells and its mechanisms. *The Korean Journal of Physiology & Pharmacology: Official Journal of the Korean Physiological Society and the Korean Society of Pharmacology*, *17*(1), 43.

Ajibawo-Aganbi, U., Saleem, S., Khan, S. Z. A., Veliginti, S., Bastidas, M. V. P., Lungba, R. M., & Cancarevic, I. (2020). Can nutritional adequacy help evade neurodegeneration in older age? A review. *Cureus*, *12*(10).

Akbar, N., Khan, N. A., Muhammad, J. S., & Siddiqui, R. (2022). The role of gut microbiome in cancer genesis and cancer prevention. *Health Sciences Review*, *2*, 100010.

Akieda-Asai, S., Ma, H., & Date, Y. (2019). Palmitic acid induces guanylin gene expression through the Toll-like receptor 4/nuclear factor-κB pathway in rat macrophages. *American Journal of Physiology-Cell Physiology*, *317*(6), 1239–1246.

Al Obeed, O. A., Alkhayal, K. A., Al Sheikh, A., Zubaidi, A. M., Vaali-Mohammed, M. A., Boushey, R., Mckerrow, J. H., & Abdulla, M. H. (2014). Increased expression of tumor necrosis factor-α is associated with advanced colorectal cancer stages. *World Journal of Gastroenterology: WJG*, *20*(48), 18390.

Alam, M. T., Amos, G. C., Murphy, A. R., Murch, S., Wellington, E. M., & Arasaradnam, R. P. (2020). Microbial imbalance in inflammatory bowel disease patients at different taxonomic levels. *Gut Pathogens*, *12*, 1–8.

Albashir, A. A. D. (2020). The potential impacts of obesity on COVID-19. *Clinical Medicine*, *20*(4), 109.

Allen, L., Williams, J., Townsend, N., Mikkelsen, B., Roberts, N., Foster, C., & Wickramasinghe, K. (2017). Socioeconomic status and non-communicable disease behavioural risk factors in low-income and lower-middle-income countries: A systematic review. *The Lancet Global Health*, *5*(3), 277–289.

Alloubani, A., Nimer, R., & Samara, R. (2021). Relationship between hyperlipidemia, cardiovascular disease and stroke: A systematic review. *Current Cardiology Reviews*, *17*(6).

Almeida, D. T., Curvelo, F. M., Costa, M. M., Viana, T. V., & Lima, P. C. (2017). Oxidative stability of crude palm oil after deep frying akara (Fried Bean Paste. *Food Science and Technology (Campinas*, *38*(1), 142–147.

Almeida, E. S., Carmona, P. O., Mendonça, S., Dias, A. C. B., Castellón, E. R., Cecilia, J. A., Silva Júnior, I. J., & Monteiro, S. (2024). The role of carotenes in preventing oxidation during palm oil processing: Adsorption studies. *Industrial Crops and Products*, *216*, 118691.

Alnahdi, A., John, A., & Raza, H. (2019). Augmentation of glucotoxicity, oxidative stress, apoptosis and mitochondrial dysfunction in HepG2 cells by palmitic acid. *Nutrients*, *11*(9), 1979.

Alouache, A. (2025). *Vitamin A: Benefits and Consequences of Its Deficiency on Health*. Available at: https://www.intechopen.com/online-first/1218298

Althouse, A. D., & Chow, Z. R. (2019). Comment on "post-hoc power: if you must, at least try to understand". *Annals of Surgery, 270*(6), e78-e79.

Alves-Bezerra, M., & Cohen, D. E. (2017). Triglyceride metabolism in the liver. *Comprehensive Physiology*, *8*(1), 1.

Alzamil, H. (2020). Elevated serum TNF-α is related to obesity in type 2 diabetes mellitus and is associated with glycemic control and insulin resistance. *Journal of Obesity*, *2020*(1), 5076858.

Ameer, F., Scandiuzzi, L., Hasnain, S., Kalbacher, H., & Zaidi, N. (2014). De novo lipogenesis in health and disease. *Metabolism*, *63*(7), 895–902.

American Heart Associations. (2017). *Dietary Fats and Cardiovascular Disease: A Presidential Advisory From the American Heart Association.* Available at: https://www.ahajournals.org/doi/10.1161/CIR.0000000000000510

Amico, A., Wootan, M. G., Jacobson, M. F., Leung, C. I. N. D. Y., & WILLETT, A. W. (2021). The demise of artificial trans fat: A history of a public health achievement. *The Milbank Quarterly*, *99*(3), 746–770.

Amine, H., Benomar, Y., & Taouis, M. (2021). Palmitic acid promotes resistin-induced insulin resistance and inflammation in SH-SY5Y human neuroblastoma. *Scientific Reports*, *11*(1), 5427.

Amoah, I., Lim, J. J., Osei, E. O., Arthur, M., Tawiah, P., Oduro, I. N., Aduama-Larbi, M. S., Lowor, S. T., & Rush, E. (2022). Effect of cocoa beverage and dark chocolate consumption on blood pressure in those with normal and elevated blood pressure: A systematic review and meta-analysis. *Foods*, *11*(13), 1962.

Andersen, K., & Pedersen, B. K. (2008). The role of inflammation in vascular insulin resistance with focus on IL-6. *Hormone and Metabolic Research*, *40*(09), 635–639.

Anitha, M., Reichardt, F., Tabatabavakili, S., Nezami, B. G., Chassaing, B., Mwangi, S., Vijay-Kumar, M., Gewirtz, A., & Srinivasan, S. (2016). Intestinal dysbiosis contributes to the delayed gastrointestinal transit in high-fat diet fed mice. *Cellular and Molecular Gastroenterology and Hepatology*, *2*(3), 328–339.

Annevelink, C. E., Sapp, P. A., Petersen, K. S., Shearer, G. C., & Kris-Etherton, P. M. (2023). Diet-derived and diet-related endogenously produced palmitic acid: Effects on metabolic regulation and cardiovascular disease risk. *Journal of Clinical Lipidology*, *17*(5), 577–586.

Ansell, B., & Richard Hobbs, F. D. (2006). The potential for CETP inhibition to reduce cardiovascular disease risk. *Current Medical Research and Opinion*, *22*(12), 2467–2478.

Anwar, F., Bhanger, M. I., Iqbal, S., & Sultana, B. (2006). Fatty acid composition of different margarines and butters from Pakistan with special emphasis on trans unsaturated contents. *Journal of Food Quality*, *29*(1), 87–96.

Apovian, C. M. (2016). Obesity: Definition, comorbidities, causes, and burden. *Am J Manag Care*, *22*(7 Suppl), 176–185.

Argote Camacho, A. X., Gonzalez Ramirez, A. R., Perez Alonso, A. J., Rejon Garcia, J. D., Olivares Urbano, M. A., Torne Poyatos, P., Rios Arrabal, S., & Núñez, M. I. (2021). Metalloproteinases 1 and 3 as potential biomarkers in breast cancer development. *International Journal of Molecular Sciences*, *22*(16), 9012.

Arora, S., Ahmad, S., Irshad, R., Goyal, Y., Rafat, S., Siddiqui, N., Dev, K., Husain, M., Ali, S., Mohan, A., & Syed, M. A. (2019). TLRs in pulmonary diseases. *Life sciences*, *233*, 116671.

Aryana, K. J., Resurreccion, A. V. A., Chinnan, M. S., & Beuchat, L. R. (2003). Functionality of palm oil as a stabilizer in peanut butter. *Journal of Food Science*, *68*(4), 1301–1307.

Asahara, S. I., Inoue, H., & Kido, Y. (2022). Regulation of pancreatic β-cell mass by gene-environment interaction. *Diabetes & Metabolism Journal*, *46*(1), 38.

Asakura, K., Todoriki, H., & Sasaki, S. (2017). Relationship between nutrition knowledge and dietary intake among primary school children in Japan: Combined effect of children's and their guardians' knowledge. *Journal of Epidemiology*, *27*(10), 483–491.

Ascherio, A., & Willett, W. C. (1997). Health effects of trans fatty acids. *The American Journal of Clinical Nutrition*, *66*(4), 1006–1010.

Ascherio, A., Hennekens, C. H., Buring, J. E., Master, C., Stampfer, M. J., & Willett, W. C. (1994). Trans-fatty acids intake and risk of myocardial infarction. *Circulation*, *89*(1), 94–101.

Asmat, U., Abad, K., & Ismail, K. (2016). Diabetes mellitus and oxidative stress—A concise review. *Saudi Pharmaceutical Journal*, *24*(5), 547–553.

Asokapandian, S., Sreelakshmi, S., & Rajamanickam, G. (2021). *Lipids and oils: An overview. Food biopolymers: Structural, functional and nutraceutical properties*.

Astrup, A., Magkos, F., Bier, D. M., Brenna, J. T., Oliveira Otto, M. C., Hill, J. O., King, J. C., Mente, A., Ordovas, J. M., Volek, J. S., & Yusuf, S. (2020). Saturated fats and health: A reassessment and proposal for food-based recommendations: JACC state-of-the-art review. *Journal of the American College of Cardiology*, *76*(7), 844–857.

Azzam, A. (2021). Is the world converging to a 'Western diet'? *Public Health Nutrition*, *24*(2), 309–317.

Baer, D. J., Judd, J. T., Clevidence, B. A., & Tracy, R. P. (2004). Dietary fatty acids affect plasma markers of inflammation in healthy men fed controlled diets: A randomized crossover study. *The American Journal of Clinical Nutrition*, *79*(6), 969–973.

Bahar, B., Doherty, J. V. O., & TJ, S. (2016). A cold water extract of Fucus vesiculosus inhibits lipopolysaccharide (LPS) induced pro-inflammatory responses in the porcine colon ex-vivo model. *Innov Food Sci Emerg Technol*, *37*, 229–236.

Baig, S., Joseph, S. A., Tayler, H., Abraham, R., Owen, M. J., Williams, J., Kehoe, P. G., & Love, S. (2010). Distribution and expression of picalm in Alzheimer disease. *Journal of Neuropathology & Experimental Neurology*, *69*(10), 1071–1077.

Baird, L., & Dinkova-Kostova, A. T. (2011). The cytoprotective role of the Keap1–Nrf2 pathway. *Archives of Toxicology*, *85*, 241–272.

Baird, L., & Yamamoto, M. (2020). The Keap1-Nrf2 pathway: From mechanism to medical applications. In *Oxidative Stress*, 125–147. Academic Press.

Bakaloudi, D. R., Halloran, A., Rippin, H. L., Oikonomidou, A. C., Dardavesis, T. I., Williams, J., Wickramasinghe, K., Breda, J., & Chourdakis, M. (2021). Intake and adequacy of the vegan diet. A systematic review of the evidence. *Clinical Nutrition*, *40*(5), 3503–3521.

Baker, P., Machado, P., Santos, T., Sievert, K., Backholer, K., Hadjikakou, M., Russell, C., Huse, O., Bell, C., Scrinis, G., & Worsley, A. (2020). Ultra-processed foods and the nutrition transition: Global, regional and national trends, food systems transformations and political economy drivers. *Obesity Reviews*, *21*(12), 13126.

Bar-Yoseph, F., Lifshitz, Y., & Cohen, T. (2013). Review of sn-2 palmitate oil implications for infant health. *Prostaglandins, Leukotrienes and Essential Fatty Acids*, *89*(4), 139–143.

Barber, S. (Finder). (2025). *How many vegetarians and vegans are in the UK in 2025?* Available at: https://www.finder.com/uk/stats-facts/uk-diet-trends

Bardelčíková, A., Šoltys, J., & Mojžiš, J. (2023). Oxidative stress, inflammation and colorectal cancer: An overview. *Antioxidants*, *12*(4), 901.

Barkun, A., Love, J., Gould, M., Pluta, H., & Steinhart, A. H. (2013). Bile acid malabsorption in chronic diarrhea: Pathophysiology and treatment. *Canadian Journal of Gastroenterology and Hepatology*, *27*(11), 653–659.

Bassett, J. K., Severi, G., Hodge, A. M., MacInnis, R. J., Gibson, R. A., Hopper, J. L., English, D. R., & Giles, G. G. (2013). Plasma phospholipid fatty acids, dietary fatty acids and prostate cancer risk. *International journal of cancer*, *133*(8), 1882–1891.

Basson, A. R., Chen, C., Sagl, F., Trotter, A., Bederman, I., Gomez-Nguyen, A., Sundrud, M. S., Ilic, S., Cominelli, F., & Rodriguez-Palacios, A. (2021). Regulation of intestinal inflammation by dietary fats. *Frontiers in Immunology*, *11*, 604989.

Bastide, N. M., Pierre, F. H., & Corpet, D. E. (2011). Heme iron from meat and risk of colorectal cancer: A meta-analysis and a review of the mechanisms involved. *Cancer Prevention Research*, *4*(2), 177–184.

Beenakkers, A. T., Horst, D. J., & Marrewijk, W. J. A. (1981). Role of lipids in energy metabolism. *Energy Metabolism in Insects*, 53–100.

Béghin, L., Marchandise, X., Lien, E., Bricout, M., Bernet, J. P., Lienhardt, J. F., Jeannerot, F., Menet, V., Requillart, J. C., Marx, J., & Groot, N. (2019). Growth, stool consistency and bone mineral content in healthy term infants fed sn-2-palmitate-enriched starter infant formula: A randomized, double-blind, multicentre clinical trial. *Clinical Nutrition*, *38*(3), 1023–1030.

Ben-Dror, K., & Birk, R. (2019). Oleic acid ameliorates palmitic acid-induced ER stress and inflammation markers in naive and cerulein-treated exocrine pancreas cells. *Bioscience Reports*, *39*(5), 20190054.

Bennedsen, A. L., Furbo, S., Bjarnsholt, T., Raskov, H., Gögenur, I., & Kvich, L. (2022). The gut microbiota can orchestrate the signaling pathways in colorectal cancer. *Apmis*, *130*(3), 121–139.

Benomar, Y., Gertler, A., Lacy, P., Crépin, D., Ould Hamouda, H., Riffault, L., & Taouis, M. (2013). Central resistin overexposure induces insulin resistance through Toll-like receptor 4. *Diabetes*, *62*(1), 102–114.

Berberich, A. J., & Hegele, R. A. (2022). A modern approach to dyslipidemia. *Endocrine Reviews*, *43*(4), 611–653.

Berchtold, L. A., Prause, M., Størling, J., & Mandrup-Poulsen, T. (2016). Cytokines and pancreatic β-cell apoptosis. *Advances in Clinical Chemistry*, *75*, 99–158.

Bernardi, F., & Mariani, G. (2021). Biochemical, molecular and clinical aspects of coagulation factor VII and its role in hemostasis and thrombosis. *Haematologica*, *106*(2), 351.

Bianchi, V. E., Herrera, P. F., & Laura, R. (2021). Effect of nutrition on neurodegenerative diseases. A systematic review. *Nutritional Neuroscience*, *24*(10), 810–834.

Billany, R. E., Thopte, A., Adenwalla, S. F., March, D. S., Burton, J. O., & Graham-Brown, M. P. (2023). Associations of health literacy with self-management behaviours and health outcomes in chronic kidney disease: A systematic review. *Journal of Nephrology*, *36*(5), 1267–1281.

Bjermo, H., Iggman, D., Kullberg, J., Dahlman, I., Johansson, L., Persson, L., Berglund, J., Pulkki, K., Basu, S., Uusitupa, M., & Rudling, M. (2012). Effects of n-6 PUFAs compared with SFAs on liver fat, lipoproteins, and inflammation in abdominal obesity: A randomized controlled trial. *The American Journal of Clinical Nutrition*, *95*(5), 1003–1012.

Boariu, S. M., Scutariu, A. M., Reurean Pintilei, D., Tarcea, M., Guiné, R. P., & Ferreira, M. (2024). Food literacy assessment of a sample of Romanian higher education students. *Sustainability*, *16*(3), 1034.

Boateng, J., Verghese, M., Chawan, C. B., Shackelford, L., Walker, L. T., Khatiwada, J., & Williams, D. S. (2006). Red palm oil suppresses the formation of azoxymethane (AOM) induced aberrant crypt foci (ACF) in Fisher 344 male rats. *Food and Chemical Toxicology*, *44*(10), 1667–1673.

Boateng, L., Ansong, R., Owusu, W., & Steiner-Asiedu, M. (2016). Coconut oil and palm oil's role in nutrition, health and national development: A review. *Ghana Medical Journal*, *50*(3), 189–196.

Bobo, G., Nicolau-Lapeña, I., & Aguiló-Aguayo, I. (2021). Conventional Oils and Oilseeds: Composition and Nutritional Importance. *Oil and Oilseed Processing: Opportunities and Challenges*, *p.23*.

Bogueva, D., & McClements, D. J. (2023). Safety and Nutritional Risks Associated with Plant-Based Meat Alternatives. *Sustainability*, *15*(19), 14336.

Bonanome, A., & Grundy, S. M. (1988). Effect of dietary stearic acid on plasma cholesterol and lipoprotein levels. *New England Journal of Medicine*, *318*(19), 1244–1248.

Bonser, L. R., & Erle, D. J. (2017). Airway mucus and asthma: The role of MUC5AC and MUC5B. *Journal of Clinical Medicine*, *6*(12), 112.

Boon, C. M., Ng, M. H., Choo, Y. M., & Mok, S. L. (2013). Super, red palm and palm oleins improve the blood pressure, heart size, aortic media thickness and lipid profile in spontaneously hypertensive rats. *PloS One*, *8*(2), 55908.

Boren, J., Chapman, M. J., Krauss, R. M., Packard, C. J., Bentzon, J. F., Binder, C. J., ... & Ginsberg, H. N. (2020). Low-density lipoproteins cause atherosclerotic cardiovascular disease: pathophysiological, genetic, and therapeutic insights: a consensus statement from the European Atherosclerosis Society Consensus Panel. *European heart journal*, *41*(24), 2313-2330.

Boskou, D., Blekas, G., & Tsimidou, M. (2006). Olive oil composition. In *Olive oil* (pp. 41–72). AOCS press.

Brandes, L. J., Warrington, R. C., Arron, R. J., Bogdanovic, R. P., Fang, W., Queen, G. M., Stein, D. A., Tong, J., Zaborniak, C. L., & LaBella, F. S. (1994). Enhanced cancer growth in mice administered daily human-equivalent doses of some H1-antihistamines: Predictive in vitro correlates. *JNCI: Journal of the National Cancer Institute*, *86*(10), 770–775.

Bräuninger, H., Krüger, S., Bacmeister, L., Nyström, A., Eyerich, K., Westermann, D., & Lindner, D. (2023). Matrix metalloproteinases in coronary artery disease and myocardial infarction. *Basic Research in Cardiology*, *118*(1), 18.

Bressan, J., & Hermsdorff, H. H. (2016). Saturated fatty acids trigger TLR4-mediated inflammatory response. *Atherosclerosis*, *244*, 211–215.

Briggs, M. A., Petersen, K. S., & Kris-Etherton, P. M. (2017). Saturated fatty acids and cardiovascular disease: Replacements for saturated fat to reduce cardiovascular risk. *Healthcare*, *5*(2), 29.

Brodeur, M. R., Bouvet, C., Barrette, M., & Moreau, P. (2013). Palmitic acid increases medial calcification by inducing oxidative stress. *Journal of Vascular Research*, *50*(5), 430–441.

Brody, T. (1999). Digestion and absorption of lipids. *Nutritional biochemistry*, 93.

Brøns, C., Jensen, C. B., Storgaard, H., Hiscock, N. J., White, A., Appel, J. S., Jacobsen, S., Nilsson, E., Larsen, C. M., Astrup, A., & Quistorff, B. (2009). Impact of short-term high-fat feeding on glucose and insulin metabolism in young healthy men. *The Journal of Physiology*, *587*(10), 2387–2397.

Brose, N., Rosenmund, C., & Rettig, J. (2000). Regulation of transmitter release by Unc-13 and its homologues. *Current Opinion in Neurobiology*, *10*(3), 303–311.

Brown, K. F., Rumgay, H., Dunlop, C., Ryan, M., Quartly, F., Cox, A., Deas, A., Elliss-Brookes, L., Gavin, A., Hounsome, L., & Huws, D. (2018). The fraction of cancer attributable to modifiable risk factors in England, Wales, Scotland, Northern Ireland, and the United Kingdom in 2015. *British Journal of Cancer*, *118*(8), 1130–1141.

Brunton, N. P., Mason, C., & Collins Jr, M. J. (2015). Rapid microwave assisted preparation of fatty acid methyl esters for the analysis of fatty acid profiles in foods. *Journal of Analytical Chemistry*, *70*(10), 1218-1224.

Bryda, E. C. (2013). The Mighty Mouse: The impact of rodents on advances in biomedical research. *Missouri Medicine*, *110*(3), 207.

Budin, S. B., Othman, F., Louis, S. R., Bakar, M. A., Das, S., & Mohamed, J. (2009). The effects of palm oil tocotrienol-rich fraction supplementation on biochemical parameters, oxidative stress and the vascular wall of streptozotocin-induced diabetic rats. *Clinics*, *64*(3), 235–244.

Burela, S., He, M., Trontzas, I. P., Gavrielatou, N., Schalper, K. A., Langermann, S., Flies, D. B., Rimm, D. L., & Aung, T. N. (2024). BCAM (basal cell adhesion molecule) protein expression in different tumor populations. *Discover Oncology*, *15*(1), 381.

Byrne, C. D., & Targher, G. (2015). NAFLD: a multisystem disease. *Journal of Hepatology*, *62*(1), 47–64.

Cai, H., Santiago, F. S., Prado-Lourenco, L., Wang, B., Patrikakis, M., Davenport, M. P., Maghzal, G. J., Stocker, R., Parish, C. R., Chong, B. H., & Lieschke, G. J. (2012). DNAzyme targeting c-jun suppresses skin cancer growth. *Science translational medicine*, *4*(139), 139 82-139 82.

Calder, P. C. (2015). Functional roles of fatty acids and their effects on human health. *Journal of Parenteral and Enteral Nutrition*, *39*, 18–32.

Caldwell, S., Ikura, Y., Dias, D., Isomoto, K., Yabu, A., Moskaluk, C., Pramoonjago, P., Simmons, W., Scruggs, H., Rosenbaum, N., & Wilkinson, T. (2010). Hepatocellular ballooning in NASH. *Journal of hepatology*, *53*(4), 719–723.

Calle, E. E., Rodriguez, C., Walker-Thurmond, K., & Thun, M. J. (2003). Overweight, obesity, and mortality from cancer in a prospectively studied cohort of US adults. *New England Journal of Medicine*, *348*(17), 1625–1638.

Campos, S., Doxey, J., & Hammond, D. (2011). Nutrition labels on pre-packaged foods: A systematic review. *Public Health Nutrition*, *14*(8), 1496–1506.

Candela, C. G., López, L. B., & Kohen, V. L. (2011). Importance of a balanced omega 6/omega 3 ratio for the maintenance of health. *Nutritional Recommendations. Nutricion Hospitalaria*, *26*(2), 323–329.

Canfield, L. M., Kaminsky, R. G., Taren, D. L., Shaw, E., & Sander, J. K. (2001). Red palm oil in the maternal diet increases provitamin A carotenoids in breastmilk and serum of the mother-infant dyad. *European Journal of Nutrition*, *40*(1), 30–38.

Caponio, F., Pasqualone, A., & Gomes, T. (2003). Changes in the fatty acid composition of vegetable oils in model doughs submitted to conventional or microwave heating. *International Journal of Food Science and Technology*, *38*(4), 481–486.

Carding, S., Verbeke, K., Vipond, D. T., Corfe, B. M., & Owen, L. J. (2015). Dysbiosis of the gut microbiota in disease. *Microbial Ecology in Health and Disease*, *26*(1), 26191.

Carnielli, V. P., Luijendijk, I. H., Goudoever, J. B., Sulkers, E. J., Boerlage, A. A., Degenhart, H. J., & Sauer, P. J. (1996). Structural position and amount of palmitic acid in infant formulas: Effects on fat, fatty acid, and mineral balance. *Journal of Pediatric Gastroenterology and Nutrition*, *23*(5), 553–560.

Carta, G., Murru, E., Banni, S., & Manca, C. (2017). Palmitic acid: Physiological role, metabolism and nutritional implications. *Frontiers in Physiology*, *8*, 902.

Cassells, H. B., & Haffner, S. M. (2006). The metabolic syndrome: Risk factors and management. *Journal of Cardiovascular Nursing*, *21*(4), 306–313.

Cater, N. B., & Denke, M. A. (2001). Behenic acid is a cholesterol-raising saturated fatty acid in humans123. *The American Journal of Clinical Nutrition*, *73*(1), 41–44.

Cater, N. B., Heller, H. J., & Denke, M. A. (1997). Comparison of the effects of medium-chain triacylglycerols, palm oil, and high oleic acid sunflower oil on plasma triacylglycerol fatty acids and lipid and lipoprotein concentrations in humans. *The American Journal of Clinical Nutrition*, *65*(1), 41–45.

Ceja-Galicia, Z. A., Cespedes-Acuña, C. L. A., & El-Hafidi, M. (2025). Protection strategies against palmitic acid-induced lipotoxicity in metabolic syndrome and related diseases. *International Journal of Molecular Sciences*, *26*(2), 788.

Centers for Disease Control and Prevention. (2024). *National Diabetes Statistics Report.* Available at: https://www.cdc.gov/diabetes/php/data-research/index.html

Cesari, M., Pahor, M., & Incalzi, R. A. (2010). Plasminogen activator inhibitor-1 (PAI-1): A key factor linking fibrinolysis and age-related subclinical and clinical conditions. *Cardiovascular Therapeutics*, *28*(5), 72–91.

Chai, K. C., Chou, T. C., Chen, W. M., Shia, B. C., Soong, R. S., & Wu, S. Y. (2025). Unlocking the Potential: Antihistamine Use and Breast Cancer Risk in Women With Type 2 Diabetes Mellitus. *Journal of the National Comprehensive Cancer Network*, *23*(2), 34–40.

Chajès, V., Assi, N., Biessy, C., Ferrari, P., Rinaldi, S., Slimani, N., Lenoir, G. M., Baglietto, L., His, M., Boutron-Ruault, M. C., & Trichopoulou, A. (2017). A prospective evaluation of plasma phospholipid fatty acids and breast cancer risk in the EPIC study. *Annals of Oncology*, *28*(11), 2836–2842.

Chakraborty, G., Nandakumar, S., Hirani, R., Nguyen, B., Stopsack, K. H., Kreitzer, C., Rajanala, S. H., Ghale, R., Mazzu, Y. Z., Pillarsetty, N. V. K., & Lee, G. S. M. (2022). The impact of PIK3R1 mutations and insulin–PI3K–glycolytic pathway regulation in prostate cancer. *Clinical Cancer Research*, *28*(16), 3603–3617.

Chambers, W. (1879). *Chambers's encyclopædia: A dictionary of universal knowledge for the people* (Vol. 2). JB Lippincott & Company.

Chang, C., & Lin, H. (2016). Dysbiosis in gastrointestinal disorders. *Best Practice & Research Clinical Gastroenterology*, *30*(1), 3–15.

Chang, K., Gunter, M. J., Rauber, F., Levy, R. B., Huybrechts, I., Kliemann, N., Millett, C., & Vamos, E. P. (2023). Ultra-processed food consumption, cancer risk and cancer mortality: A large-scale prospective analysis within the UK Biobank. *EClinicalMedicine*, *56*.

Charles-Messance, H., Mitchelson, K. A., Castro, E. D. M., Sheedy, F. J., & Roche, H. M. (2020). Regulating metabolic inflammation by nutritional modulation. *Journal of Allergy and Clinical Immunology*, *146*(4), 706–720.

Chatterjee, S., Khunti, K., & Davies, M. J. (2017). Type 2 diabetes. *The lancet*, *389*(10085), 2239–2251.

Checa, J., & Aran, J. M. (2020). Reactive oxygen species: Drivers of physiological and pathological processes. *Journal of Inflammation Research*, 1057–1073.

Chemical Entities of Biological Interest. (2021). *CHEBI:35366 - fatty acid*. Available at: https://www.ebi.ac.uk/chebi/searchId.do?chebiId=35366

Chen, H., Sun, Q., Zhang, C., She, J., Cao, S., Cao, M., Zhang, N., Adiila, A. V., Zhong, J., Yao, C., & Wang, Y. (2021). Identification and validation of CYBB, CD86, and C3AR1 as the key genes related to macrophage infiltration of gastric cancer. *Frontiers in Molecular Biosciences*, *8*, 756085.

Chen, L., Zhao, Z. W., Zeng, P. H., Zhou, Y. J., & Yin, W. J. (2022). Molecular mechanisms for ABCA1-mediated cholesterol efflux. *Cell Cycle*, *21*(11), 1121–1139.

Chen, R., Tang, X., Wang, Y., Wang, B., & Mao, F. (2025). Protein palmitoylation: an emerging regulator of inflammatory signaling and diseases. *Frontiers in Immunology*, *16*, 1652741.

Chen, S., Xie, M., & Liu, Y. (2025). TLR2 promotes the progression of diabetes mellitus with atherosclerosis via activating NLRP3 inflammasome and MyD88/NF-κB signaling pathway. *Scientific Reports*, *15*(1), 16348.

Chen, X., Li, L., Liu, X., Luo, R., Liao, G., Li, L., & Chen, Y. (2018). Oleic acid protects saturated fatty acid mediated lipotoxicity in hepatocytes and rat of non-alcoholic steatohepatitis. *Life Sciences*, *203*, 291–304.

Cheng, J., Bai, W., & Caranasos, T. (2025). MicroRNAs and mRNAs: A Comparative Analysis in Translation and Commercialization. *Engineered Regeneration*.

Cheng, L., Yu, Y., Szabo, A., Wu, Y., Wang, H., Camer, D., & Huang, X. F. (2015). Palmitic acid induces central leptin resistance and impairs hepatic glucose and lipid metabolism in male mice. *The Journal of Nutritional Biochemistry*, *26*(5), 541–548.

Cherfils-Vicini, J., Platonova, S., Gillard, M., Laurans, L., Validire, P., Caliandro, R., Magdeleinat, P., Mami-Chouaib, F., Dieu-Nosjean, M. C., Fridman, W. H., & Damotte, D. (2010). Triggering of TLR7 and TLR8 expressed by human lung cancer cells induces cell survival and chemoresistance. *The Journal of Clinical Investigation*, *120*(4), 1285–1297.

Chew, N. W., Ng, C. H., Tan, D. J. H., Kong, G., Lin, C., Chin, Y. H., Lim, W. H., Huang, D. Q., Quek, J., Fu, C. E., & Xiao, J. (2023). The global burden of metabolic disease: Data from 2000 to 2019. *Cell Metabolism*, *35*(3), 414–428.

Chianese, R., Coccurello, R., Viggiano, A., Scafuro, M., Fiore, M., Coppola, G., Operto, F. F., Fasano, S., Laye, S., Pierantoni, R., & Meccariello, R. (2018). Impact of dietary fats on brain functions. *Current Neuropharmacology*, *16*(7), 1059–1085.

Chiu, H. H., & Kuo, C. H. (2020). Gas chromatography-mass spectrometry-based analytical strategies for fatty acid analysis in biological samples. *Journal of Food and Drug Analysis*, *28*(1), 60–73.

Chothe, P. P., Czuba, L. C., Moore, R. H., & Swaan, P. W. (2018). Human bile acid transporter ASBT (SLC10A2) forms functional non-covalent homodimers and higher order oligomers. *Biochimica et Biophysica Acta (BBA)-Biomembranes*, *1860*(3), 645–653.

Choudhury, N., Tan, L., & Truswell, A. S. (1995). Comparison of palmolein and olive oil: Effects on plasma lipids and vitamin E in young adults. *The American Journal of Clinical Nutrition*, *61*(5), 1043–1051.

Chow, C.K. (2007). *Fatty acids in foods and their health implications*. Available at: https://www.google.co.uk/books/edition/_/Hcl0fkcrfbEC?hl=en&gbpv=0

Christoph, M. J., An, R., & Ellison, B. (2016). Correlates of nutrition label use among college students and young adults: A review. *Public Health Nutrition*, *19*(12), 2135–2148.

Christoph, M. J., Larson, N., Laska, M. N., & Neumark-Sztainer, D. (2018). Nutrition facts panels: Who uses them, what do they use, and how does use relate to dietary intake? *Journal of the Academy of Nutrition and Dietetics*, *118*(2), 217–228.

Chun, Y. J., Sohn, S. K., Song, H. K., Lee, S. M., Youn, Y. H., Lee, S., & Park, H. (2015). Associations of colorectal cancer incidence with nutrient and food group intakes in Korean adults: A case-control study. *Clinical Nutrition Research*, *4*(2), 110.

Chung, N. (2023). Impact of the ketogenic diet on body fat, muscle mass, and exercise performance: a review. *Physical Activity and Nutrition*, *27*(4), 1.

Church, S. M. (2006). The history of food composition databases. *Nutrition Bulletin*, *31*(1), 15–20.

Civardi, E., Garofoli, F., Longo, S., Mongini, M. E., Grenci, B., Mazzucchelli, I., Angelini, M., Castellazzi, A., Fasano, F., Grinzato, A., & Fanos, V. (2017). Safety, growth, and support to healthy gut microbiota by an infant formula enriched with functional compounds. *Clinical Nutrition*, *36*(1), 238–245.

Clandinin, M. T., Cheema, S., Field, C. J., Garg, M. L., Venkatraman, J., & Clandinin, T. R. (1991). Dietary fat: Exogenous determination of membrane structure and cell function. *The FASEB Journal*, *5*(13), 2761–2769.

Clandinin, M. T., Cook, S. L., Konrad, S. D., Goh, Y. K., & French, M. A. (1999a). The effect of palmitic acid on lipoprotein cholesterol levels and endogenous cholesterol synthesis in hyperlipidemic subjects. *Lipids*, *34*, 121–124.

Clegg, M. E., Ribes, A. T., Reynolds, R., Kliem, K., & Stergiadis, S. (2021). A comparative assessment of the nutritional composition of dairy and plant-based dairy alternatives

available for sale in the UK and the implications for consumers' dietary intakes. *Food Research International*, *148*, 110586.

Clemente-Suárez, V. J., Beltrán-Velasco, A. I., Redondo-Flórez, L., Martín-Rodríguez, A., & Tornero-Aguilera, J. F. (2023). Global impacts of western diet and its effects on metabolism and health: A narrative review. *Nutrients*, *15*(12), 2749.

Cochrane (2024). *9.1.4. When not to use meta-analysis in a review.* Available at: https://handbook-5-1.cochrane.org/chapter_9/9_1_4_when_not_to_use_meta_analysis_in_a_review.htm

Coker, O. O., Wu, W. K. K., Wong, S. H., Sung, J. J., & Yu, J. (2020). Altered gut archaea composition and interaction with bacteria are associated with colorectal cancer. *Gastroenterology*, *159*(4), 1459–1470.

Colleselli, K., Stierschneider, A., & Wiesner, C. (2023). An update on toll-like receptor 2, its function and dimerization in pro-and anti-inflammatory processes. *International Journal of Molecular Sciences*, *24*(15), 12464.

Concetti, J., & Wilson, C. L. (2018). NFKB1 and cancer: Friend or foe? *Cells*, *7*(9), 133.

Conlon, M. A., Sambanthamurthi, R., Tan, Y. A., Sundram, K., Fairus, S., & Abeywardena, M. Y. (2020). Consumption of an oil palm fruit extract promotes large bowel health in rats. *Nutrients*, *12*(3), 644.

Cook-Mills, J. M., Marchese, M. E., & Abdala-Valencia, H. (2011). Vascular cell adhesion molecule-1 expression and signaling during disease: Regulation by reactive oxygen species and antioxidants. *Antioxidants & Redox Signaling*, *15*(6), 1607–1638.

Cordain, L., Eaton, S. B., Sebastian, A., Mann, N., Lindeberg, S., Watkins, B. A., O'Keefe, J. H., & Brand-Miller, J. (2005). Origins and evolution of the Western diet: Health implications for the 21st century. *The American Journal of Clinical Nutrition*, *81*(2), 341–354.

Coto, E., Díaz-Corte, C., Tranche, S., Gómez, J., Alonso, B., Iglesias, S., Reguero, J. R., López-Larrea, C., & Coto-Segura, P. (2018). Gene variants in the NF-KB pathway (NFKB1, NFKBIA, NFKBIZ) and their association with type 2 diabetes and impaired renal function. *Human Immunology*, *79*(6), 494–498.

Coto, E., Reguero, J. R., Avanzas, P., Pascual, I., Martín, M., Hevia, S., Morís, C., Díaz-Molina, B., Lambert, J. L., Alonso, B., & Cuesta-LLavona, E. (2019). Gene variants in the NF-KB pathway (NFKB1, NFKBIA, NFKBIZ) and risk for early-onset coronary artery disease. *Immunology Letters*, *208*, 39–43.

Coughlin, S. S., Vernon, M., Hatzigeorgiou, C., & George, V. (2020). Health literacy, social determinants of health, and disease prevention and control. *Journal of Environment and Health Sciences*, *6*(1), 3061.

Coussens, L. M., & Werb, Z. (2002). Inflammation and cancer. *Nature*, *420*(6917), 860–867.

Craig, M., Yarrarapu, S. N. S., & Dimri, M. (2018). Biochemistry, cholesterol. *StatPearls Publishing, Treasure Island (FL).* Available at: https://europepmc.org/article/nbk/nbk513326

Craig, W. J. (2009). Health effects of vegan diets. *The American Journal of Clinical Nutrition*, *89*(5), 1627–1633.

Crescenzo, R., Mazzoli, A., Cancelliere, R., Bianco, F., Giacco, A., Liverini, G., Dulloo, A. G., & Iossa, S. (2017). Polyunsaturated fatty acids stimulate de novo lipogenesis and improve glucose homeostasis during refeeding with high fat diet. *Frontiers in Physiology*, *8*, 248144.

Cresci, G. A., & Bawden, E. (2015). Gut microbiome: What we do and don't know. *Nutrition in Clinical Practice*, *30*(6), 734–746.

Crupkin, M., & Zambelli, A. (2008). Detrimental impact of trans fats on human health: Stearic acid-rich fats as possible substitutes. *Comprehensive Reviews in Food Science and Food Safety*, *7*(3), 271–279.

Csala, M., Kardon, T., Legeza, B., Lizák, B., Mandl, J., Margittai, É., & Bánhegyi, G. (2015). On the role of 4-hydroxynonenal in health and disease. *Biochimica et Biophysica Acta (BBA)-Molecular Basis of Disease*, *1852*(5), 826–838.

Cuesta, C., Rodenas, S., Merinero, M., Rodriguez-Gil, S., & Sanchez-Muniz, F. J. (1998). Lipoprotein profiles and serum peroxide levels of aged women consuming palmolein or oleic acid-rich sunflower oil diets. *European Journal of Clinical Nutrition*, *52*(9), 675–683.

Cunha, M., Saboga-Nunes, L., & Cunha, B. (2017). Education for health, dietary habits, nutritional status and indicators of metabolic risk. *Procedia-Social and Behavioral Sciences*, *237*, 875–881.

Dasu, M. R., Devaraj, S., Park, S., & Jialal, I. (2010). Increased toll-like receptor (TLR) activation and TLR ligands in recently diagnosed type 2 diabetic subjects. *Diabetes Care*, *33*(4), 861–868.

Davis, C., Bryan, J., Hodgson, J., & Murphy, K. (2015). Definition of the Mediterranean diet: A literature review. *Nutrients*, *7*(11), 9139–9153.

Davis, T. C., Williams, M. V., Marin, E., Parker, R. M., & Glass, J. (2002). Health literacy and cancer communication. *CA: A Cancer Journal for Clinicians*, *52*(3), 134–149.

Dayrit, F. M. (2015). The properties of lauric acid and their significance in coconut oil. *Journal of the American Oil Chemists' Society*, *92*, 1–15.

De, S. K., Devadas, K., & Notkins, A. L. (2002). Elevated levels of tumor necrosis factor alpha (TNF-α) in human immunodeficiency virus type 1-transgenic mice: Prevention of death by antibody to TNF-α. *Journal of Virology*, *76*(22), 11710–11714.

Deb, A., Deshmukh, B., Ramteke, P., Bhati, F. K., & Bhat, M. K. (2021). Resistin: A journey from metabolism to cancer. *Translational Oncology*, *14*(10), 101178.

Debras, C., Chazelas, E., Srour, B., Kesse-Guyot, E., Julia, C., Zelek, L., Agaësse, C., Druesne-Pecollo, N., Galan, P., Hercberg, S., & Latino-Martel, P. (2020). Total and added sugar intakes, sugar types, and cancer risk: Results from the prospective NutriNet-Santé cohort. *The American Journal of Clinical Nutrition*, *112*(5), 1267–1279.

Defo, P. B. D., Wankeu-Nya, M., Ngadjui, E., Fozin, G., Kemka, F., Kamanyi, A., & Watcho, P. (2017). Palm oil diet-induced obesity impairs male rat reproductive performance. *Ann Reprod Med Treat*, *2*(2).

Denke, M. A., & Grundy, S. M. (1992). Comparison of effects of lauric acid and palmitic acid on plasma lipids and lipoproteins. *The American Journal of Clinical Nutrition*, *56*(5), 895–898.

Dennis, K. M., & Heather, L. C. (2023). Post-translational palmitoylation of metabolic proteins. *Frontiers in physiology*, *14*, 1122895.

Deshmukh, P., Unni, S., Krishnappa, G., & Padmanabhan, B. (2017). The Keap1–Nrf2 pathway: Promising therapeutic target to counteract ROS-mediated damage in cancers and neurodegenerative diseases. *Biophysical Reviews*, *9*, 41–56.

Desoye, G., & Herrera, E. (2021). Adipose tissue development and lipid metabolism in the human fetus: The 2020 perspective focusing on maternal diabetes and obesity. *Progress in Lipid Research*, *81*, 101082.

Dhaka, V., Gulia, N., Ahlawat, K. S., & Khatkar, B. S. (2011). Trans fats—Sources, health risks and alternative approach-A review. *Journal of Food Science and Technology*, *48*, 534–541.

Diabetes UK. (2024). *How many people in the UK have diabetes?* Available at: https://www.diabetes.org.uk/about-us/about-the-charity/our-strategy/statistics

DiNicolantonio, J. J., & O'Keefe, J. (2021). The importance of maintaining a low omega-6/omega-3 ratio for reducing the risk of autoimmune diseases, asthma, and allergies. *Missouri Medicine*, *118*(5), 453.

DiNicolantonio, J. J., Lucan, S. C., & O'Keefe, J. H. (2016). The evidence for saturated fat and for sugar related to coronary heart disease. *Progress in Cardiovascular Diseases*, *58*(5), 464–472.

Dinu, M., & Martini, D. (2023). Ultra-processed foods, diet quality and human health. *Nutrients*, *15*(13), 2890.

Dissanayaka, D. S., Jayasena, V., Rainey-Smith, S. R., Martins, R. N., & Fernando, W. B. (2024). The Role of Diet and Gut Microbiota in Alzheimer's Disease. *Nutrients*, *16*(3), 412.

Ditchfield, C., Kushida, M. M., Mazalli, M. R., & Sobral, P. J. (2023a). Can chocolate be classified as an ultra-processed food? A short review on processing and health aspects to help answer this question. *Foods*, *12*(16), 3070.

Djordjevic, J., Ledina, T., Baltic, M. Z., Trbovic, D., Babic, M., & Bulajic, S. (2019). Fatty acid profile of milk. *IOP Conference Series: Earth and Environmental Science*, *333*, 012057.

Djuricic, I., & Calder, P. C. (2021). Beneficial outcomes of omega-6 and omega-3 polyunsaturated fatty acids on human health: An update for 2021. *Nutrients*, *13*(7), 2421.

Dludla, P. V., Mabhida, S. E., Ziqubu, K., Nkambule, B. B., Mazibuko-Mbeje, S. E., Hanser, S., Basson, A. K., Pheiffer, C., & Kengne, A. P. (2023). Pancreatic β-cell dysfunction in type 2 diabetes: Implications of inflammation and oxidative stress. *World Journal of Diabetes*, *14*(3), 130.

Domínguez, R., Pateiro, M., Gagaoua, M., Barba, F. J., Zhang, W., & Lorenzo, J. M. (2019). A comprehensive review on lipid oxidation in meat and meat products. *Antioxidants*, *8*(10), 429.

Donaldson, J., Madziva, M. T., & Erlwanger, K. H. (2016). The effects of high-fat diets composed of different animal and vegetable fat sources on the health status and tissue lipid profiles of male Japanese quail (Coturnix coturnix japonica. *Asian-Australasian Journal of Animal Sciences*, *30*(5), 700.

Dong, Z., Zhuang, Q., Ning, M., Wu, S., Lu, L., & Wan, X. (2020). Palmitic acid stimulates NLRP3 inflammasome activation through TLR4-NF-κB signal pathway in hepatic stellate cells. *Annals of Translational Medicine*, *8*(5), 168.

Dratwa, M., Wysoczańska, B., Łacina, P., Kubik, T., & Bogunia-Kubik, K. (2020). TERT—regulation and roles in cancer formation. *Frontiers in Immunology*, *11*, 589929.

Duan, H., Song, W., Zhao, J., & Yan, W. (2023). Polyunsaturated Fatty Acids (PUFAs): Sources, digestion, absorption, application and their potential adjunctive effects on visual fatigue. *Nutrients*, *15*(11), 2633.

Duarte, A., Martins, J., Lopes, C., Silva, M. J., Augusto, C., Martins, S. P., & Rosário, R. (2024). Health Literacy and Its Association with the Adoption of the Mediterranean Diet: A Cross-Sectional Study. *Nutrients*, *16*(14), 2176.

Dunmore, S. J., & Brown, J. E. (2013). The role of adipokines in b-cell failure of type 2 diabetes. *J Endocrinol*, *216*(1), 37–45.

Dwyer, J. (1995). Overview: Dietary approaches for reducing cardiovascular disease risks. *The Journal of Nutrition*, *125*(suppl_3), 656–665.

Edem, D. O. (2002). Palm oil: Biochemical, physiological, nutritional, hematological and toxicological aspects: A review. *Plant Foods for Human Nutrition*, *57*, 319–341.

Egan, A. M., & Dinneen, S. F. (2014). What is diabetes? *Medicine*, *42*(12), 679–681.

Eguchi, N., Vaziri, N. D., Dafoe, D. C., & Ichii, H. (2021). The role of oxidative stress in pancreatic β cell dysfunction in diabetes. *International Journal of Molecular Sciences*, *22*(4), 1509.

Eklund, C. M. (2009). Proinflammatory cytokines in CRP baseline regulation. *Advances in Clinical Chemistry*, *48*, 111–136.

El Maataoui, A., Jebrane, I., Khlil, N., Naamane, A., & Kamal, N. (2015). Comparison of two biomarkers of inflammation, the erythrocyte sedimentation rate and C-reactive protein measurements: A crosssectional study. *Journal of Chemical and Pharmaceutical Research*, *7*(12), 546–550.

El-Demerdash F. M., Tousson E. M., Kurzepa J., & Habib S. L. (2018). Xenobiotics, oxidative stress, and antioxidants. *Oxidative medicine and cellular longevity*, 9758951.

Elizabeth, L., Machado, P., Zinöcker, M., Baker, P., & Lawrence, M. (2020). Ultra-processed foods and health outcomes: A narrative review. *Nutrients*, *12*(7), 1955.

Engelberts, I., Sundram, K., Houwelingen, A. C., Hornstra, G., Kester, A. D. M., Ceska, M., Francot, G. M., Linden, C. J., & Buurman, W. A. (1993). The effect of replacement of dietary fat by palm oil on in vitro cytokine release. *British Journal of Nutrition*, *69*(1), 159–167.

Enser, M., Hallett, K., Hewitt, B., Fursey, G. A. J., & Wood, J. D. (1996). Fatty acid content and composition of English beef, lamb and pork at retail. *Meat Science*, *42*(4), 443–456.

Estes, C., Razavi, H., Loomba, R., Younossi, Z., & Sanyal, A. J. (2018). Modeling the epidemic of nonalcoholic fatty liver disease demonstrates an exponential increase in burden of disease. *Hepatology*, *67*(1), 123–133.

Euromonitor International. (2024). *Voice of the Consumer: Consumer Health 2024.* Available at: https://www.euromonitor.com/voice-of-the-consumer-consumer-health-2024/report

European Parliament. (2018). *Diet, Nutrition and the Prevention of Chronic Diseases.* Available at: https://www.europarl.europa.eu/RegData/etudes/ATAG/2018/614706/EPRS_ATA(2018)614706_EN.pdf

European Parliament. (2014). *Food Information to Consumers' (FIC) Regulation.* Available at: https://www.legislation.gov.uk/uksi/2014/1855/contents

Fabiszewska, A. U., & Bialecka-Florjanczyk, E. (2014). Factors influencing synthesis of extracellular lipases by Yarrowia lipolytica in medium containing vegetable oils. *The Journal of Microbiology, Biotechnology and Food Sciences*, *4*(3), 231.

Faivre, J., & Giacosa, A. (1998). Primary prevention of colorectal cancer through fibre supplementation. *European Journal of Cancer Prevention*, *7*(2), 29–32.

Falade, A. O., Oboh, G., Ademiluyi, A. O., & Odubanjo, O. V. (2015). Consumption of thermally oxidized palm oil diets alters biochemical indices in rats. *Beni-Suef University Journal of Basic and Applied Sciences*, *4*(2), 150–156.

Fan, A., Wu, X., Wu, H., Li, L., Huang, R., Zhu, Y., Qiu, Y., Fu, J., Ren, J., & Zhu, C. (2014). Atheroprotective effect of oleoylethanolamide (OEA) targeting oxidized LDL. *PloS one*, *9*(1), 85337.

Fan, Y., Li, D., Qian, J., Liu, Y., Feng, H., & Li, D. (2016). Increased expression of FERM domain-containing 4A protein is closely associated with the development of rectal cancer. *Experimental and Therapeutic Medicine*, *11*(2), 421–426.

Fanelli, S. M., Jonnalagadda, S. S., Pisegna, J. L., Kelly, O. J., Krok-Schoen, J. L., & Taylor, C. A. (2020). Poorer diet quality observed among US adults with a greater number of clinical chronic disease risk factors. *Journal of Primary Care & Community Health*, *11*, 2150132720945898.

Fang, W., Liu, Y., Chen, Q., Xu, D., Liu, Q., Cao, X., Hao, T., Zhang, L., Mai, K., & Ai, Q. (2022). Palmitic acid induces intestinal lipid metabolism disorder, endoplasmic reticulum stress and inflammation by affecting phosphatidylethanolamine content in large yellow croaker Larimichthys crocea. *Frontiers in Immunology*, *13*, 984508.

Farmer, B. C., Williams, H. C., Devanney, N. A., Piron, M. A., Nation, G. K., Carter, D. J., Walsh, A. E., Khanal, R., Young, L. E., Kluemper, J. C., & Hernandez, G. (2021). APOE4 lowers energy expenditure in females and impairs glucose oxidation by increasing flux through aerobic glycolysis. *Molecular Neurodegeneration*, *16*(1), 62.

Faruqui, A. (2017). Post Prandial Hyperglycemia: A Real Threat for Patients with Type 2 Diabetes Mellitus. *Advances in Diabetes and Metabolism*, *5*(3), 43–51.

Fatima, G., Dzupina, A., Mahdi, A. A., Fedacko, J., Magomedova, A., & Yousif, N. G. (2025). Role of vitamin-E as a vital nutrient in health and diseases. *Indian Journal of Clinical Biochemistry*, 1–14.

Fatima, S., Hu, X., Gong, R. H., Huang, C., Chen, M., Wong, H. L. X., Bian, Z., & Kwan, H. Y. (2019). Palmitic acid is an intracellular signaling molecule involved in disease development. *Cellular and Molecular Life Sciences*, *76*, 2547–2557.

Fattore, E., & Fanelli, R. (2013). Palm oil and palmitic acid: A review on cardiovascular effects and carcinogenicity. *International Journal of Food Sciences and Nutrition*, *64*(5), 648–659.

Fedacko, J., Takahashi, T., Singh, R. B., Pella, D., Chibisov, S., Hristova, K., Pella, D., Elkilany, G. N., Juneja, L. R., Behl, S., & Singh, J. (2022). Western diets and risk of non-communicable diseases. In *Functional Foods and Nutraceuticals in Metabolic and Non-communicable diseases* (pp. 3–21). Academic Press.

FEDIOL - *The EU Vegetable Oil and Proteinmeal Industry.* (2023). *Vegetable oils production, imports, exports and consumption.* Available at: https://www.fediol.eu/data/oils%25202023.pdf

Feghali, C. A., & Wright, T. M. (1997). Cytokines in acute and chronic inflammation. *Front Biosci*, *2*(1), 12–26.

Fehér, A., Gazdecki, M., Véha, M., Szakály, M., & Szakály, Z. (2020). A Comprehensive Review of the Benefits of and the Barriers to the Switch to a Plant-Based Diet. *Sustainability*, *12*(10).

Feingold, K.R. (2024). *Introduction to lipids and lipoproteins.* Available at: https://www.ncbi.nlm.nih.gov/books/NBK305896/

Ference, B. A., Ginsberg, H. N., Graham, I., Ray, K. K., Packard, C. J., Bruckert, E., Hegele, R. A., Krauss, R. M., Raal, F. J., Schunkert, H., & Watts, G. F. (2017). Low-density lipoproteins cause atherosclerotic cardiovascular disease. 1. Evidence from genetic, epidemiologic, and clinical studies. A consensus statement from the European Atherosclerosis Society Consensus Panel. *European Heart Journal*, *38*(32), 2459–2472.

Filippou, A., Teng, K. T., Berry, S. E., & Sanders, T. A. B. (2014). Palmitic acid in the sn-2 position of dietary triacylglycerols does not affect insulin secretion or glucose homeostasis in healthy men and women. *European Journal of Clinical Nutrition*, *68*(9), 1036–1041.

Filippou, C. D., Tsioufis, C. P., Thomopoulos, C. G., Mihas, C. C., Dimitriadis, K. S., Sotiropoulou, L. I., Chrysochoou, C. A., Nihoyannopoulos, P. I., & Tousoulis, D. M. (2020). Dietary approaches to stop hypertension (DASH) diet and blood pressure reduction in adults with and without hypertension: A systematic review and meta-analysis of randomized controlled trials. *Advances in Nutrition*, *11*(5), 1150–1160.

Fiolet, T., Srour, B., Sellem, L., Kesse-Guyot, E., Allès, B., Méjean, C., Deschasaux, M., Fassier, P., Latino-Martel, P., Beslay, M., Hercberg, S., Lavalette, C., Monteiro, C. A., Julia, C., & Touvier, M. (2018). Consumption of ultra-processed foods and cancer risk: results from NutriNet-Santé prospective cohort. *BMJ (Clinical research ed.)*, *360*, k322.

Flint, A. C., Conell, C., Ren, X., Banki, N. M., Chan, S. L., Rao, V. A., Melles, R. B., & Bhatt, D. L. (2019). Effect of systolic and diastolic blood pressure on cardiovascular outcomes. *New England Journal of Medicine*, *381*(3), 243–251.

Food and Agriculture Organization of the United Nations. (2023). *Vegetable oil production, World.* Available at: https://ourworldindata.org/grapher/vegetable-oil-production

Forsythe, C. E., French, M. A., Goh, Y. K., & Clandinin, M. T. (2007). Cholesterolaemic influence of palmitic acid in the sn-1, 3 v. The sn-2 position with high or low dietary linoleic acid in healthy young men. *British Journal of Nutrition*, *98*(2), 337–344.

Frankenberg, A. D., Marina, A., Song, X., Callahan, H. S., Kratz, M., & Utzschneider, K. M. (2017). A high-fat, high-saturated fat diet decreases insulin sensitivity without changing intra-abdominal fat in weight-stable overweight and obese adults. *European Journal of Nutrition*, *56*, 431–443.

Frazão, J. B., Thain, A., Zhu, Z., Luengo, M., Condino-Neto, A., & Newburger, P. E. (2015). Regulation of CYBB gene expression in human phagocytes by a distant upstream NF-κB binding site. *Journal of Cellular Biochemistry*, *116*(9), 2008–2017.

French, M. A., Sundram, K., & Clandinin, M. T. (2002). Cholesterolaemic effect of palmitic acid in relation to other dietary fatty acids. *Asia Pacific Journal of Clinical Nutrition*, *11*, 401–407.

Frolova, L., Drastich, P., Rossmann, P., Klimesova, K., & Tlaskalova-Hogenova, H. (2008). Expression of Toll-like receptor 2 (TLR2), TLR4, and CD14 in biopsy samples of patients with inflammatory bowel diseases: Upregulated expression of TLR2 in terminal ileum of

patients with ulcerative colitis. *Journal of Histochemistry & Cytochemistry*, *56*(3), 267–274.

Fryzova, R., Pohanka, M., Martinkova, P., Cihlarova, H., Brtnicky, M., Hladky, J., & Kynicky, J. (2018). Oxidative stress and heavy metals in plants. *Reviews of Environmental Contamination and Toxicology*, *245*, 129–156.

Fuchs, F. D., & Whelton, P. K. (2020). High blood pressure and cardiovascular disease. *Hypertension*, *75*(2), 285–292.

Fujimoto, W. Y. (2000). The importance of insulin resistance in the pathogenesis of type 2 diabetes mellitus. *The American Journal of Medicine*, *108*(6), 9–14.

Gaeini, Z., Bahadoran, Z., & Mirmiran, P. (2022). Saturated fatty acid intake and risk of type 2 diabetes: An updated systematic review and dose–response Meta-analysis of Cohort studies. *Advances in Nutrition*, *13*(6), 2125–2135.

Ganafa, A. A., Socci, R. R., Eatman, D., Silvestrov, N., Abukhalaf, I. K., & Bayorh, M. A. (2002). Effect of palm oil on oxidative stress-induced hypertension in Sprague-Dawley rats. *American Journal of Hypertension*, *15*(8), 725–731.

Gao, D., Griffiths, H. R., & Bailey, C. J. (2009). Oleate protects against palmitate-induced insulin resistance in L6 myotubes. *British Journal of Nutrition*, *102*(11), 1557–1563.

García-Montero, C., Fraile-Martínez, O., Gómez-Lahoz, A. M., Pekarek, L., Castellanos, A. J., Noguerales-Fraguas, F., Coca, S., Guijarro, L. G., García-Honduvilla, N., Asúnsolo, A., & Sanchez-Trujillo, L. (2021). Nutritional components in Western diet versus Mediterranean diet at the gut microbiota–immune system interplay. *Implications for Health and Disease. Nutrients*, *13*(2), 699.

García, L. C., & Hernández, A. N. M. (2020). Beneficial effects of cocoa and dark chocolate polyphenols on health. *The FASEB Journal*, *34*(S1), 1–1.

Gastaldello, A., Giampieri, F., Giuseppe, R., Grosso, G., Baroni, L., & Battino, M. (2022). The rise of processed meat alternatives: A narrative review of the manufacturing, composition, nutritional profile and health effects of newer sources of protein, and their place in healthier diets. *Trends in Food Science & Technology*.

Gatti, R. C., Liang, J., Velichevskaya, A., & Zhou, M. (2019). Sustainable palm oil may not be so sustainable. *Science of the Total Environment*, *652*, 48–51.

Gehring, J., Touvier, M., Baudry, J., Julia, C., Buscail, C., Srour, B., Hercberg, S., Péneau, S., Kesse-Guyot, E., & Allès, B. (2021). Consumption of ultra-processed foods by pesco-vegetarians, vegetarians, and vegans: Associations with duration and age at diet initiation. *The Journal of Nutrition*, *151*(1), 120–131.

Geng, L., Liu, K., & Zhang, H. (2023). Lipid oxidation in foods and its implications on proteins. *Frontiers in Nutrition*, *10*, 1192199.

German, J. B. (2011). Dietary lipids from an evolutionary perspective: Sources, structures and functions. *Maternal & Child Nutrition*, *7*, 2–16.

German, J. B., & Dillard, C. J. (2004). Saturated fats: What dietary intake? *The American Journal of Clinical Nutrition*, *80*(3), 550–559.

Ghafoorunissa, R., V., & Sesikaran, B. (1995). Palmolein and groundnut oil have comparable effects on blood lipids and platelet aggregation in healthy Indian subjects. *Lipids*, *30*(12), 1163–1169.

Gharby, S. (2022). Refining vegetable oils: Chemical and physical refining. *The Scientific World Journal*, *2022*(1), 6627013.

Ghezzal, S., Postal, B. G., Quevrain, E., Brot, L., Seksik, P., Leturque, A., Thenet, S., & Carriere, V. (2020). Palmitic acid damages gut epithelium integrity and initiates inflammatory cytokine production. *Biochimica et Biophysica Acta (BBA)-Molecular and Cell Biology of Lipids*, *1865*(2), 158530.

Giacomin, V. (2018). The transformation of the global palm oil cluster: Dynamics of cluster competition between Africa and Southeast Asia (c. 1900–1970. *Journal of Global History*, *13*(3), 374–398.

Gibney, M. J., Forde, C. G., Mullally, D., & Gibney, E. R. (2017). Ultra-processed foods in human health: A critical appraisal. *The American Journal of Clinical Nutrition*, *106*(3), 717–724.

Gifford, K. D. (2002). Dietary fats, eating guides, and public policy: History, critique, and recommendations. *The American Journal of Medicine*, *113*(9), 89–106.

Gilbert, J. A., Blaser, M. J., Caporaso, J. G., Jansson, J. K., Lynch, S. V., & Knight, R. (2018). Current understanding of the human microbiome. *Nature Medicine*, *24*(4), 392–400.

Gillingham, L. G., Harris-Janz, S., & Jones, P. J. (2011). Dietary monounsaturated fatty acids are protective against metabolic syndrome and cardiovascular disease risk factors. *Lipids*, *46*, 209–228.

Giloteaux, L., Goodrich, J. K., Walters, W. A., Levine, S. M., Ley, R. E., & Hanson, M. R. (2016). Reduced diversity and altered composition of the gut microbiome in individuals with myalgic encephalomyelitis/chronic fatigue syndrome. *Microbiome*, *4*, 1–12.

Ginsberg, H. N., & MacCallum, P. R. (2009). The obesity, metabolic syndrome, and type 2 diabetes mellitus pandemic: Part I. Increased cardiovascular disease risk and the importance of atherogenic dyslipidemia in persons with the metabolic syndrome and type 2 diabetes mellitus. *Journal of the Cardiometabolic Syndrome*, *4*(2), 113–119.

Giuffrida, F., Marmet, C., Tavazzi, I., Fontannaz, P., Sauser, J., Lee, L. Y., & Destaillats, F. (2018). Quantification of 1, 3-olein-2-palmitin (OPO) and palmitic acid in sn-2 position of triacylglycerols in human milk by liquid chromatography coupled with mass spectrometry. *Molecules*, *24*(1), 22.

Global survey of the omega-3 fatty acids, docosahexaenoic acid and eicosapentaenoic acid in the blood stream of healthy adults. (2016). *Progress in Lipid Research*, *63*, 132–152.

Golay, A., & Bobbioni, E. (1997). The role of dietary fat in obesity. *International Journal of Obesity and Related Metabolic Disorders: Journal of the International Association for the Study of Obesity*, *21*, 2–11.

González-Monroy, C., Gómez-Gómez, I., Olarte-Sánchez, C. M., & Motrico, E. (2021). Eating behaviour changes during the COVID-19 pandemic: A systematic review of longitudinal studies. *International Journal of Environmental Research and Public Health*, *18*(21), 11130.

Goodpaster, B. H., & Kelley, D. E. (1998). Role of muscle in triglyceride metabolism. *Current Opinion in Lipidology*, *9*(3), 231–236.

Gordon, M. (1993). Fats and fatty foods. *Food industries manual*, 288–327.

Gori, M., Altomare, A., Cocca, S., Solida, E., Ribolsi, M., Carotti, S., Rainer, A., Francesconi, M., Morini, S., Cicala, M., & Pier Luca Guarino, M. (2020). Palmitic acid affects intestinal epithelial barrier integrity and permeability in vitro. *Antioxidants*, *9*(5), 417.

Gosmanova, E. O., Mikkelsen, M. K., Molnar, M. Z., Lu, J. L., Yessayan, L. T., Kalantar-Zadeh, K., & Kovesdy, C. P. (2016). Association of systolic blood pressure variability with mortality, coronary heart disease, stroke, and renal disease. *Journal of the American College of Cardiology*, *68*(13), 1375–1386.

Gowd, V., Bao, T., Wang, L., Huang, Y., Chen, S., Zheng, X., Cui, S., & Chen, W. (2018). Antioxidant and antidiabetic activity of blackberry after gastrointestinal digestion and human gut microbiota fermentation. *Food Chemistry*, *269*, 618–627.

Gowthaman, R., Gowthaman, N., Rajangam, M. K., & Srinivasan, K. (2007). Database of neurodegenerative disorders. *Bioinformation*, *2*(4), 153.

Goyal, R., & Deshmukh, N. (2018). Food label reading: Read before you eat. *Journal of Education and Health Promotion*, *7*(1), 56.

Grassian, D. T. (2020). The dietary behaviors of participants in UK-based meat reduction and vegan campaigns–A longitudinal, mixed-methods study. *Appetite*, *154*, 104788.

Greten, F. R., & Grivennikov, S. I. (2019). Inflammation and cancer: Triggers, mechanisms, and consequences. *Immunity*, *51*(1), 27–41.

Grimmig, T., Matthes, N., Hoeland, K., Tripathi, S., Chandraker, A., Grimm, M., Moench, R., Moll, E. M., Friess, H., Tsaur, I., & Blaheta, R. A. (2015). TLR7 and TLR8 expression increases tumor cell proliferation and promotes chemoresistance in human pancreatic cancer. *International Journal of Oncology*, *47*(3), 857–866.

Gruffat, D., Bauchart, D., Thomas, A., Parafita, E., & Durand, D. (2021). Fatty acid composition and oxidation in beef muscles as affected by ageing times and cooking methods. *Food Chemistry*, *343*, 128476.

Guasch-Ferré, M., Babio, N., Martínez-González, M. A., Corella, D., Ros, E., Martin-Pelaez, S., Estruch, R., Arós, F., Gómez-Gracia, E., Fiol, M., & Santos-Lozano, J. M. (2015). Dietary fat intake and risk of cardiovascular disease and all-cause mortality in a population at high risk of cardiovascular disease. *The American Journal of Clinical Nutrition*, *102*(6), 1563–1573.

Gunstone, F. D., Harwood, J. L., & Dijkstra, A. J. (2007). *The Lipid handbook with CD-ROM* (No. 18073). Available at: https://www.google.co.uk/books/edition/_/INZa6WmqDA8C?hl=en&gbpv=0

Guo, Q., Jin, Y., Chen, X., Ye, X., Shen, X., Lin, M., Zeng, C., Zhou, T., & Zhang, J. (2024). NF-κB in biology and targeted therapy: New insights and translational implications. *Signal Transduction and Targeted Therapy*, *9*(1), 53.

Gupta, I., & Gaykalova, D. A. (2024). Unveiling the role of PIK3R1 in cancer: A comprehensive review of regulatory signaling and therapeutic implications. In *Seminars in Cancer Biology* (Vol. 106, pp. 58–86).

Gupta, R., Lakshmy, R., Abraham, R. A., Reddy, K. S., Jeemon, P., & Prabhakaran, D. (2013). Serum omega-6/omega-3 ratio and risk markers for cardiovascular disease in an industrial population of Delhi. *Food and Nutrition Sciences (Print*, *4*(9A), 94.

Gupta, V., Singh, A. K., Gupta, V., Kumar, S., Srivastava, N., Jafar, T., & Pant, A. B. (2011). Association of circulating resistin with metabolic risk factors in Indian females having metabolic syndrome. *Toxicology International*, *18*(2), 168.

Gusev, E., & Zhuravleva, Y. (2022). Inflammation: A new look at an old problem. *International Journal of Molecular Sciences*, *23*(9), 4596.

Hacker, K. (2024). The burden of chronic disease. *Mayo Clinic Proceedings: Innovations, Quality & Outcomes*, *8*(1), 112–119.

Han, L., Sheng, B., Zeng, Q., Yao, W., & Jiang, Q. (2020). Correlation between MMP2 expression in lung cancer tissues and clinical parameters: A retrospective clinical analysis. *BMC Pulmonary Medicine*, *20*, 1–9.

Hannoodee, S., & Nasuruddin, D. N. (2024). Acute inflammatory response. In *StatPearls [Internet*. StatPearls Publishing.

Hariharan, D., Vellanki, K., & Kramer, H. (2015). The Western diet and chronic kidney disease. *Current Hypertension Reports*, *17*, 1–9.

Haritha, K., Kalyani, L., & Rao, A. L. (2014). Health benefits of dark chocolate. *Journal of Advanced Drug Delivery*, *1*(4), 184–195.

Harrison, S., Couture, P., & Lamarche, B. (2020). Diet quality, saturated fat and metabolic syndrome. *Nutrients*, *12*(11), 3232.

Hart, T. L., Damani, J. J., DiMattia, Z. S., Tate, K. E., Jafari, F., & Petersen, K. S. (2025). Dietary polyunsaturated to saturated fatty acid ratio as an indicator for LDL cholesterol response: a systematic review and Meta-Analysis of randomized clinical trials. *Advances in Nutrition*, *16*(10), 100502.

Hasan, A., Akhter, N., Al-Roub, A., Thomas, R., Kochumon, S., Wilson, A., Koshy, M., Al-Ozairi, E., Al-Mulla, F., & Ahmad, R. (2019). TNF-α in combination with palmitate enhances IL-8 production via the MyD88-independent TLR4 signaling pathway: Potential relevance to metabolic inflammation. *International Journal of Molecular Sciences*, *20*(17), 4112.

Hasan, N., & Yang, H. (2019). Factors affecting the composition of the gut microbiota, and its modulation. *PeerJ*, *7*, 7502.

Hassim, N. A. M., & Dian, L. N. H. M. (2017). Usage of palm oil, palm kernel oil and their fractions as confectionery fats. *Journal of Oil Palm Research*, *29*(3), 301–310.

Haththotuwa, R. N., Wijeyaratne, C. N., & Senarath, U. (2020). Worldwide epidemic of obesity. In *Obesity and obstetrics* (pp. 3–8). Elsevier.

Hausmann, M., Kiessling, S., Mestermann, S., Webb, G., Spöttl, T., Andus, T., Schölmerich, J., Herfarth, H., Ray, K., Falk, W., & Rogler, G. (2002). Toll-like receptors 2 and 4 are up-regulated during intestinal inflammation. *Gastroenterology*, *122*(7), 1987–2000.

Havlicekova, Z., Jesenak, M., Banovcin, P., & Kuchta, M. (2015). Beta-palmitate–a natural component of human milk in supplemental milk formulas. *Nutrition Journal*, *15*, 1–8.

Hawkes, C. (2010). The influence of trade liberalization and global dietary change: The case of vegetable oils, meat and highly processed foods. *Trade, Food, Diet and Health: Perspectives and Policy Options*, *35*, 59.

Hayashi, T., Fujita, K., Nojima, S., Hayashi, Y., Nakano, K., Ishizuya, Y., Wang, C., Yamamoto, Y., Kinouchi, T., Matsuzaki, K., & Jingushi, K. (2018). High-fat diet-induced

inflammation accelerates prostate cancer growth via IL6 signaling. *Clinical Cancer Research*, *24*(17), 4309–4318.

Heck, R. T., Saldaña, E., Lorenzo, J. M., Correa, L. P., Fagundes, M. B., Cichoski, A. J., Menezes, C. R., Wagner, R., & Campagnol, P. C. B. (2019). Hydrogelled emulsion from chia and linseed oils: A promising strategy to produce low-fat burgers with a healthier lipid profile. *Meat Science*, *156*, 174–182.

Heck, R. T., Vendruscolo, R. G., Araújo Etchepare, M., Cichoski, A. J., Menezes, C. R., Barin, J. S., Lorenzo, J. M., Wagner, R., & Campagnol, P. C. B. (2017). Is it possible to produce a low-fat burger with a healthy n− 6/n− 3 PUFA ratio without affecting the technological and sensory properties? *Meat Science*, *130*, 16–25.

Heileson, J. L. (2020). Dietary saturated fat and heart disease: A narrative review. *Nutrition Reviews*, *78*(6), 474–485.

Henein, M. Y., Vancheri, S., Longo, G., & Vancheri, F. (2022). The role of inflammation in cardiovascular disease. *International Journal of Molecular Sciences*, *23*(21), 12906.

Herrera, E., & Ortega-Senovilla, H. (2010). Maternal lipid metabolism during normal pregnancy and its implications to fetal development. *Clinical Lipidology*, *5*(6), 899–911.

Hinz, M., & Scheidereit, C. (2014). The IκB kinase complex in NF-κB regulation and beyond. *EMBO Reports*, *15*(1), 46–61.

Hivert, M. F., Manning, A. K., McAteer, J. B., Florez, J. C., Dupuis, J., Fox, C. S., O'Donnell, C. J., Cupples, L. A., & Meigs, J. B. (2008). Common variants in the adiponectin gene (ADIPOQ) associated with plasma adiponectin levels, type 2 diabetes, and diabetes-related quantitative traits: The Framingham Offspring Study. *Diabetes*, *57*(12), 3353–3359.

Hoenig, J. M., & Heisey, D. M. (2001). The abuse of power: the pervasive fallacy of power calculations for data analysis. *The American Statistician, 55*(1), 19-24.

Hooijmans, C. R., Rovers, M. M., Vries, R. B., Leenaars, M., Ritskes-Hoitinga, M., & Langendam, M. W. (2014). SYRCLE's risk of bias tool for animal studies. *BMC Medical Research Methodology*, *14*(1), 43.

Hooper, L., Martin, N., Jimoh, O. F., Kirk, C., Foster, E., & Abdelhamid, A. S. (2020). *Reduction in saturated fat intake for cardiovascular disease. Cochrane database of systematic reviews*, (8).

Hossain, M. M., Tasnim, S., Sultana, A., Faizah, F., Mazumder, H., Zou, L., & Ma, P. (2020). Epidemiology of mental health problems in COVID-19: A review. *F1000Research*, *9*, 636.

Hou, Y., Dan, X., Babbar, M., Wei, Y., Hasselbalch, S. G., Croteau, D. L., & Bohr, V. A. (2019). Ageing as a risk factor for neurodegenerative disease. *Nature Reviews Neurology*, *15*(10), 565–581.

Hove, I., Lefevere, E., Groef, L., Sergeys, J., Salinas-Navarro, M., Libert, C., Vandenbroucke, R., & Moons, L. (2016). MMP-3 deficiency alleviates endotoxin-induced acute inflammation in the posterior eye segment. *International Journal of Molecular Sciences*, *17*(11), 1825.

Hu, F. B. (2002). Dietary pattern analysis: A new direction in nutritional epidemiology. *Current Opinion in Lipidology*, *13*(1), 3–9.

Huang, Q., Li, S., Cheng, P., Deng, M., He, X., Wang, Z., Yang, C. H., Zhao, X. Y., & Huang, J. (2017). High expression of anti-apoptotic protein Bcl-2 is a good prognostic factor in colorectal cancer: Result of a meta-analysis. *World Journal of Gastroenterology*, *23*(27), 5018.

Huang, S., Rutkowsky, J. M., Snodgrass, R. G., Ono-Moore, K. D., Schneider, D. A., Newman, J. W., Adams, S. H., & Hwang, D. H. (2012). Saturated fatty acids activate TLR-mediated proinflammatory signaling pathways [S. *Journal of Lipid Research*, *53*(9), 2002–2013.

Huang, X., & Ahn, D. U. (2019). Lipid oxidation and its implications to meat quality and human health. *Food Science and Biotechnology*, *28*(5), 1275–1285.

Huebbe, P., & Rimbach, G. (2020). Historical reflection of food processing and the role of legumes as part of a healthy balanced diet. *Foods*, *9*(8), 1056.

Hugon, P., Lagier, J. C., Colson, P., Bittar, F., & Raoult, D. (2017). Repertoire of human gut microbes. *Microbial Pathogenesis*, *106*, 103–112.

Human insulin receptor substrate-1 gene (IRS1): Chromosomal localization to 2q35-q36. 1 and identification of a simple tandem repeat DNA polymorphism. (1993). *Diabetologia*, *36*(4), 335–337.

Hunter, J. E., Zhang, J., & Kris-Etherton, P. M. (2010). Cardiovascular disease risk of dietary stearic acid compared with trans, other saturated, and unsaturated fatty acids: A systematic review. *The American Journal of Clinical Nutrition*, *91*(1), 46–63.

Iacob, S., & Iacob, D. G. (2019). Infectious threats, the intestinal barrier, and its trojan horse: Dysbiosis. *Frontiers in Microbiology*, *10*, 448283.

Ibrahim, N. I., Fairus, S., & Mohamed, I. N. (2020). The Effects and Potential Mechanism of Oil Palm Phenolics in Cardiovascular Health: A Review on Current Evidence. *Nutrients*, *12*(7), 2055.

Idris, C. A. C., Karupaiah, T., Sundram, K., Tan, Y. A., Balasundram, N., Leow, S. S., Nasruddin, N. S., & Sambanthamurthi, R. (2014). Oil palm phenolics and vitamin E reduce atherosclerosis in rabbits. *Journal of Functional Foods*, *7*, 541–550.

Iggman, D., Rosqvist, F., Larsson, A., Ärnlöv, J., Beckman, L., Rudling, M., & Risérus, U. (2014). Role of dietary fats in modulating cardiometabolic risk during moderate weight gain: A randomized double-blind overfeeding trial (LIPOGAIN study. *Journal of the American Heart Association*, *3*(5), 001095.

Ikenouchi, J., & Umeda, M. (2010). FRMD4A regulates epithelial polarity by connecting Arf6 activation with the PAR complex. *Proceedings of the National Academy of Sciences*, *107*(2), 748–753.

Iko Afé, O. H., Kpoclou, Y. E., Douny, C., Anihouvi, V. B., Igout, A., Mahillon, J., & Scippo, M. L. (2021). Chemical hazards in smoked meat and fish. *Food Science & Nutrition*, *9*(12).

Im, Y. R., Hunter, H., Gracia Hahn, D., Duret, A., Cheah, Q., Dong, J., Fairey, M., Hjalmarsson, C., Li, A., Lim, H. K., & McKeown, L. (2021). A systematic review of animal models of NAFLD finds high-fat, high-fructose diets most closely resemble human NAFLD. *Hepatology*, *74*(4), 1884–1901.

Innih, C., O'Kelly, D., Douglas, F., Arenhart, C., O'Brien, K., & Kirwan, L. B. (2024). Nutritics GB23 database: An enhancement of the McCance Widdowson's The Composition of Foods Integrated Dataset (CoFID) database. *Proceedings of the Nutrition Society*, *83*(OCE4), 340.

Innis, S. M. (1991). Essential fatty acids in growth and development. *Progress in Lipid Research*, *30*(1), 39–103.

Innis, S. M. (2011). Dietary triacylglycerol structure and its role in infant nutrition. *Advances in Nutrition*, *2*(3), 275–283.

Innis, S. M. (2016). Palmitic acid in early human development. *Critical Reviews in Food Science and Nutrition*, *56*(12), 1952–1959.

Innis, S. M., Dyer, R., Nelson, C. M., S.L., & Harris, R. S. (1994). Evidence that palmitic acid is absorbed assn-2 monoacylglycerol from human milk by breast-fed infants. *Lipids*, *29*(8), 541-545.

Inoue, J. I., Gohda, J., Akiyama, T., & Semba, K. (2007). NF-κB activation in development and progression of cancer. *Cancer Science*, *98*(3), 268–274.

Iqbal, J., & Hussain, M. M. (2009). Intestinal lipid absorption. *American Journal of Physiology-Endocrinology and Metabolism*, *296*(6), 1183–1194.

Ishii, M., Maeda, A., Tani, S., & Akagawa, M. (2015). Palmitate induces insulin resistance in human HepG2 hepatocytes by enhancing ubiquitination and proteasomal degradation of key insulin signaling molecules. *Archives of Biochemistry and Biophysics*, *566*, 26–35.

Ismail, R. (2005). Palm oil and palm olein frying applications. *Asia Pacific Journal of Clinical Nutrition*, *14*(4), 414.

Ismail, S. R., Maarof, S. K., Siedar Ali, S., & Ali, A. (2018). Systematic review of palm oil consumption and the risk of cardiovascular disease. *PLoS One*, *13*(2), 0193533.

Isolauri, E. (2012). Development of healthy gut microbiota early in life. *Journal of Paediatrics and Child Health*, *48*, 1–6.

Israël, A. (2010). The IKK complex, a central regulator of NF-κB activation. *Cold Spring Harbor Perspectives in Biology*, *2*(3), 000158.

Iyer, S. S., & Cheng, G. (2012). Role of interleukin 10 transcriptional regulation in inflammation and autoimmune disease. *Critical ReviewsTM in Immunology*, *32*(1).

Jacobs, K. M., Bhave, S. R., Ferraro, D. J., Jaboin, J. J., Hallahan, D. E., & Thotala, D. (2012). GSK-3β: A bifunctional role in cell death pathways. *International Journal of Cell Biology*, *2012*(1), 930710.

Jafri, Z., Li, Y., Zhang, J., O'Meara, C. H., & Khachigian, L. M. (2025). Jun, an oncological foe or friend? *International Journal of Molecular Sciences*, *26*(2), 555.

Jayawardena, R., Swarnamali, H., Ranasinghe, P., & Misra, A. (2021). Health effects of coconut oil: Summary of evidence from systematic reviews and meta-analysis of interventional studies. *Diabetes & Metabolic Syndrome: Clinical Research & Reviews*, *15*(2), 549–555.

Jena, P. K., Sheng, L., Nguyen, M., Lucente, J., Hu, Y., Li, Y., Maezawa, I., Jin, L. W., & Wan, Y. J. Y. (2020). Dysregulated bile acid receptor-mediated signaling and IL-17A induction are implicated in diet-associated hepatic health and cognitive function. *Biomarker Research*, *8*(1), 59.

Jensen, M. D., Ryan, D. H., Apovian, C. M., Ard, J. D., Comuzzie, A. G., Donato, K. A., Hu, F. B., Hubbard, V. S., Jakicic, J. M., Kushner, R. F., & Loria, C. M. (2014). AHA/ACC/TOS guideline for the management of overweight and obesity in adults: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society. *Circulation*, *129*(25_suppl_2), 102–138.

Jomova, K., Raptova, R., Alomar, S. Y., Alwasel, S. H., Nepovimova, E., Kuca, K., & Valko, M. (2023). Reactive oxygen species, toxicity, oxidative stress, and antioxidants: Chronic diseases and aging. *Archives of Toxicology*, *97*(10), 2499–2574.

Jones, P., Shears, P., Hillier, D., & Clarke-Hill, C. (2002). Customer perceptions of services brands: A case study of the three major fast food retailers in the UK. *Management Research News*, *25*(6/7), 41–49.

Joshi-Barve, S., Barve, S. S., Amancherla, K., Gobejishvili, L., Hill, D., Cave, M., Hote, P., & McClain, C. J. (2007). Palmitic acid induces production of proinflammatory cytokine interleukin-8 from hepatocytes. *Hepatology*, *46*(3), 823–830.

Jóźwiak, J. J. (2023). Global, regional, and national burden of diabetes from 1990 to 2021, with projections of prevalence to 2050: A systematic analysis for the Global Burden of Disease Study 2021. *Lancet*, *402*(10397).

Jump, D. B., & Clarke, S. D. (1999). Regulation of gene expression by dietary fat. *Annual Review of Nutrition*, *19*(1), 63–90.

Just, S., Mondot, S., Ecker, J., Wegner, K., Rath, E., Gau, L., Streidl, T., Hery-Arnaud, G., Schmidt, S., Lesker, T. R., & Bieth, V. (2018). The gut microbiota drives the impact of bile acids and fat source in diet on mouse metabolism. *Microbiome*, *6*(1), 134.

Juul, F., Vaidean, G., Lin, Y., Deierlein, A. L., & Parekh, N. (2021). Ultra-processed foods and incident cardiovascular disease in the Framingham Offspring Study. *Journal of the American College of Cardiology*, *77*(12), 1520–1531.

K.W., & Kang, Y. (2011). Involvement of the TLR4 (Toll-like receptor4) signaling pathway in palmitate-induced INS-1 beta cell death. *Molecular and Cellular Biochemistry*, *354*, 207–217.

Kahleova, H., Levin, S., & Barnard, N. (2017). Cardio-metabolic benefits of plant-based diets. *Nutrients*, *9*(8), 848.

Kanagaratnam, S., Mat Sahri, M., Idris, N. A., Tangavelu, T., & Ahmad, M. J. (1995). Palm-based trans-free roll-in margarine. *Palm Oil Devel*, *48*, 7–12.

Kang, G. G., Trevaskis, N. L., Murphy, A. J., & Febbraio, M. A. (2023). Diet-induced gut dysbiosis and inflammation: key drivers of obesity-driven NASH. *Iscience*, *26*(1).

Kany, S., Vollrath, J. T., & Relja, B. (2019). Cytokines in inflammatory disease. *International journal of molecular sciences*, *20*(23), 6008.

Kappally, S., Shirwaikar, A., & Shirwaikar, A. (2015). Coconut oil–a review of potential applications. *Hygeia JD Med*, *7*(2), 34–41.

Karacor, K., & Cam, M. (2015). Effects of oleic acid. *Medical Science and Discovery*, *2*(1), 125–132.

Karjalainen, M. K., Holmes, M. V., Wang, Q., Anufrieva, O., Kähönen, M., Lehtimäki, T., Havulinna, A. S., Kristiansson, K., Salomaa, V., Perola, M., & Viikari, J. S. (2020). Apolipoprotein AI concentrations and risk of coronary artery disease: A Mendelian randomization study. *Atherosclerosis*, *299*, 56–63.

Karupaiah, T., & Sundram, K. (2007). Effects of stereospecific positioning of fatty acids in triacylglycerol structures in native and randomized fats: A review of their nutritional implications. *Nutrition & Metabolism*, *4*(1), 16.

Karupaiah, T., Tan, C. H., Chinna, K., & Sundram, K. (2011). The chain length of dietary saturated fatty acids affects human postprandial lipemia. *Journal of the American College of Nutrition*, *30*(6), 511–521.

Kaspar, J. W., Niture, S. K., & Jaiswal, A. K. (2009). Nrf2: INrf2 (Keap1) signaling in oxidative stress. *Free Radical Biology and Medicine*, *47*(9), 1304–1309.

Kassi, E., Pervanidou, P., Kaltsas, G., & Chrousos, G. (2011). Metabolic syndrome: Definitions and controversies. *BMC Medicine*, *9*, 1–13.

Kato, I., & Sun, J. (2023). Microbiome and diet in colon cancer development and treatment. *The Cancer Journal*, *29*(2), 89–97.

Kattoor, A. J., Pothineni, N. V. K., Palagiri, D., & Mehta, J. L. (2017). Oxidative stress in atherosclerosis. *Current Atherosclerosis Reports*, *19*, 1–11.

Kaviani, S., Taylor, C. M., Stevenson, J. L., Cooper, J. A., & Paton, C. M. (2019). A 7-day high-PUFA diet reduces angiopoietin-like protein 3 and 8 responses and postprandial triglyceride levels in healthy females but not males: A randomized control trial. *BMC Nutrition*, *5*, 1–11.

Kawano, Y., & Cohen, D. E. (2013). Mechanisms of hepatic triglyceride accumulation in non-alcoholic fatty liver disease. *Journal of Gastroenterology*, *48*(4), 434–441.

Kelly, F. D., Sinclair, A. J., Mann, N. J., Turner, A. H., Raffin, F. L., Blandford, M. V., & Pike, M. J. (2002). Short-term diets enriched in stearic or palmitic acids do not alter plasma lipids, platelet aggregation or platelet activation status. *European Journal of Clinical Nutrition*, *56*(6), 490–499.

Kelly, R. K., Pollard, Z., Young, H., Piernas, C., Lentjes, M., Mulligan, A., Huybrechts, I., Carter, J. L., Key, T. J., & Perez-Cornago, A. (2022). Evaluation of the New Individual Fatty Acid Dataset for UK Biobank: Analysis of Intakes and Sources in 207,997 Participants. *Nutrients*, *14*(17), 3603.

Kenneth, R. (2024). *The effect of diet on cardiovascular disease and lipid and lipoprotein levels.* Available at: https://www.ncbi.nlm.nih.gov/sites/books/NBK570127/

Késmárky, G., Kenyeres, P., Rábai, M., & Tóth, K. (2008). Plasma viscosity: A forgotten variable. *Clinical Hemorheology and Microcirculation*, *39*(1–4), 243–246.

Key, T. J., Papier, K., & Tong, T. Y. (2022). Plant-based diets and long-term health: Findings from the EPIC-Oxford study. *Proceedings of the Nutrition Society*, *81*(2), 190–198.

Khan, M. A., Khan, Z. A., Shoeb, F., Fatima, G., Khan, R. H., & Khan, M. M. (2023). Role of de novo lipogenesis in inflammation and insulin resistance in Alzheimer's disease. *International Journal of Biological Macromolecules*, *p.124859*.

Khanapure, S. P., Garvey, D. S., Janero, D. R., & Gordon Letts, L. (2007). Eicosanoids in inflammation: Biosynthesis, pharmacology, and therapeutic frontiers. *Current Topics in Medicinal Chemistry*, *7*(3), 311–340.

Khehra, R., Fairchild, R. M., & Morgan, M. Z. (2018). UK children's breakfast cereals–an oral health perspective. *British Dental Journal*, *225*(2), 164–169.

Kiela, P. R., & Ghishan, F. K. (2016). Physiology of intestinal absorption and secretion. *Best Practice & Research Clinical Gastroenterology*, *30*(2), 145–159.

Kifli, H. A. (1981). *Studies on palm oil with special reference to interestterification* (Doctoral dissertation, University of St Andrews).

Kim, H., Caulfield, L. E., Garcia-Larsen, V., Steffen, L. M., Coresh, J., & Rebholz, C. M. (2019). Plant-based diets are associated with a lower risk of incident cardiovascular

disease, cardiovascular disease mortality, and all-cause mortality in a general population of middle-aged adults. *Journal of the American Heart Association*, *8*(16), 012865.

Kim, J. W., Hong, K. J., Chung, B. S., & Hur, J. W. (1997). Characteristics of mayonnaise prepared with palm oil. *Korean Journal of Food Science and Technology*, *29*(2), 261–265.

Klinger, J., Berens, E. M., & Schaeffer, D. (2023). Health literacy and the role of social support in different age groups: Results of a German cross-sectional survey. *BMC Public Health*, *23*(1), 2259.

Kochikuzhyil, B. M., Devi, K., & Fattepur, S. R. (2010). Effect of saturated fatty acid-rich dietary vegetable oils on lipid profile, antioxidant enzymes and glucose tolerance in diabetic rats. *Indian Journal of Pharmacology*, *42*(3), 142–145.

Kong, D. H., Kim, Y. K., Kim, M. R., Jang, J. H., & Lee, S. (2018). Emerging roles of vascular cell adhesion molecule-1 (VCAM-1) in immunological disorders and cancer. *International Journal of Molecular Sciences*, *19*(4), 1057.

Korbecki, J., & Bajdak-Rusinek, K. (2019). The effect of palmitic acid on inflammatory response in macrophages: An overview of molecular mechanisms. *Inflammation Research*, *68*, 915–932.

Kovilakath, A., Wohlford, G., & Cowart, L. A. (2023). Circulating sphingolipids in heart failure. *Frontiers in Cardiovascular Medicine*, *10*, 1154447.

Kozlov, A. V., Javadov, S., & Sommer, N. (2024). Cellular ROS and Antioxidants: Physiological and Pathological Role. *Antioxidants*, *13*(5), 602.

Kozłowska, A., & Szostak-Węgierek, D. (2022). Targeting cardiovascular diseases by flavonols: An update. *Nutrients*, *14*(7), 1439.

Kraus, S., Heilmayr, R., & Koch, N. (2024). Spillovers to Manufacturing Plants from Multimillion Dollar Plantations: Evidence from the Indonesian Palm Oil Boom. *Journal of the Association of Environmental and Resource Economists*, *11*(3), 613–656.

Kris-Etherton, P. M., Taylor, D. S., Yu-Poth, S., Huth, P., Moriarty, K., Fishell, V., Hargrove, R. L., Zhao, G., & Etherton, T. D. (2000). Polyunsaturated fatty acids in the food chain in the United States. *The American Journal of Clinical Nutrition*, *71*(1), 179–188.

Kübeck, R., Bonet-Ripoll, C., Hoffmann, C., Walker, A., Müller, V. M., Schüppel, V. L., Lagkouvardos, I., Scholz, B., Engel, K. H., Daniel, H., & Schmitt-Kopplin, P. (2016). Dietary fat and gut microbiota interactions determine diet-induced obesity in mice. *Molecular Metabolism*, *5*(12), 1162–1174.

Kuchta, A., Lebiedzińska, A., Fijałkowski, M., Gałąska, R., Kreft, E., Totoń, M., Czaja, K., Kozłowska, A., Ćwiklińska, A., Kortas-Stempak, B., & Strzelecki, A. (2016). Impact of plant-based diet on lipid risk factors for atherosclerosis. *Cardiology Journal*, *23*(2), 141–148.

Küllenberg, D., Taylor, L. A., Schneider, M., & Massing, U. (2012). Health effects of dietary phospholipids. *Lipids in Health and Disease*, *11*(1), 3.

Kumar, V. B., Lee, C. H., Su, T. C., Lin, C. C., Mohammedsaleh, Z. M., Yeh, C. M., Kiefer, R., & Lin, S. H. (2023). Prognostic and Clinical Implications of UNC13C expression in Hepatocellular Carcinoma Patients. *International Journal of Medical Sciences*, *20*(9), 1235.

Kumon, H., Yoshino, Y., Funahashi, Y., Mori, H., Ueno, M., Ozaki, Y., Yamazaki, K., Ochi, S., Mori, T., Iga, J. I., & Nagai, M. (2021). PICALM mRNA expression in the blood of patients with neurodegenerative diseases and geriatric depression. *Journal of Alzheimer's Disease*, *79*(3), 1055–1062.

Kunnath, A. J., Sack, D. E., & Wilkins, C. H. (2024). Relative predictive value of sociodemographic factors for chronic diseases among All of Us participants: A descriptive analysis. *BMC Public Health*, *24*(1), 405.

Kunzmann, A. T., Murray, L. J., Cardwell, C. R., McShane, C. M., McMenamin, U. C., & Cantwell, M. M. (2013). PTGS2 (Cyclooxygenase-2) expression and survival among colorectal cancer patients: A systematic review. *Cancer Epidemiology, Biomarkers & Prevention*, *22*(9), 1490–1497.

Kushner, I., & Antonelli, M. J. (2015). What should we regard as an "elevated" C-reactive protein level? *Annals of Internal Medicine*, *163*(4), 326.

Ladeia, A. M., Costa-Matos, E., Barata-Passos, R., & Guimarães, A. C. (2008). A palm oil–rich diet may reduce serum lipids in healthy young individuals. *Nutrition*, *24*(1), 11–15.

Lagoumintzis, G., & Patrinos, G. P. (2023). Triangulating nutrigenomics, metabolomics and microbiomics toward personalized nutrition and healthy living. *Hum Genomics*, *17*, 109.

Laine, P. S., Schwartz, E. A., Wang, Y., Zhang, W. Y., Karnik, S. K., Musi, N., & Reaven, P. D. (2007). Palmitic acid induces IP-10 expression in human macrophages via NF-κB activation. *Biochemical and Biophysical Research Communications*, *358*(1), 150–155.

Lamptey, R. N., Chaulagain, B., Trivedi, R., Gothwal, A., Layek, B., & Singh, J. (2022). A review of the common neurodegenerative disorders: Current therapeutic approaches and the potential role of nanotherapeutics. *International Journal of Molecular Sciences*, *23*(3), 1851.

Lane, M. M., Gamage, E., Du, S., Ashtree, D. N., McGuinness, A. J., Gauci, S., Baker, P., Lawrence, M., Rebholz, C. M., Srour, B., & Touvier, M. (2024). Ultra-processed food exposure and adverse health outcomes: Umbrella review of epidemiological meta-analyses. *Bmj*, *384*.

Lang, T., & Meng, L. (2025). CYBB identified as a key immune hub gene linking lung cancer and atrial fibrillation. *Journal of Cardiothoracic Surgery*, *20*(1), 262.

Lankinen, M. A., Stančáková, A., Uusitupa, M., Ågren, J., Pihlajamäki, J., Kuusisto, J., Schwab, U., & Laakso, M. (2015). Plasma fatty acids as predictors of glycaemia and type 2 diabetes. *Diabetologia*, *58*, 2533–2544.

Latif, R. (2013). Chocolate/cocoa and human health: A review. *Neth J Med*, *71*(2), 63–68.

Laugerette, F., Furet, J. P., Debard, C., Daira, P., Loizon, E., Géloën, A., Soulage, C. O., Simonet, C., Lefils-Lacourtablaise, J., Bernoud-Hubac, N., & Bodennec, J. (2012). Oil composition of high-fat diet affects metabolic inflammation differently in connection with endotoxin receptors in mice. *American Journal of Physiology-Endocrinology and Metabolism*, *302*(3), 374–386.

Laurell, H., Bouisson, M., Berthelémy, P., Rochaix, P., Déjean, S., Besse, P., Susini, C., Pradayrol, L., Vaysse, N., & Buscail, L. (2006). Identification of biomarkers of human pancreatic adenocarcinomas by expression profiling and validation with gene expression analysis in endoscopic ultrasound-guided fine needle aspiration samples. *World Journal of Gastroenterology: WJG*, *12*(21), 3344.

Lauterbach, M. A., & Wunderlich, F. T. (2017). Macrophage function in obesity-induced inflammation and insulin resistance. *Pflügers Archiv-European Journal of Physiology*, *469*, 385–396.

Laville, M., & Nazare, J. A. (2009). Diabetes, insulin resistance and sugars. *Obesity Reviews*, *10*, 24–33.

Lawrence, T. (2010). Macrophages and NF-κB in cancer. In *NF-kB in Health and Disease* (pp. 171–184).

Le Chatelier, E., Nielsen, T., Qin, J., Prifti, E., Hildebrand, F., Falony, G., Almeida, M., Arumugam, M., Batto, J. M., Kennedy, S., & Leonard, P. (2013). Richness of human gut microbiome correlates with metabolic markers. *Nature*, *500*(7464), 541–546.

Lee, E. C., Anand, V. V., Razavi, A. C., Alebna, P. L., Muthiah, M. D., Siddiqui, M. S., Chew, N. W., & Mehta, A. (2024). The Global Epidemic of Metabolic Fatty Liver Disease. *Current Cardiology Reports*, 1–12.

Lee, H. J., Yong, H. I., Kim, M., Choi, Y. S., & Jo, C. (2020). Status of meat alternatives and their potential role in the future meat market—A review. *Asian-Australasian Journal of Animal Sciences*, *33*(10), 1533.

Lee, H., Tu, T. H., Park, B. S., Yang, S., & Kim, J. G. (2019). Adiponectin Reverses the Hypothalamic Microglial Inflammation during Short-Term Exposure to Fat-Rich Diet. *International journal of molecular sciences*, *20*(22), 5738.

Lee, J. Y., Bae, E., Kim, H. Y., Lee, K. M., Yoon, S. S., & Lee, D. C. (2021). High-Fat-Diet–Induced Oxidative Stress Linked to the Increased Colonization of Lactobacillus sakei in an Obese Population. *Microbiology Spectrum*, *9*(1), 10–1128.

Lee, J. Y., Sohn, K. H., Rhee, S. H., & Hwang, D. (2001). Saturated fatty acids, but not unsaturated fatty acids, induce the expression of cyclooxygenase-2 mediated through Toll-like receptor 4. *Journal of Biological Chemistry*, *276*(20), 16683–16689.

Lee, Y., Lai, H. T., Oliveira Otto, M. C., Lemaitre, R. N., McKnight, B., King, I. B., Song, X., Huggins, G. S., Vest, A. R., Siscovick, D. S., & Mozaffarian, D. (2020). Serial biomarkers of de novo lipogenesis fatty acids and incident heart failure in older adults: The cardiovascular health study. *Journal of the American Heart Association*, *9*(4), 014119.

Lehner, A., Staub, K., Aldakak, L., Eppenberger, P., Rühli, F., Martin, R. D., & Bender, N. (2021). Impact of omega-3 fatty acid DHA and EPA supplementation in pregnant or breast-feeding women on cognitive performance of children: Systematic review and meta-analysis. *Nutrition Reviews*, *79*(5), 585–598.

Leong, K. S., & Wilding, J. P. (1999). Obesity and diabetes. *Best Practice & Research Clinical Endocrinology & Metabolism*, *13*(2), 221–237.

LeRoith, D. (2002). β-cell dysfunction and insulin resistance in type 2 diabetes: Role of metabolic and genetic abnormalities. *The American journal of medicine*, *113*(6), 3–11.

Li, P., Li, L., Zhang, C., Cheng, X., Zhang, Y., Guo, Y., Long, M., Yang, S., & He, J. (2019). Palmitic acid and β-hydroxybutyrate induce inflammatory responses in bovine endometrial cells by activating oxidative stress-mediated NF-κB signaling. *Molecules*, *24*(13), 2421.

Li, X., Zhai, Y., Zhao, J., He, H., Li, Y., Liu, Y., Feng, A., Li, L., Huang, T., Xu, A., & Lyu, J. (2021). Impact of metabolic syndrome and it's components on prognosis in patients with cardiovascular diseases: A meta-analysis. *Frontiers in Cardiovascular Medicine*, *8*, 704145.

Li, Y., Hruby, A., Bernstein, A. M., Ley, S. H., Wang, D. D., Chiuve, S. E., Sampson, L., Rexrode, K. M., Rimm, E. B., Willett, W. C., & Hu, F. B. (2015). Saturated fats compared with unsaturated fats and sources of carbohydrates in relation to risk of coronary heart disease: A prospective cohort study. *Journal of the American College of Cardiology*, *66*(14), 1538–1548.

Lian, C. Y., Zhai, Z. Z., Li, Z. F., & Wang, L. (2020). High fat diet-triggered non-alcoholic fatty liver disease: A review of proposed mechanisms. *Chemico-Biological Interactions*, *330*, 109199.

Liang, J., Huang, Y., Yang, C., Huang, S., Xie, J., Nong, X., Liu, J., Zhang, Y., & Zhang, Z. (2021). The effect of PPP2CA expression on the prognosis of patients with hepatocellular carcinoma and its molecular biological characteristics. *Journal of Gastrointestinal Oncology*, *12*(6), 3008.

Liao, T. H., Hamosh, P., & Hamosh, M. (1984). Fat digestion by lingual lipase: Mechanism of lipolysis in the stomach and upper small intestine. *Pediatric Research*, *18*(5), 402–409.

Libby, P. (2021). Inflammation in atherosclerosis—No longer a theory. *Clinical Chemistry*, *67*(1), 131–142.

Liew, M. Y. B., Ghazali, H. M., Yazid, A. M., Lai, O. M., Chow, M. C., Yusoff, M. S. A., & Long, K. (2001). Rheological properties of ice cream emulsion prepared from lipase-catalyzed transesterified palm kernel olein: Anhydrous milk fat mixture. *Journal of Food Lipids*, *8*(2), 131–146.

Lin, X. M., Luo, W., Wang, H., Li, R. Z., Huang, Y. S., Chen, L. K., & Wu, X. P. (2019). The role of prostaglandin-endoperoxide synthase-2 in chemoresistance of non-small cell lung cancer. *Frontiers in Pharmacology*, *10*, 836.

Lin, X., Liou, Y. H., Li, Y., Gong, L., Li, Y., Hao, Y., Pham, B., Xu, S., Jiang, Z., Li, L., & Peng, Y. (2020). FAM13A represses AMPK activity and regulates hepatic glucose and lipid metabolism. *Iscience*, *23*(3).

Lindheim, L., Bashir, M., Münzker, J., Trummer, C., Zachhuber, V., Leber, B., Horvath, A., Pieber, T. R., Gorkiewicz, G., Stadlbauer, V., & Obermayer-Pietsch, B. (2017). Alterations in gut microbiome composition and barrier function are associated with reproductive and metabolic defects in women with polycystic ovary syndrome (PCOS): A pilot study. *PloS One*, *12*(1), 0168390.

Listenberger, L. L., Han, X., Lewis, S. E., Cases, S., Farese Jr, R. V., Ory, D. S., & Schaffer, J. E. (2003). Triglyceride accumulation protects against fatty acid-induced lipotoxicity. *Proceedings of the National Academy of Sciences*, *100*(6), 3077-3082.

Litmanovitz, I., Davidson, K., Eliakim, A., Regev, R. H., Dolfin, T., Arnon, S., Bar-Yoseph, F., Goren, A., Lifshitz, Y., & Nemet, D. (2013). High beta-palmitate formula and bone strength in term infants: A randomized, double-blind, controlled trial. *Calcified Tissue International*, *92*, 35–41.

Liu, B. N., Liu, X. T., Liang, Z. H., & Wang, J. H. (2021). Gut microbiota in obesity. *World journal of gastroenterology*, *27*(25), 3837.

Liu, B., Zong, G., Zhu, L., Hu, Y., Manson, J. E., Wang, M., Rimm, E. B., Hu, F. B., & Sun, Q. (2024). Chocolate intake and risk of type 2 diabetes: Prospective cohort studies. *Bmj*, *387*.

Liu, C. C., Kanekiyo, T., Xu, H., & Bu, G. (2013). Apolipoprotein E and Alzheimer disease: Risk, mechanisms and therapy. *Nature Reviews Neurology*, *9*(2), 106–118.

Liu, L., Feng, R., Guo, F., Li, Y., Jiao, J., & Sun, C. (2015). Targeted metabolomic analysis reveals the association between the postprandial change in palmitic acid, branched-chain amino acids and insulin resistance in young obese subjects. *Diabetes Research and Clinical Practice*, *108*(1), 84–93.

Liu, M., Sun, J., Cui, J., Chen, W., Guo, H., Barbetti, F., & Arvan, P. (2015). INS-gene mutations: From genetics and beta cell biology to clinical disease. *Molecular Aspects of Medicine*, *42*, 3–18.

Liu, Q., Bengmark, S., & Qu, S. (2010). The role of hepatic fat accumulation in pathogenesis of non-alcoholic fatty liver disease (NAFLD. *Lipids in Health and Disease*, *9*(1), 42.

Liu, T., Zhang, L., Joo, D., & Sun, S. C. (2017). NF-κB signaling in inflammation. *Signal Transduction and Targeted Therapy*, *2*(1), 1–9.

Liu, X., Zhao, K., Yang, X., & Zhao, Y. (2019). Gut microbiota and metabolome response of Decaisnea insignis seed oil on metabolism disorder induced by excess alcohol consumption. *Journal of Agricultural and Food Chemistry*, *67*(38), 10667–10677.

Lobo, A. J., Jones, S. C., Juby, L. D., & Axon, A. T. (1992). Plasma viscosity in inflammatory bowel disease. *Journal of clinical pathology*, *45*(1), 54–57.

Lockyer, S., & Stanner, S. (2016). Coconut oil-a nutty idea? *Nutrition Bulletin*, *41*(1).

Loomba, R., & Sanyal, A. J. (2013). The global NAFLD epidemic. *Nature Reviews Gastroenterology & Hepatology*, *10*(11), 686–690.

López-Gómez, C., Santiago-Fernández, C., García-Serrano, S., García-Escobar, E., Gutiérrez-Repiso, C., Rodríguez-Díaz, C., ... & García-Fuentes, E. (2020). Oleic acid protects against insulin resistance by regulating the genes related to the PI3K signaling pathway. *Journal of Clinical Medicine*, *9*(8), 2615.

Lopez-Huertas, E. (2010). Health effects of oleic acid and long chain omega-3 fatty acids (EPA and DHA) enriched milks. A review of intervention studies. *Pharmacological Research*, *61*(3), 200–207.

Lopez-Lopez, A., Lopez-Sabater, M. C., Campoy-Folgoso, C., Rivero-Urgell, M., & Castellote-Bargallo, A. I. (2002). Fatty acid and sn-2 fatty acid composition in human milk from Granada (Spain) and in infant formulas. *European Journal of Clinical Nutrition*, *56*(12), 1242–1254.

Lucci, P., Borrero, M., Ruiz, A., Pacetti, D., Frega, N. G., Diez, O., Ojeda, M., Gagliardi, R., Parra, L., & Angel, M. (2016). Palm oil and cardiovascular disease: A randomized trial of the effects of hybrid palm oil supplementation on human plasma lipid patterns. *Food & Function*, *7*(1), 347–354.

Luiten, C. M., Steenhuis, I. H., Eyles, H., Mhurchu, C. N., & Waterlander, W. E. (2016). Ultra-processed foods have the worst nutrient profile, yet they are the most available packaged products in a sample of New Zealand supermarkets. *Public Health Nutrition*, *19*(3), 530–538.

Lumley, T., Diehr, P., Emerson, S., & Chen, L. (2002). The importance of the normality assumption in large public health data sets. *Annual review of public health, 23*(1), 151-169.

Lunn, J., & Theobald, H. E. (2006). The health effects of dietary unsaturated fatty acids. *Nutrition Bulletin*, *31*(3), 178–224.

Lunn, J., & Theobald, H. E. (2006). The health effects of dietary unsaturated fatty acids. *Nutrition Bulletin*, *31*(3), 178–224.

Luukkonen, P. K., Sädevirta, S., Zhou, Y., Kayser, B., Ali, A., Ahonen, L., Lallukka, S., Pelloux, V., Gaggini, M., Jian, C., & Hakkarainen, A. (2018). Saturated fat is more metabolically harmful for the human liver than unsaturated fat or simple sugars. *Diabetes Care*, *41*(8), 1732–1739.

Lv, C., Wang, Y., Zhou, C., Ma, W., Yang, Y., Xiao, R., & Yu, H. (2018). Effects of dietary palm olein on the cardiovascular risk factors in healthy young adults. *Food & Nutrition Research*, *62*, 10–29219.

Lv, Y., Meng, B., Dong, H., Jing, T., Wu, N., Yang, Y., Huang, L., Moses, R. E., O'Malley, B. W., Mei, B., & Li, X. (2016). Upregulation of GSK3β contributes to brain disorders in elderly REGγ-knockout mice. *Neuropsychopharmacology*, *41*(5), 1340–1349.

Lynn, M. (1992). The profitability of the early nineteenth-century palm oil trade. *African Economic History*, *20*, 77–97.

Lytrivi, M., Castell, A. L., Poitout, V., & Cnop, M. (2020). Recent insights into mechanisms of β-cell lipo-and glucolipotoxicity in type 2 diabetes. *Journal of Molecular Biology*, *432*(5), 1514–1534.

Ma, C., Vasu, R., & Zhang, H. (2019). The role of long-chain fatty acids in inflammatory bowel disease. *Mediators of Inflammation*.

Ma, H., & Shieh, K. J. (2006). Cholesterol and human health. *The Journal of American Science*, *2*(1), 46–50.

Ma, Q., Li, L. Y., Le, J. Y., Lu, D. L., Qiao, F., Zhang, M. L., Du, Z. Y., & Li, D. L. (2018). Dietary microencapsulated oil improves immune function and intestinal health in Nile tilapia fed with high-fat diet. *Aquaculture*, *496*, 19–29.

Ma, X., Su, J., Zhao, S., He, Y., Li, S., Yang, X., Zhai, S., Rong, S., Zhang, X., Xu, G., & Xie, X. (2022). CCL3 Promotes Proliferation of Colorectal Cancer Related with TRAF6/NF-κB Molecular Pathway. *Contrast Media & Molecular Imaging*, *2022*(1), 2387192.

Macfarlane, G. T., & Cummings, J. H. (1999). Probiotics and prebiotics: Can regulating the activities of intestinal bacteria benefit health? *Bmj*, *318*(7189), 999–1003.

Macura, B., Kiecka, A., & Szczepanik, M. (2024). Intestinal permeability disturbances: Causes, diseases and therapy. *Clinical and Experimental Medicine*, *24*(1), 232.

Maddur, M. S., Miossec, P., Kaveri, S. V., & Bayry, J. (2012). Th17 cells: Biology, pathogenesis of autoimmune and inflammatory diseases, and therapeutic strategies. *The American Journal of Pathology*, *181*(1), 8–18.

Madoromae, H., & Lertcanawanichakul, M. (2025). Red Palm Oil: Nutritional Composition, Bioactive Properties, and Potential Applications in Health and Cosmetics: A Narrative Review. *Molecules*, *30*(22), 4402.

Magder, S. (2018). The meaning of blood pressure. *Critical Care*, *22*(1), 257.

Magne, F., Gotteland, M., Gauthier, L., Zazueta, A., Pesoa, S., Navarrete, P., & Balamurugan, R. (2020). The firmicutes/bacteroidetes ratio: A relevant marker of gut dysbiosis in obese patients? *Nutrients*, *12*(5), 1474.

Magri, T. P., Fernandes, F. S., Souza, A. S., Langhi, L. G., Barboza, T., Misan, V., Mucci, D. B., Santos, R. M., Nunes, T. F., Souza, S. A., & Mello Coelho, V. (2015). Interesterified fat or palm oil as substitutes for partially hydrogenated fat in maternal diet can predispose obesity in adult male offspring. *Clinical Nutrition*, *34*(5), 904–910.

Maki, K. C., Dicklin, M. R., & Kirkpatrick, C. F. (2021). Saturated fats and cardiovascular health: Current evidence and controversies. *Journal of Clinical Lipidology*, *15*(6), 765–772.

Malekmohammad, K., Bezsonov, E. E., & Rafieian-Kopaei, M. (2021). Role of lipid accumulation and inflammation in atherosclerosis: Focus on molecular and cellular mechanisms. *Frontiers in Cardiovascular Medicine*, *8*, 707529.

Malesza, I. J., Malesza, M., Walkowiak, J., Mussin, N., Walkowiak, D., Aringazina, R., Bartkowiak-Wieczorek, J., & Mądry, E. (2021). High-fat, western-style diet, systemic inflammation, and gut microbiota: A narrative review. *Cells*, *10*(11), 3164.

Mancini, A., Imperlini, E., Nigro, E., Montagnese, C., Daniele, A., Orrù, S., & Buono, P. (2015). Biological and nutritional properties of palm oil and palmitic acid: Effects on health. *Molecules*, *20*(9), 17339–17361.

Manichanh, C., Borruel, N., Casellas, F., & Guarner, F. (2012). The gut microbiota in IBD. *Nature Reviews Gastroenterology & Hepatology*, *9*(10), 599–608.

Mann, G. V. (1994). Metabolic consequences of dietary trans fatty acids. *The Lancet*, *343*(8908), 1268–1271.

Mansour, S. R., Moustafa, M. A. A., Saad, B. M., Hamed, R., & Moustafa, A. R. (2021). Impact of diet on human gut microbiome and disease risk. *New Microbes and New Infections*, *41*, 100845.

Mantovani, A., & Dalbeni, A. (2022). Recent Developments in NAFLD. *International Journal of Molecular Sciences*, *23*(5), 2882.

Marie, C., Pitton, C., Fitting, C., & Cavaillon, J. M. (1996). Regulation by anti-inflammatory cytokines (IL-4, IL-10, IL-13, TGFβ) of interleukin-8 production by LPS-and/or TNFα-activated human polymorphonuclear cells. *Mediators of Inflammation*, *5*, 334–340.

Marrón-Ponce, J. A., Tolentino-Mayo, L., Hernández-F, M., & Batis, C. (2018). Trends in ultra-processed food purchases from 1984 to 2016 in Mexican households. *Nutrients*, *11*(1), 45.

Martín-Peláez, S., Fito, M., & Castaner, O. (2020). Mediterranean diet effects on type 2 diabetes prevention, disease progression, and related mechanisms. *A Review. Nutrients*, *12*(8), 2236.

Martín-Torres, S., Tello-Jiménez, J. A., López-Blanco, R., González-Casado, A., & Cuadros-Rodríguez, L. (2022). Monitoring the shelf life of refined vegetable oils under market storage conditions—A kinetic chemofoodmetric approach. *Molecules*, *27*(19), 6508.

Martinez, K. B., Leone, V., & Chang, E. B. (2017). Western diets, gut dysbiosis, and metabolic diseases: Are they linked? *Gut Microbes*, *8*(2), 130–142.

Martínez, M., & Mougan, I. (1998). Fatty acid composition of human brain phospholipids during normal development. *Journal of Neurochemistry*, *71*(6), 2528–2533.

Marzuki, A., Arshad, F., Razak, T. A., & Jaarin, K. (1991). Influence of dietary fat on plasma lipid profiles of Malaysian adolescents. *The American Journal of Clinical Nutrition*, *53*(4), 1010–1014.

Mathers, C.D., Lopez, A.D. and Murray, C.J., 2006. The burden of disease and mortality by condition: data, methods, and results for 2001. *Global burden of disease and risk factors*. Available at: https://www.ncbi.nlm.nih.gov/books/NBK11808/

Mathew, T.K. and Tadi, P., 2020. Blood glucose monitoring. *StatPearls Publishing, Treasure Island (FL).* Available at: https://europepmc.org/article/NBK/nbk555976

Matin, A., Brandić, I., Gubor, M., Pezo, L., Krička, T., Matin, B., Jurišić, V., & Antonović, A. (2024). Effect of conduction drying on nutrient and fatty acid profiles: A comparative analysis of hazelnuts and walnuts. *Frontiers in Sustainable Food Systems*, *8*, 1351309.

Mattiuzzi, C., & Lippi, G. (2019). Current cancer epidemiology. *Journal of Epidemiology and Global Health*, *9*(4), 217–222.

Mattson, F. H. (1989). A changing role for dietary monounsaturated fatty acids. *Journal of the American Dietetic Association*, *89*(3), 387–391.

Mattson, F. H., & Grundy, S. M. (1985). Comparison of effects of dietary saturated, monounsaturated, and polyunsaturated fatty acids on plasma lipids and lipoproteins in man. *Journal of Lipid Research*, *26*(2), 194–202.

Maurice, J., & Manousou, P. (2018). Non-alcoholic fatty liver disease. *Clinical medicine*, *18*(3), 245.

Mayneris-Perxachs, J., Cardellini, M., Hoyles, L., Latorre, J., Davato, F., Moreno-Navarrete, J. M., Arnoriaga-Rodríguez, M., Serino, M., Abbott, J., Barton, R. H., & Puig, J. (2021). Iron status influences non-alcoholic fatty liver disease in obesity through the gut microbiome. *Microbiome*, *9*(1), 104.

Mba, O. I., Dumont, M. J., & Ngadi, M. (2015). Palm oil: Processing, characterization and utilization in the food industry–A review. *Food Bioscience*, *10*, 26–41.

McGeachy, M. J., Cua, D. J., & Gaffen, S. L. (2019). The IL-17 family of cytokines in health and disease. *Immunity*, *50*(4), 892–906.

McIntosh, G. H. (2001). Cereal foods, fibres and the prevention of cancers. *Australian Journal of Nutrition and Dietetics*, *58*(4), 35–35.

McPherson, K., Marsh, T., Brown, M., & Britain, G. (2007). *Tackling obesities: Future choices: Modelling future trends in obesity and the impact on health*. Department of Innovation, Universities and Skills.

Medina, V. A., & Rivera, E. S. (2010). Histamine receptors and cancer pharmacology. *British Journal of Pharmacology*, *161*(4), 755–767.

Mehrotra, V., Sehgal, S. K., & Bangale, N. R. (2019). Fat structure and composition in human milk and infant formulas: Implications in infant health. *Clinical Epidemiology and Global Health*, *7*(2), 153-159.

Mei, J., Qian, M., Hou, Y., Liang, M., Chen, Y., Wang, C., & Zhang, J. (2024). Association of saturated fatty acids with cancer risk: A systematic review and meta-analysis. *Lipids in Health and Disease*, *23*(1), 32.

Meijaard, E., Brooks, T. M., Carlson, K. M., Slade, E. M., Garcia-Ulloa, J., Gaveau, D. L., Lee, J. S. H., Santika, T., Juffe-Bignoli, D., Struebig, M. J., & Wich, S. A. (2020). The environmental impacts of palm oil in context. *Nature Plants*, *6*(12), 1418–1426.

Menni, C., Jackson, M. A., Pallister, T., Steves, C. J., Spector, T. D., & Valdes, A. M. (2017). Gut microbiome diversity and high-fibre intake are related to lower long-term weight gain. *International Journal of Obesity*, *41*(7), 1099–1105.

Mensink, R. P. (2008). Effects of products made from a high-palmitic acid, trans-free semiliquid fat or a high-oleic acid, low-trans semiliquid fat on the serum lipoprotein profile and on C-reactive protein concentrations in humans. *European Journal of Clinical Nutrition*, *62*(5), 617–624.

Mercola, J., & D'Adamo, C. R. (2023). Linoleic acid: A narrative review of the effects of increased intake in the standard American diet and associations with chronic disease. *Nutrients*, *15*(14), 3129.

Messina, M. J., Sievenpiper, J. L., Williamson, P., Kiel, J., & Erdman, J. W. (2023). Ultra-processed foods: A concept in need of revision to avoid targeting healthful and sustainable plant-based foods. In *Public Health Nutrition* (pp. 1–2).

Metoudi, M., Bauer, A., Haffner, T., & Kassam, S. (2025). A cross-sectional survey exploring knowledge, beliefs and barriers to whole food plant-based diets amongst registered

dietitians in the United Kingdom and Ireland. *Journal of Human Nutrition and Dietetics*, *38*(1), 13386.

Michels, N., Specht, I. O., Heitmann, B. L., Chajès, V., & Huybrechts, I. (2021). Dietary trans-fatty acid intake in relation to cancer risk: A systematic review and meta-analysis. *Nutrition Reviews*, *79*(7), 758–776.

Michor, F., Iwasa, Y., & Nowak, M. A. (2004). Dynamics of cancer progression. *Nature Reviews Cancer*, *4*(3), 197–205.

Mielck, A., Vogelmann, M., & Leidl, R. (2014). Health-related quality of life and socioeconomic status: Inequalities among adults with a chronic disease. *Health and Quality of Life Outcomes*, *12*, 1–1.

Mierziak, J., Kostyn, K., Boba, A., Czemplik, M., Kulma, A., & Wojtasik, W. (2021). Influence of the bioactive diet components on the gene expression regulation. *Nutrients*, *13*(11), 3673.

Milard, M., Laugerette, F., Durand, A., Buisson, C., Meugnier, E., Loizon, E., Louche-Pelissier, C., Sauvinet, V., Garnier, L., Viel, S., & Bertrand, K. (2019). Milk polar lipids in a high-fat diet can prevent body weight gain: Modulated abundance of gut bacteria in relation with fecal loss of specific fatty acids. *Molecular Nutrition & Food Research*, *63*(4), 1801078.

Miles, E. A., & Calder, P. C. (2017). The influence of the position of palmitate in infant formula triacylglycerols on health outcomes. *Nutrition Research*, *44*, 1–8.

Mintel. (2018) *Mintel Global New Products Database (GNPD).* Available at: https://www.mintel.com/press-centre/veganuary-uk-overtakes-germany-as-worlds-leader-for-vegan-food-launches/

Mirzaei, S., Paskeh, M. D. A., Entezari, M., Mirmazloomi, S., Hassanpoor, A., Aboutalebi, M., Rezaei, S., Hejazi, E. S., Kakavand, A., Heidari, H., & Salimimoghadam, S. (2022). SOX2

function in cancers: Association with growth, invasion, stemness and therapy response. *Biomedicine & Pharmacotherapy*, *156*, 113860.

Mohan, S., & Campbell, N. R. (2009). Salt and high blood pressure. *Clinical Science*, *117*(1), 1–11.

Mohd Hanafiah, K., Abd Mutalib, A. H., Miard, P., Goh, C. S., Mohd Sah, S. A., & Ruppert, N. (2021). Impact of Malaysian palm oil on sustainable development goals: Co-benefits and trade-offs across mitigation strategies. *Sustainability Science*, 1–23.

Moller, D. E. (2000). Potential role of TNF-α in the pathogenesis of insulin resistance and type 2 diabetes. *Trends in Endocrinology & Metabolism*, *11*(6), 212–217.

Monteiro, C. A., Cannon, G., Levy, R. B., Moubarac, J. C., Louzada, M. L., Rauber, F., Khandpur, N., Cediel, G., Neri, D., Martinez-Steele, E., & Baraldi, L. G. (2019). Ultra-processed foods: What they are and how to identify them. *Public Health Nutrition*, *22*(5), 936–941.

Monteiro, C. A., Levy, R. B., Claro, R. M., Castro, I. R. R., & Cannon, G. (2010). Increasing consumption of ultra-processed foods and likely impact on human health: Evidence from Brazil. *Public Health Nutrition*, *14*(1), 5–13.

Monteiro, C. A., Moubarac, J. C., Cannon, G., Ng, S. W., & Popkin, B. (2013). Ultra-processed products are becoming dominant in the global food system. *Obesity Reviews*, *14*, 21–28.

Monteiro, C. A., Moubarac, J. C., Levy, R. B., Canella, D. S., Costa Louzada, M. L., & Cannon, G. (2018). Household availability of ultra-processed foods and obesity in nineteen European countries. *Public Health Nutrition*, *21*(1), 18–26.

Montoya, C., Cochard, B., Flori, A., Cros, D., Lopes, R., Cuellar, T., ... & Billotte, N. (2014). Genetic architecture of palm oil fatty acid composition in cultivated oil palm (Elaeis guineensis Jacq.) compared to its wild relative E. oleifera (HBK) Cortés. *PloS one*, *9*(5), e95412.

Mortensen, M. B., & Nordestgaard, B. G. (2020). Elevated LDL cholesterol and increased risk of myocardial infarction and atherosclerotic cardiovascular disease in individuals aged 70–100 years: A contemporary primary prevention cohort. *The Lancet*, *396*(10263), 1644–1652.

Mortensen, M. B., Dzaye, O., Bøtker, H. E., Jensen, J. M., Maeng, M., Bentzon, J. F., Kanstrup, H., Sørensen, H. T., Leipsic, J., Blankstein, R., & Nasir, K. (2023). Low-density lipoprotein cholesterol is predominantly associated with atherosclerotic cardiovascular disease events in patients with evidence of coronary atherosclerosis: The western Denmark heart registry. *Circulation*, *147*(14), 1053–1063.

Mosca, A., Leclerc, M., & Hugot, J. P. (2016). Gut microbiota diversity and human diseases: Should we reintroduce key predators in our ecosystem? *Frontiers in Microbiology*, *7*, 455.

Mukherjee, S., & Mitra, A. (2009). Health effects of palm oil. *Journal of Human Ecology*, *26*(3), 197–203.

Mukhtar, Y., Galalain, A., & Yunusa, U. (2020). A modern overview on diabetes mellitus: A chronic endocrine disorder. *European Journal of Biology*, *5*(2), 1–14.

Müller, H., Jordal, O., Kierulf, P., Kirkhus, B., & Pedersen, J. I. (1998). Replacement of partially hydrogenated soybean oil by palm oil in margarine without unfavorable effects on serum lipoproteins. *Lipids*, *33*(9), 879–887.

Muñoz-Alvarez, K. Y., Gutiérrez-Aguilar, R., & Frigolet, M. E. (2024). Metabolic effects of milk fatty acids: A literature review. *Nutrition Bulletin*, *49*(1), 19–39.

Muralidharan, J., Galiè, S., Hernández-Alonso, P., Bulló, M., & Salas-Salvadó, J. (2019). Plant-based fat, dietary patterns rich in vegetable fat and gut microbiota modulation. *Frontiers in Nutrition*, *6*, 157.

Murphy, D. J. (2024). Sustainable vegetable oils. In *Functional Dietary Lipids* (pp. 31–47). Woodhead Publishing.

Murru, E., Manca, C., Carta, G., & Banni, S. (2022). Impact of dietary palmitic acid on lipid metabolism. *Frontiers in Nutrition*, *9*, 861664.

Myers, S. P., & Hawrelak, J. (2004). The causes of intestinal dysbiosis: A review. *Altern Med Rev*, *9*(2), 180–197.

Nadalin, V., Cotterchio, M., & Kreiger, N. (2003). Antihistamine use and breast cancer risk. *International Journal of Cancer*, *106*(4), 566–568.

Nagpal, R., Yadav, H., & Marotta, F. (2014). Gut microbiota: The next-gen frontier in preventive and therapeutic medicine? *Frontiers in Medicine*, *1*, 15.

Naj, A. C., Jun, G., Beecham, G. W., Wang, L. S., Vardarajan, B. N., Buros, J., Gallins, P. J., Buxbaum, J. D., Jarvik, G. P., Crane, P. K., & Larson, E. B. (2011). Common variants at MS4A4/MS4A6E, CD2AP, CD33 and EPHA1 are associated with late-onset Alzheimer's disease. *Nature Genetics*, *43*(5), 436–441.

Nájera Espinosa, S., Hadida, G., Jelmar Sietsma, A., Alae-Carew, C., Turner, G., Green, R., & Scheelbeek, P. (2025). Mapping the evidence of novel plant-based foods: A systematic review of nutritional, health, and environmental impacts in high-income countries. *Nutrition Reviews*, *83*(7), 1626–1646.

Nangia, R., Singh, H., & Kaur, K. (2016). Prevalence of cardiovascular disease (CVD) risk factors. *Medical Journal Armed Forces India*, *72*(4), 315–319.

Natali, F., Siculella, L., Salvati, S., & Gnoni, G. V. (2007). Oleic acid is a potent inhibitor of fatty acid and cholesterol synthesis in C6 glioma cells. *Journal of Lipid Research*, *48*(9), 1966-1975.

National Health Service (NHS) England. (2024). *Health Survey for England, 2022 Part 2.* Available at: https://digital.nhs.uk/data-and-information/publications/statistical/health-survey-for-england/2022-part-2/adult-overweight-and-obesity

National Health Service (NHS). (2023). *Fat: the facts.* Available at: https://www.nhs.uk/live-well/eat-well/food-types/different-fats-nutrition/

Nedkoff, L., Briffa, T., Zemedikun, D., Herrington, S., & Wright, F. L. (2023). *Global trends in atherosclerotic cardiovascular disease*. Clinical Therapeutics.

Nelson, R. H. (2013). Hyperlipidemia as a risk factor for cardiovascular disease. *Primary Care: Clinics in Office Practice*, *40*(1), 195–211.

Nesaretnam, K., Guthrie, N., Chambers, A. F., & Carroll, K. K. (1995). Effect of tocotrienols on the growth of a human breast cancer cell line in culture. *Lipids*, *30*(12), 1139–1143.

Nestel, P., Clifton, P., & Noakes, M. (1994). Effects of increasing dietary palmitoleic acid compared with palmitic and oleic acids on plasma lipids of hypercholesterolemic men. *Journal of Lipid Research*, *35*(4), 656–662.

Ng, T. K., Hassan, K., Lim, J. B., Lye, M. S., & Ishak, R. (1991). Nonhypercholesterolemic effects of a palm-oil diet in Malaysian volunteers. *The American Journal of Clinical Nutrition*, *53*(4), 1015–1020.

Ng, T. K., Hayes, K. C., DeWitt, G. F., Jegathesan, M., Satgunasingam, N., Ong, A. S., & Tan, D. (1992). Dietary palmitic and oleic acids exert similar effects on serum cholesterol and lipoprotein profiles in normocholesterolemic men and women. *Journal of the American College of Nutrition*, *11*(4), 383–390.

Nguyen, T. H., & Vu, D. C. (2023). A review on phytochemical composition and potential health-promoting properties of walnuts. *Food Reviews International*, *39*(1), 397–423.

NHS England. (2023). *Addressing low levels of health literacy; a determinant of poor health.* Available at: https://library.nhs.uk/addressing-low-levels-of-health-literacy-a-determinant-of-poor-health/

NHS England. (2025). *Tackling digital exclusion and health literacy: How libraries can help bridge the gap.* Available at: https://www.england.nhs.uk/blog/tackling-digital-exclusion-and-health-literacy-how-libraries-can-help-bridge-the-gap/

NHS. (2023). *Fat: the Facts.* Available at: https://www.nhs.uk/live-well/eat-well/food-types/different-fats-nutrition/

Nicholls, S. J., & Nelson, A. J. (2019). HDL and cardiovascular disease. *Pathology*, *51*(2), 142–147.

Nikouli, E., Kormas, K. A., Jin, Y., Olsen, Y., Bakke, I., & Vadstein, O. (2021). Dietary lipid effects on gut microbiota of first feeding Atlantic salmon (Salmo salar L. *Frontiers in Marine Science*, *8*, 665576.

Nizar, N. N. A., Marikkar, J. M. N., & Hashim, D. M. (2013). Differentiation of lard, chicken fat, beef fat and mutton fat by GCMS and EA-IRMS techniques. *Journal of Oleo Science*, *62*(7), 459–464.

Nomayo, A., Schwiertz, A., Rossi, R., Timme, K., Foster, J., Zelenka, R., Tvrdik, J., & Jochum, F. (2020). Infant formula with cow's milk fat and prebiotics affects intestinal flora, but not the incidence of infections during infancy in a double-blind randomized controlled trial. *Molecular and Cellular Pediatrics*, *7*, 1–12.

Nuzzo RL (2021). Post hoc Power. *PM & R : The Journal of Injury, Function, and Rehabilitation, 13*(4), 422–424.

Nyberg, S. T., Singh-Manoux, A., Pentti, J., Madsen, I. E., Sabia, S., Alfredsson, L., Bjorner, J. B., Borritz, M., Burr, H., Goldberg, M., & Heikkilä, K. (2020). Association of healthy lifestyle with years lived without major chronic diseases. *JAMA Internal Medicine*, *180*(5), 760–768.

Nye, C., Kim, J., Kalhan, S. C., & Hanson, R. W. (2008). Reassessing triglyceride synthesis in adipose tissue. *Trends in Endocrinology & Metabolism*, *19*(10), 356–361.

Nyquist, N. F., Krogdahl, Å., Penn, M., Kaldhusdahl, M., Thomassen, M., & Haug, A. (2013). Growth and gut health in chickens on diets varying in fatty acid composition and selenium content. *Acta Agriculturae Scandinavica, Section A-Animal Science*, *63*(3), 151–162.

O'Garra, A., Barrat, F. J., Castro, A. G., Vicari, A., & Hawrylowicz, C. (2008). Strategies for use of IL-10 or its antagonists in human disease. *Immunological Reviews*, *223*(1), 114–131.

Obradovic, M., Sudar-Milovanovic, E., Soskic, S., Essack, M., Arya, S., Stewart, A. J., ... & Isenovic, E. R. (2021). Leptin and obesity: role and clinical implication. *Frontiers in endocrinology*, *12*, 585887.

Odia, O. J., Ofori, S., & Maduka, O. (2015). Palm oil and the heart: A review. *World Journal of Cardiology*, *7*(3), 144.

Office for Health Improvement and Disparities. (2025). *SACN statement on expressing energy, fat and carbohydrate intakes and recommendations.* Available at: https://www.gov.uk/government/publications/sacn-statement-on-expressing-fat-and-carbohydrate-recommendations/sacn-statement-on-expressing-energy-fat-and-carbohydrate-intakes-and-recommendations

Office for Health Improvement and Disparities (OHID) (2025). *National Diet and Nutrition Survey 2019 to 2023.* Available at: https://www.gov.uk/government/statistics/national-diet-and-nutrition-survey-2019-to-2023

Office for Health Improvement and Disparities. (2024). *The Eatwell Guide.* Available at: https://www.gov.uk/government/publications/the-eatwell-guide

Office for National Statistics (2024). *Risk factors for pre-diabetes and undiagnosed type 2 diabetes in England: 2013 to 2019.* Available at: https://www.ons.gov.uk/peoplepopulationandcommunity/healthandsocialcare/healthineq

ualities/bulletins/riskfactorsforprediabetesandundiagnosedtype2diabetesinengland/2013to2019

Oguntibeju, O. O., Esterhuyse, A. J., & Truter, E. J. (2009). Red palm oil: Nutritional, physiological and therapeutic roles in improving human wellbeing and quality of life. *British Journal of Biomedical Science*, *66*(4), 216–222.

Olivares-Tenorio, M. L., Cortes-Prieto, C. M., Londoño-Univio, N. C., Rojas-Díaz, D. A., Quintanilla-Carvajal, M. X., Tibaquira-Perez, L. N., ... & Garcia-Nuñez, J. A. (2024). Bioactive compounds in palm oil: A comprehensive review of recent advances in physicochemical characteristics, health-promoting properties and technologies for extraction, concentration, fractionation, encapsulation and functional food applications. *Journal of Food Composition and Analysis*, *132*, 106306.

Olli, K. E., Rapp, C., O'Connell, L., Collins, C. B., McNamee, E. N., Jensen, O., Jedlicka, P., Allison, K. C., Goldberg, M. S., Gerich, M. E., & Frank, D. N. (2020). Muc5ac expression protects the colonic barrier in experimental colitis. *Inflammatory Bowel Diseases*, *26*(9), 1353–1367.

Olson, R. E. (1998). Discovery of the lipoproteins, their role in fat transport and their significance as risk factors. *The Journal of Nutrition*, *128*(2), 439–443.

Omer, E., & Chiodi, C. (2024). Fat digestion and absorption: Normal physiology and pathophysiology of malabsorption, including diagnostic testing. *Nutrition in Clinical Practice*, *39*, S6-S16.

Oostindjer, M., Alexander, J., Amdam, G. V., Andersen, G., Bryan, N. S., Chen, D., Corpet, D. E., Smet, S., Dragsted, L. O., Haug, A., & Karlsson, A. H. (2014). The role of red and processed meat in colorectal cancer development: A perspective. *Meat Science*, *97*(4), 583–596.

Osborne, R. H., Elmer, S., Hawkins, M., Cheng, C. C., Batterham, R. W., Dias, S., Good, S., Monteiro, M. G., Mikkelsen, B., Nadarajah, R. G., & Fones, G. (2022). Health literacy development is central to the prevention and control of non-communicable diseases. *BMJ Global Health*, *7*(12), 010362.

Ostfeld, R., Howarth, D., Reiner, D., & Krasny, P. (2019). Peeling back the label—Exploring sustainable palm oil ecolabelling and consumption in the United Kingdom. *Environmental Research Letters*, *14*(1), 014001.

Ouchi, N., & Walsh, K. (2007). Adiponectin as an anti-inflammatory factor. *Clinica chimica acta*, *380*(1–2), 24–30.

Ouellette, R. J., & Rawn, J. D. (2018). *Organic chemistry: Structure, mechanism, synthesis*. Academic Press.

Our World in Data. (2024). *Palm oil.* Available at: https://ourworldindata.org/palm-oil

Page-McCaw, A., Ewald, A. J., & Werb, Z. (2007). Matrix metalloproteinases and the regulation of tissue remodelling. *Nature Reviews Molecular Cell Biology*, *8*(3), 221–233.

Page, M. J., McKenzie, J. E., Bossuyt, P. M., Boutron, I., Hoffmann, T. C., Mulrow, C. D., Shamseer, L., Tetzlaff, J. M., Akl, E. A., Brennan, S. E., & Chou, R. (2021). The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. *Bmj*, *372*.

Pagliai, G., Dinu, M., Madarena, M. P., Bonaccio, M., Iacoviello, L., & Sofi, F. (2021). Consumption of ultra-processed foods and health status: A systematic review and meta-analysis. *British Journal of Nutrition*, *125*(3), 308–318.

Pala, V., Krogh, V., Muti, P., Chajès, V., Riboli, E., Micheli, A., Saadatian, M., Sieri, S., & Berrino, F. (2001). Erythrocyte membrane fatty acids and subsequent breast cancer: A prospective Italian study. *Journal of the National Cancer Institute*, *93*(14), 1088–1095.

Palomer, X., Pizarro-Delgado, J., Barroso, E., & Vázquez-Carrera, M. (2018). Palmitic and oleic acid: The yin and yang of fatty acids in type 2 diabetes mellitus. *Trends in Endocrinology & Metabolism*, *29*(3), 178–190.

Pande, G., Akoh, C. C., & Lai, O. M. (2012). Food uses of palm oil and its components. In *Palm oil* (pp. 561–586). AOCS Press.

Paniagua, J. A., Sacristana, A. G., Sánchez, E., Romero, I., Vidal-Puig, A., Berral, F. J., Escribano, A., Moyano, M. J., Peréz-Martinez, P., López-Miranda, J., & Pérez-Jiménez, F. (2007). A MUFA-rich diet improves posprandial glucose, lipid and GLP-1 responses in insulin-resistant subjects. *Journal of the American College of Nutrition*, *26*(5), 434–444.

Park, E. J., Lee, A. Y., Park, S., Kim, J. H., & Cho, M. H. (2014). Multiple pathways are involved in palmitic acid-induced toxicity. *Food and Chemical Toxicology*, *67*, 26–34.

Park, J. S., Kang, D. H., & Bae, S. H. (2015). Concerted action of p62 and Nrf2 protects cells from palmitic acid-induced lipotoxicity. *Biochemical and Biophysical Research Communications*, *466*(1), 131–137.

Paszczyk, B., & Łuczyńska, J. (2020). The comparison of fatty acid composition and lipid quality indices in hard cow, sheep, and goat cheeses. *Foods*, *9*(11), 1667.

Patricia, J. J., & Dhamoon, A. S. (2019). Physiology, digestion. *StatPearls Publishing, Treasure Island (FL).* Available at: https://europepmc.org/article/NBK/nbk544242

Patterson, E., O'Doherty, R. M., Murphy, E. F., Wall, R., O'Sullivan, O., Nilaweera, K., Fitzgerald, G. F., Cotter, P. D., Ross, R. P., & Stanton, C. (2014). Impact of dietary fatty acids on metabolic activity and host intestinal microbiota composition in C57BL/6J mice. *British Journal of Nutrition*, *111*(11), 1905–1917.

Patterson, E., Wall, R., Fitzgerald, G. F., Ross, R. P., & Stanton, C. (2012). Health implications of high dietary omega-6 polyunsaturated fatty acids. *Journal of Nutrition and Metabolism*, *2012*(1), 539426.

Pedersen, A. N., Kondrup, J., & Børsheim, E. (2013). Health effects of protein intake in healthy adults: A systematic literature review. *Food & Nutrition Research*, *57*(1), 21245.

Pedersen, J. I., Müller, H., Seljeflot, I., & Kirkhus, B. (2005). Palm oil versus hydrogenated soybean oil: Effects on serum lipids and plasma haemostatic variables. *Asia Pacific Journal of Clinical Nutrition*, *14*(4), 348.

Pendrill, F., Persson, U. M., Godar, J., Kastner, T., Moran, D., Schmidt, S., & Wood, R. (2019). Agricultural and forestry trade drives large share of tropical deforestation emissions. *Global Environmental Change*, *56*, 1–10.

Perez-Oliveira, S., Vazquez-Coto, D., Pardo, S., Blázquez-Estrada, M., Menéndez-González, M., Siso, P., Suárez, E., García-Fernández, C., Fages, B. D. L. C., Coto, E., & Álvarez, V. (2024). NFKB1 variants were associated with the risk of Parkinson´ s disease in male. *Journal of Neural Transmission*, *131*(7), 773–779.

Perna, M., & Hewlings, S. (2022). Saturated fatty acid chain length and risk of cardiovascular disease: A systematic review. *Nutrients*, *15*(1), 30.

Phillips, C. M., Chen, L. W., Heude, B., Bernard, J. Y., Harvey, N. C., Duijts, L., Mensink-Bout, S. M., Polanska, K., Mancano, G., Suderman, M., & Shivappa, N. (2019). Dietary inflammatory index and non-communicable disease risk: A narrative review. *Nutrients*, *11*(8), 1873.

Piccolis, M., Bond, L. M., Kampmann, M., Pulimeno, P., Chitraju, C., Jayson, C. B., Vaites, L. P., Boland, S., Lai, Z. W., Gabriel, K. R., & Elliott, S. D. (2019). Probing the global cellular responses to lipotoxicity caused by saturated fatty acids. *Molecular Cell*, *74*(1), 32–44.

Pickett, J. R., Wu, Y., & Ta, H. T. (2025). VCAM-1 as a common biomarker in inflammatory bowel disease and colorectal cancer: Unveiling the dual anti-inflammatory and anti-cancer capacities of anti-VCAM-1 therapies. *Cancer and Metastasis Reviews*, *44*(2), 40.

Pizzino, G., Irrera, N., Cucinotta, M., Pallio, G., Mannino, F., Arcoraci, V., Squadrito, F., Altavilla, D., & Bitto, A. (2017). Oxidative stress: Harms and benefits for human health. *Oxidative Medicine and Cellular Longevity*.

Polley, K. R., Miller, M. K., Johnson, M., Vaughan, R., Paton, C. M., & Cooper, J. A. (2018). Metabolic responses to high-fat diets rich in MUFA v. *PUFA. British Journal of Nutrition*, *120*(1), 13–22.

Portegies, M. L. P., Koudstaal, P. J., & Ikram, M. A. (2016). Cerebrovascular disease. *Handbook of clinical neurology*, *138*, 239–261.

Posada, L. R., Shi, J., Kakuda, Y., & Xue, S. J. (2007). Extraction of tocotrienols from palm fatty acid distillates using molecular distillation. *Separation and Purification Technology*, *57*(2), 220–229.

Pothuraju, R., Rachagani, S., Krishn, S. R., Chaudhary, S., Nimmakayala, R. K., Siddiqui, J. A., Ganguly, K., Lakshmanan, I., Cox, J. L., Mallya, K., & Kaur, S. (2020). Molecular implications of MUC5AC-CD44 axis in colorectal cancer progression and chemoresistance. *Molecular Cancer*, *19*(1), 37.

Prasad, M., Rajagopal, P., Devarajan, N., Veeraraghavan, V. P., Palanisamy, C. P., Cui, B., Patil, S., & Jayaraman, S. (2022). A comprehensive review on high-fat diet-induced diabetes mellitus: An epigenetic view. *The Journal of Nutritional Biochemistry*, *107*, 109037.

Prescott, J. A., Mitchell, J. P., & Cook, S. J. (2021). Inhibitory feedback control of NF-κB signalling in health and disease. *Biochemical Journal*, *478*(13), 2619–2664.

Prescott, S. L., D'Adamo, C. R., Holton, K. F., Ortiz, S., Overby, N., & Logan, A. C. (2023). Beyond plants: The ultra-processing of global diets is harming the health of people, places, and planet. *International Journal of Environmental Research and Public Health*, *20*(15), 6461.

Prodöhl, I. (2023). *Globalizing the Soybean: Fat, Feed, and Sometimes Food, c*. Taylor & Francis.

Public Health England (PHE). (2019). *Saturated fats and health: SACN report.* Available at: Available at: https://www.gov.uk/government/publications/saturated-fats-and-health-sacn-report

Public Health England (PHE). (2021) *Composition of foods integrated dataset (CoFID).* Available at: https://www.gov.uk/government/publications/composition-of-foods-integrated-dataset-cofid

Pye, O., Cramb, R., & McCarthy, J. F. (2016). Deconstructing the roundtable on sustainable palm oil. In *The oil palm complex: Smallholders, agribusiness and the state in Indonesia and Malaysia* (pp. 409–411).

Pyo, Y., & Kwon, K. H. (2025). Therapeutic potential of red palm oil as antioxidant for men's health. *Men's Health*, *21*, 5-10.

Qaid, M. M., & Abdelrahman, M. M. (2016). Role of insulin and other related hormones in energy metabolism—A review. *Cogent Food & Agriculture*, *2*(1), 1267691.

Qian, S., Wei, Z., Yang, W., Huang, J., Yang, Y., & Wang, J. (2022). The role of BCL-2 family proteins in regulating apoptosis and cancer therapy. *Frontiers in Oncology*, *12*, 985363.

Qian, Y. R., Zhao, Y. J., & Zhang, F. (2025). Protein palmitoylation: biological functions, disease, and therapeutic targets. *MedComm*, *6*(3), e70096.

Qiu, T., Yang, X., Wang, J., Pan, C., Chu, X., Xiong, J., Xie, J., Chang, Y., Wang, C., & Zhang, J. (2022). Obesity-induced elevated palmitic acid promotes inflammation and glucose

metabolism disorders through GPRs/NF-κB/KLF7 pathway. *Nutrition & Diabetes*, *12*(1), 23.

Quadram Institute. (2019). *UK Composition of Foods Integrated Dataset (CoFID) searchable website.* Available at: https://www.quadram.ac.uk/UKfoodcomposition

Quadram Institute. (2021). *Food Labelling Dataset 2021.* Available at: https://fnnbri.quadram.ac.uk/labelling/

Quinton, B. (1990). The greening of McDonalds. *Restaurants & Institutions*, *100*(32), 28–42.

Rader, D. J., & Hovingh, G. K. (2014). HDL and cardiovascular disease. *The Lancet*, *384*(9943), 618–625.

Rafaqat, S., Gluscevic, S., Mercantepe, F., Rafaqat, S., & Klisic, A. (2024). Interleukins: Pathogenesis in non-alcoholic fatty liver disease. *Metabolites*, *14*(3), 153.

Rahmania, H., Indrayanto, G., Windarsih, A., Fernando, D., Bakar, N. K. A., & Rohman, A. (2024). Review of the Application of Molecular Spectroscopic and Chromatographic-based Methods for Determination of Trans Fatty Acids in Food Products. *Applied Food Research*, 100657.

Raile, K., O'Connell, M., Galler, A., Werther, G., Kühnen, P., Krude, H., & Blankenstein, O. (2011). Diabetes caused by insulin gene (INS) deletion: Clinical characteristics of homozygous and heterozygous individuals. *European Journal of Endocrinology*, *165*(2), 255–260.

Rakhra, V., Galappaththy, S. L., Bulchandani, S., & Cabandugama, P. K. (2020). Obesity and the western diet: How we got here. *Missouri Medicine*, *117*(6), 536.

Rampersaud, A. M., Dunk, C. E., Lye, S. J., & Renaud, S. J. (2020). Palmitic acid induces inflammation in placental trophoblasts and impairs their migration toward smooth muscle cells through plasminogen activator inhibitor-1. *Molecular Human Reproduction*, *26*(11), 850–865.

Rand, M. L., Hennissen, A. A., & Hornstra, G. (1988). Effects of dietary palm oil on arterial thrombosis, platelet responses and platelet membrane fluidity in rats. *Lipids*, *23*(11), 1019–1023.

Ratnayake, W. N., & Galli, C. (2009). Fat and fatty acid terminology, methods of analysis and fat digestion and metabolism. *Annals of Nutrition & Metabolism*, *55*(1/3), 8–43.

Rauber, F., Costa Louzada, M. L., Steele, E. M., Rezende, L. F., Millett, C., Monteiro, C. A., & Levy, R. B. (2019). Ultra-processed foods and excessive free sugar intake in the UK: a nationally representative cross-sectional study. *BMJ Open*, *9*(10), 027546.

Raulin, A. C., Doss, S. V., Trottier, Z. A., Ikezu, T. C., Bu, G., & Liu, C. C. (2022). ApoE in Alzheimer's disease: Pathophysiology and therapeutic strategies. *Molecular Neurodegeneration*, *17*(1), 72.

Razolli, D. S., Araújo, T. M., Sant'Ana, M. R., Kirwan, P., Cintra, D. E., Merkle, F. T., & Velloso, L. A. (2020). Proopiomelanocortin processing in the hypothalamus is directly regulated by saturated fat: Implications for the development of obesity. *Neuroendocrinology*, *110*(1–2), 92–104.

Rebersek, M. (2021). Gut microbiome and its role in colorectal cancer. *BMC Cancer*, *21*(1), 1325.

Reichardt, F., Chassaing, B., Nezami, B. G., Li, G., Tabatabavakili, S., Mwangi, S., Uppal, K., Liang, B., Vijay-Kumar, M., Jones, D., & Gewirtz, A. T. (2017). Western diet induces colonic nitrergic myenteric neuropathy and dysmotility in mice via saturated fatty acid- and lipopolysaccharide-induced TLR4 signalling. *The Journal of Physiology*, *595*(5), 1831–1846.

Reis Menta, P. L., Umanets, A., Miyamoto, J. E., Siqueira, B. P., Sartoratto, A., Ignacio-Souza, L., Torsoni, A. S., Torsoni, M. A., Leal, R. F., Antunes, A. E. C., & Milanski, M. (2024).

Interesterified palm oil leads to distinct variation in the intestinal microbiota of mice. *Food Bioscience*, *62*, 105032.

Remig, V., Franklin, B., Margolis, S., Kostas, G., Nece, T., & Street, J. C. (2010). Trans fats in America: A review of their use, consumption, health implications, and regulation. *Journal of the American Dietetic Association*, *110*(4), 585–592.

Reynoso, R., Salgado, L. M., & Calderón, V. (2003). High levels of palmitic acid lead to insulin resistance due to changes in the level of phosphorylation of the insulin receptor and insulin receptor substrate-1. *Molecular and Cellular Biochemistry*, *246*(1), 155–162.

Riccardi, G., Giacco, R., & Rivellese, A. A. (2004). Dietary fat, insulin sensitivity and the metabolic syndrome. *Clinical Nutrition*, *23*(4), 447–456.

Ricchi, M., Odoardi, M. R., Carulli, L., Anzivino, C., Ballestri, S., Pinetti, A., Fantoni, L. I., Marra, F., Bertolotti, M., Banni, S., & Lonardo, A. (2009). Differential effect of oleic and palmitic acid on lipid accumulation and apoptosis in cultured hepatocytes. *Journal of Gastroenterology and Hepatology*, *24*(5), 830–840.

Rice Bradley, B. H. (2018). Dietary fat and risk for type 2 diabetes: A review of recent research. *Current Nutrition Reports*, *7*, 214–226.

Rice, A. L., & Burns, J. B. (2010). Moving from efficacy to effectiveness: Red palm oil's role in preventing vitamin A deficiency. *Journal of the American College of Nutrition*, *29*(sup3), 302–313.

Richter, M., Boeing, H., Grünewald-Funk, D., Heseker, H., Kroke, A., Leschik-Bonnet, E., Oberritter, H., Strohm, D., & Watzl, B. (2016). Vegan diet. Position of the German nutrition society (DGE. *Ernahrungs Umschau*, *63*(04), 92–102.

Rinella, M. E., Lazarus, J. V., Ratziu, V., Francque, S. M., Sanyal, A. J., Kanwal, F., Romero, D., Abdelmalek, M. F., Anstee, Q. M., Arab, J. P., & Arrese, M. (2023). A multi-society

Delphi consensus statement on new fatty liver disease nomenclature. *Annals of Hepatology*, 101133.

Rinninella, E., Raoul, P., Cintoni, M., Franceschi, F., Miggiano, G. A. D., Gasbarrini, A., & Mele, M. C. (2019). What is the healthy gut microbiota composition? A changing ecosystem across age, environment, diet, and diseases. *Microorganisms*, *7*(1), 14.

Roche, H. M. (1999). Unsaturated fatty acids. *Proceedings of the Nutrition Society*, *58*(2), 397–401.

Rochlani, Y., Pothineni, N. V., Kovelamudi, S., & Mehta, J. L. (2017). Metabolic syndrome: Pathophysiology, management, and modulation by natural compounds. *Therapeutic Advances in Cardiovascular Disease*, *11*(8), 215–225.

Röder, P. V., Wu, B., Liu, Y., & Han, W. (2016). Pancreatic regulation of glucose homeostasis. *Experimental & molecular medicine*, *48*(3), 219–219.

Rohm, T. V., Meier, D. T., Olefsky, J. M., & Donath, M. Y. (2022). Inflammation in obesity, diabetes, and related disorders. *Immunity*, *55*(1), 31–55.

Rohm, T. V., Meier, D. T., Olefsky, J. M., & Donath, M. Y. (2022). Inflammation in obesity, diabetes, and related disorders. *Immunity*, *55*(1), 31–55.

Rohrmann, S., & Linseisen, J. (2016). Processed meat: The real villain? *Proceedings of the Nutrition Society*, *75*(3), 233–241.

Rosa, P. M., Antoniassi, R., Freitas, S. C., Bizzo, H. R., Zanotto, D. L., Oliveira, M. F., & Castiglioni, V. B. R. (2009). Chemical composition of brazilian sunflower varieties/composición química de las variedades de girasol brasileñas/composition chimique de sortes de tournesol brésiliennes. *Helia*, *32*(50), 145–156.

Rosqvist, F., Iggman, D., Kullberg, J., Cedernaes, J., Johansson, H. E., Larsson, A., Johansson, L., Ahlström, H., Arner, P., Dahlman, I., & Risérus, U. (2014). Overfeeding

polyunsaturated and saturated fat causes distinct effects on liver and visceral fat accumulation in humans. *Diabetes*, *63*(7), 2356–2368.

Rowlands, G., Protheroe, J., Winkley, J., Richardson, M., Seed, P. T., & Rudd, R. (2015). A mismatch between population health literacy and the complexity of health information: An observational study. *British Journal of General Practice*, *65*(635), 379–386.

Ruan, M., Bu, Y., Wu, F., Zhang, S., Chen, R., Li, N., Liu, Z., & Wang, H. (2021). Chronic consumption of thermally processed palm oil or canola oil modified gut microflora of rats. *Food Science and Human Wellness*, *10*(1), 94–102.

Ruderman, N. B., Williamson, J. R., & Brownlee, M. (1992). Glucose and diabetic vascular disease 1. *The FASEB Journal*, *6*(11), 2905–2914.

Rumora, A. E., LoGrasso, G., Hayes, J. M., Mendelson, F. E., Tabbey, M. A., Haidar, J. A., Lentz, S. I., & Feldman, E. L. (2019). The divergent roles of dietary saturated and monounsaturated fatty acids on nerve function in murine models of obesity. *Journal of Neuroscience*, *39*(19), 3770–3781.

Rustan, A. C., & Drevon, C. A. (2005). Fatty acids: Structures and properties. *Encyclopedia of Life Sciences*, *1*, 1–7.

Saadatian-Elahi, M., Norat, T., Goudable, J., & Riboli, E. (2004). Biomarkers of dietary fatty acid intake and the risk of breast cancer: A meta-analysis. *International Journal of Cancer*, *111*(4), 584–591.

Safari, Z., & Gérard, P. (2019). The links between the gut microbiome and non-alcoholic fatty liver disease (NAFLD. *Cellular and Molecular Life Sciences*, *76*(8), 1541–1558.

Sakurai, Y., Kubota, N., Yamauchi, T., & Kadowaki, T. (2021). Role of insulin resistance in MAFLD. *International Journal of Molecular Sciences*, *22*(8), 4156.

Sales-Campos, H., Souza, P., Crema Peghini, B., Silva, J., & Ribeiro Cardoso, C. (2013). An overview of the modulatory effects of oleic acid in health and disease. *Mini Reviews in Medicinal Chemistry*, *13*(2), 201–210.

Sales, R. C., Medeiros, P. C., Spreafico, F., Velasco, P. C., Gonçalves, F. K., Martín-Hernández, R., Mantilla-Escalante, D. C., Gil-Zamorano, J., Peres, W. A., Souza, S. A., & Dávalos, A. (2018). Olive oil, palm oil, and hybrid palm oil distinctly modulate liver transcriptome and induce NAFLD in mice fed a high-fat diet. *International Journal of Molecular Sciences*, *20*(1), 8.

Sampaio, K. A., Ceriani, R., Silva, S. M., Taham, T., & Meirelles, A. J. (2011). Steam deacidification of palm oil. *Food and Bioproducts Processing*, *89*(4), 383–390.

Sanchez-Muniz, F. J., Merinero, M. C., Rodríguez-Gil, S., Ordovas, J. M., Ródenas, S., & Cuesta, C. (2002). Dietary fat saturation affects apolipoprotein AII levels and HDL composition in postmenopausal women. *The Journal of Nutrition*, *132*(1), 50–54.

Sanders, T. A. B. (2024). 1—Introduction: The role of fats in human diet. In T. A. B. Sanders (Ed.), *Functional Dietary Lipids (Second Edition)* (Second Edition, pp. 1–28). Woodhead Publishing.

Sanders, T. A., Filippou, A., Berry, S. E., Baumgartner, S., & Mensink, R. P. (2011). Palmitic acid in the sn-2 position of triacylglycerols acutely influences postprandial lipid metabolism123. *The American Journal of Clinical Nutrition*, *94*(6), 1433–1441.

Santa-María, C., López-Enríquez, S., Paz, S., Geniz, I., Reyes-Quiroz, M. E., Moreno, M., Palomares, F., Sobrino, F., & Alba, G. (2023). Update on anti-inflammatory molecular mechanisms induced by oleic acid. *Nutrients*, *15*(1), 224.

Sara, P. (2018). Cardiovascular Disease (CVD): The Overview. *Inosr Applied Sciences*, *4*(1).

Saraswathi, V., Kumar, N., Ai, W., Gopal, T., Bhatt, S., Harris, E. N., Talmon, G. A., & Desouza, C. V. (2022). Myristic acid supplementation aggravates high fat diet-induced adipose inflammation and systemic insulin resistance in mice. *Biomolecules*, *12*(6), 739.

Saraswathi, V., Kumar, N., Gopal, T., Bhatt, S., Ai, W., Ma, C., Talmon, G. A., & Desouza, C. (2020). Lauric acid versus palmitic acid: Effects on adipose tissue inflammation, insulin resistance, and non-alcoholic fatty liver disease in obesity. *Biology*, *9*(11), 346.

Saraswathi, V., Kumar, N., Gopal, T., Bhatt, S., Ai, W., Ma, C., Talmon, G. A., & Desouza, C. (2020). Lauric acid versus palmitic acid: Effects on adipose tissue inflammation, insulin resistance, and non-alcoholic fatty liver disease in obesity. *Biology*, *9*(11), 346.

Sarhan, M. B., Fujiya, R., Kiriya, J., Htay, Z. W., Nakajima, K., Fuse, R., Wakabayashi, N., & Jimba, M. (2023). Health literacy among adolescents and young adults in the Eastern Mediterranean region: A scoping review. *BMJ Open*, *13*(6), 072787.

Schaffer, J. E. (2016). Lipotoxicity: Many roads to cell dysfunction and cell death: Introduction to a thematic review series. *Journal of Lipid Research*, *57*(8), 1327–1328.

Schependom, J., & D'haeseleer, M. (2023). Advances in neurodegenerative diseases. *Journal of Clinical Medicine*, *12*(5), 1709.

Scher, J. U., Ubeda, C., Artacho, A., Attur, M., Isaac, S., Reddy, S. M., Marmon, S., Neimann, A., Brusca, S., Patel, T., & Manasson, J. (2015). Decreased bacterial diversity characterizes the altered gut microbiota in patients with psoriatic arthritis, resembling dysbiosis in inflammatory bowel disease. *Arthritis & Rheumatology*, *67*(1), 128–139.

Schleifer, D. (2012). The perfect solution: How trans fats became the healthy replacement for saturated fats. *Technology and Culture*, *53*(1), 94–119.

Schlicker, E., & Kathmann, M. (2016). Role of the histamine H3 receptor in the central nervous system. *Histamine and histamine receptors in health and disease*, 277-299.

Schoeler, M., Ellero-Simatos, S., Birkner, T., Mayneris-Perxachs, J., Olsson, L., Brolin, H., Loeber, U., Kraft, J. D., Polizzi, A., Martí-Navas, M., & Puig, J. (2023). The interplay between dietary fatty acids and gut microbiota influences host metabolism and hepatic steatosis. *Nature Communications*, *14*(1), 5329.

Scholtz, S. C., Pieters, M., Oosthuizen, W., Jerling, J. C., Bosman, M. J., & Vorster, H. H. (2004). The effect of red palm olein and refined palm olein on lipids and haemostatic factors in hyperfibrinogenaemic subjects. *Thrombosis Research*, *113*(1), 13–25.

Schreiber, S., Nikolaus, S., & Hampe, J. (1998). Activation of nuclear factor κB in inflammatory bowel disease. *Gut*, *42*(4), 477–484.

Schwab, U. S., Maliranta, H. M., Sarkkinen, E. S., Savolainen, M. J., Kesäniemi, Y. A., & Uusitupa, M. I. (1996). Different effects of palmitic and stearic acid-enriched diets on serum lipids and lipoproteins and plasma cholesteryl ester transfer protein activity in healthy young women. *Metabolism*, *45*(2), 143–149.

Schwab, U. S., Niskanen, L. K., Maliranta, H. M., Savolainen, M. J., Kesemi, Y. A., & Uusitupa, M. I. (1995). Lauric and palmitic acid-enriched diets have minimal impact on serum lipid and lipoprotein concentrations and glucose metabolism in healthy young women. *The Journal of Nutrition*, *125*(3), 466–473.

Sears, B., & Perry, M. (2015). The role of fatty acids in insulin resistance. *Lipids in Health and Disease*, *14*(1), 121.

Seiwert, N., Heylmann, D., Hasselwander, S., & Fahrer, J. (2020). Mechanism of colorectal carcinogenesis triggered by heme iron from red meat. *Biochimica et Biophysica Acta (BBA)-Reviews on Cancer*, *1873*(1), 188334.

Selmin, O. I., Papoutsis, A. J., Hazan, S., Smith, C., Greenfield, N., Donovan, M. G., Wren, S. N., Doetschman, T. C., Snider, J. M., Snider, A. J., & Chow, S. H. H. (2021). N-6 high

fat diet induces gut microbiome dysbiosis and colonic inflammation. *International Journal of Molecular Sciences*, *22*(13), 6919.

Semenova, E. I., Partevian, S. A., Shulskaya, M. V., Rudenok, M. M., Lukashevich, M. V., Baranova, N. M., Doronina, O. B., Doronina, K. S., Rosinskaya, A. V., Fedotova, E. Y., & Illarioshkin, S. N. (2023). Analysis of ADORA2A. *MTA1, PTGDS, PTGS2*, *2023*(1), 9412776.

Sepehri, Z., Kiani, Z., Nasiri, A. A., & Kohan, F. (2016). Toll-like receptor 2 and type 2 diabetes. *Cellular & Molecular Biology Letters*, *21*, 1–9.

Sergi, D., Luscombe-Marsh, N., Heilbronn, L. K., Birch-Machin, M., Naumovski, N., Proud, C. G., Abeywardena, M. Y., & O'Callaghan, N. (2021). The inhibition of metabolic inflammation by EPA is associated with enhanced mitochondrial fusion and insulin signaling in human primary myotubes. *The Journal of Nutrition*, *151*(4), 810–819.

Sergi, D., Luscombe-Marsh, N., Naumovski, N., Abeywardena, M., & O'Callaghan, N. (2021). Palmitic acid, but not lauric acid, induces metabolic inflammation, mitochondrial fragmentation, and a drop in mitochondrial membrane potential in human primary myotubes. *Frontiers in Nutrition*, *8*, 663838.

Seufert, A. L., & Napier, B. A. (2023). A new frontier for fat: Dietary palmitic acid induces innate immune memory. *Immunometabolism*, *5*(2), 00021.

Shah, S. R., Alweis, R., Najim, N. I., Dharani, A. M., Jangda, M. A., Shahid, M., Kazi, A. N., & Shah, S. A. (2017). Use of dark chocolate for diabetic patients: A review of the literature and current evidence. *Journal of Community Hospital Internal Medicine Perspectives*, *7*(4), 218–221.

Shahid, R., Shoker, M., Chu, L. M., Frehlick, R., Ward, H., & Pahwa, P. (2022). Impact of low health literacy on patients' health outcomes: A multicenter cohort study. *BMC Health Services Research*, *22*(1), 1148.

Shakil, M. H., Trisha, A. T., Rahman, M., Talukdar, S., Kobun, R., Huda, N., & Zzaman, W. (2022). Nitrites in cured meats, health risk issues, alternatives to nitrites: A review. *Foods*, *11*(21), 3355.

Shantha, N. C., & Napolitano, G. E. (1992). Gas chromatography of fatty acids. *Journal of Chromatography A*, *624*(1–2), 37–51.

Shekhar, N., Tyagi, S., Rani, S., & Thakur, A. K. (2023). Potential of capric acid in neurological disorders: An overview. *Neurochemical Research*, *48*(3), 697–712.

Shen, X., Miao, S., Zhang, Y., Guo, X., Li, W., Mao, X., & Zhang, Q. (2025). Stearic acid metabolism in human health and disease. *Clinical Nutrition*, *44*, 222–238.

Shi, F., Chowdhury, R., Sofianopoulou, E., Koulman, A., Sun, L., Steur, M., & Kaptoge, S. (2025). Association of circulating fatty acids with cardiovascular disease risk: Analysis of individual-level data in three large prospective cohorts and updated meta-analysis. *European Journal of Preventive Cardiology*, *32*(3), 233–246.

Shield, K. D., Parry, C., & Rehm, J. (2014). Chronic diseases and conditions related to alcohol use. *Alcohol Research: Current Reviews*, *35*(2), 155.

Shin, J. H., Kim, C. S., Cha, J., Kim, S., Lee, S., Chae, S., Chun, W. Y., & Shin, D. M. (2022). Consumption of 85% cocoa dark chocolate improves mood in association with gut microbial changes in healthy adults: A randomized controlled trial. *The Journal of Nutritional Biochemistry*, *99*, 108854.

Shin, S. (2022). Regulation of adipose tissue biology by long-chain fatty acids: Metabolic effects and molecular mechanisms. *Journal of Obesity & Metabolic Syndrome*, *31*(2), 147.

Shoelson, S. E., Lee, J., & Goldfine, A. B. (2006). Inflammation and insulin resistance. *The Journal of Clinical Investigation*, *116*(7), 1793–1801.

Shoji, H., Arai, H., Kakiuchi, S., Ito, A., Sato, K., Jinno, S., Takahashi, N., Masumoto, K., Yoda, H., & Shimizu, T. (2024). Infant Formula with 50% or More of Palmitic Acid

Bound to the sn-2 Position of Triacylglycerols Eliminate the Association between Formula-Feeding and the Increase of Fecal Palmitic Acid Levels in Newborns: An Exploratory Study. *Nutrients*, *16*(11), 1558.

Shrestha, N., Franco, E., Arvan, P., & Cnop, M. (2021). Pathological β-cell endoplasmic reticulum stress in type 2 diabetes: Current evidence. *Frontiers in Endocrinology*, *12*, 650158.

Shrivastava, A. K., & Pandey, A. (2013). Inflammation and rheumatoid arthritis. *Journal of Physiology and Biochemistry*, *69*, 335–347.

Shu, L., Matveyenko, A. V., Kerr-Conte, J., Cho, J. H., McIntosh, C. H., & Maedler, K. (2009). Decreased TCF7L2 protein levels in type 2 diabetes mellitus correlate with downregulation of GIP-and GLP-1 receptors and impaired beta-cell function. *Human Molecular Genetics*, *18*(13), 2388–2399.

Shukla, V. K. S. (1994). Milkfat in sugar and chocolate confectionery. In *Fats in food products* (pp. 255–276). Springer US.

Sidhu, S. R. K., Kok, C. W., Kunasegaran, T., & Ramadas, A. (2023). Effect of plant-based diets on gut microbiota: A systematic review of interventional studies. *Nutrients*, *15*(6), 1510.

Sidletskaya, K., Vitkina, T., & Denisenko, Y. (2020). The role of toll-like receptors 2 and 4 in the pathogenesis of chronic obstructive pulmonary disease. *International Journal of Chronic Obstructive Pulmonary Disease*, 1481–1493.

Siemons, L., Ten Klooster, P. M., Vonkeman, H. E., Riel, P. L., Glas, C. A., & Laar, M. A. (2014). How age and sex affect the erythrocyte sedimentation rate and C-reactive protein in early rheumatoid arthritis. *BMC Musculoskeletal Disorders*, *15*, 1–7.

Silva Figueiredo, P., Carla Inada, A., Marcelino, G., Maiara Lopes Cardozo, C., de Cássia Freitas, K., de Cássia Avellaneda Guimarães, R., ... & Aiko Hiane, P. (2017). Fatty acids

consumption: the role metabolic aspects involved in obesity and its associated disorders. *Nutrients*, *9*(10), 1158.

Silva-Gomes, R., Mapelli, S. N., Boutet, M. A., Mattiola, I., Sironi, M., Grizzi, F., Colombo, F., Supino, D., Carnevale, S., Pasqualini, F., & Stravalaci, M. (2022). Differential expression and regulation of MS4A family members in myeloid cells in physiological and pathological conditions. *Journal of Leukocyte Biology*, *111*(4), 817–836.

Silva, A., & Martínez, M. C. (2023). Spatial memory deficits in Alzheimer's disease and their connection to cognitive maps' formation by place cells and grid cells. *Frontiers in Behavioral Neuroscience*, *16*, 1082158.

Silva, P., Araújo, R., Lopes, F., & Ray, S. (2023). Nutrition and food literacy: Framing the challenges to health communication. *Nutrients*, *15*(22), 4708.

Silva, Y. P., Bernardi, A., & Frozza, R. L. (2020). The role of short-chain fatty acids from gut microbiota in gut-brain communication. *Frontiers in Endocrinology*, *11*, 25.

Simopoulos, A. P. (2008). The importance of the omega-6/omega-3 fatty acid ratio in cardiovascular disease and other chronic diseases. *Experimental Biology and Medicine*, *233*(6), 674–688.

Sinar, D. R., & Charles, L. G. (1983). Glucose is the major component controlling irregular spike activity after feeding in primates. *Gastroenterology*, *85*(6), 1319–1325.

Sinclair, J., Dillon, S., & Bottoms, L. (2022). Perceptions, beliefs and behaviors of nutritional and supplementary practices in inflammatory bowel disease. *Sport Sciences for Health*, *18*(4), 1301–1310.

Sindhu, S., Akhter, N., Kochumon, S., Thomas, R., Wilson, A., Shenouda, S., Tuomilehto, J., & Ahmad, R. (2018). Increased expression of the innate immune receptor TLR10 in obesity and type-2 diabetes: Association with ROS-mediated oxidative stress. *Cellular Physiology and Biochemistry*, *45*(2), 572–590.

Sindhu, S., Al-Roub, A., Koshy, M., Thomas, R., & Ahmad, R. (2016). Palmitate-induced MMP-9 expression in the human monocytic cells is mediated through the TLR4-MyD88 dependent mechanism. *Cellular Physiology and Biochemistry*, *39*(3), 889–900.

Singh, B., Goyal, A. and Patel, B.C. (2025). *C-reactive protein: clinical relevance and interpretation.* Available at: https://www.ncbi.nlm.nih.gov/sites/books/NBK441843/

Singh, R. P. (2014). Cost effectiveness of palm oil in comparison to other oils and fats in the country with special emphasis on lower income group. *Indian Journal of Community Health*, *26*(Supp 1), 45–53.

Singh, S., Sharma, P., Sarma, D. K., Kumawat, M., Tiwari, R., Verma, V., Nagpal, R., & Kumar, M. (2023). Implication of obesity and gut microbiome dysbiosis in the etiology of colorectal cancer. *Cancers*, *15*(6), 1913.

Siopis, G., Colagiuri, S., & Allman-Farinelli, M. (2021). People with type 2 diabetes report dietitians, social support, and health literacy facilitate their dietary change. *Journal of Nutrition Education and Behavior*, *53*(1), 43–53.

Siri-Tarino, P. W., Sun, Q., Hu, F. B., & Krauss, R. M. (2010). Saturated fatty acids and risk of coronary heart disease: Modulation by replacement nutrients. *Current Atherosclerosis Reports*, *12*, 384–390.

Sivakumar, S., Lieber, S., Librizzi, D., Keber, C., Sommerfeld, L., Finkernagel, F., Roth, K., Reinartz, S., Bartsch, J. W., Graumann, J., & Müller-Brüsselbach, S. (2023). Basal cell adhesion molecule promotes metastasis-associated processes in ovarian cancer. *Clinical and Translational Medicine*, *13*(1), 1176.

Sivasubramanian, B. P., Dave, M., Panchal, V., Saifa-Bonsu, J., Konka, S., Noei, F., Nagaraj, S., Terpari, U., Savani, P., Vekaria, P. H., & Venkata, V. S. (2023). Comprehensive review of red meat consumption and the risk of cancer. *Cureus*, *15*(9).

Skretteberg, P. T., Bodegård, J., Kjeldsen, S. E., Erikssen, G., Thaulow, E., Sandvik, L., & Erikssen, J. E. (2010). Interaction between inflammation and blood viscosity predicts cardiovascular mortality. *Scandinavian Cardiovascular Journal*, *44*(2), 107–112.

Snook, J. T., Park, S., Williams, G., Tsai, Y. H., & Lee, N. (1999). Effect of synthetic triglycerides of myristic, palmitic, and stearic acid on serum lipoprotein metabolism. *European Journal of Clinical Nutrition*, *53*(8), 597–605.

Soares, J. B., Pimentel-Nunes, P., Roncon-Albuquerque, R., & Leite-Moreira, A. (2010). The role of lipopolysaccharide/toll-like receptor 4 signaling in chronic liver diseases. *Hepatology International*, *4*, 659–672.

Soliman, M. (2018). Cancer Causing Chemicals. In *Cancer Causing Substances*.

Sørensen, K., Pelikan, J. M., Röthlin, F., Ganahl, K., Slonska, Z., Doyle, G., Fullam, J., Kondilis, B., Agrafiotis, D., Uiters, E., & Falcon, M. (2015). Health literacy in Europe: Comparative results of the European health literacy survey (HLS-EU. *The European Journal of Public Health*, *25*(6), 1053–1058.

Souganidis, E., Laillou, A., Leyvraz, M., & Moench-Pfanner, R. (2013). A comparison of retinyl palmitate and red palm oil β-carotene as strategies to address vitamin A deficiency. *Nutrients*, *5*(8), 3257-3271.

Spady, D. K., Woollett, L. A., & Dietschy, J. M. (1993). Regulation of plasma LDL-cholesterol levels by dietary cholesterol and fatty acids. *Annual Review of Nutrition*, *13*(1), 355–381.

Spiazzi, B. F., Duarte, A. C., Zingano, C. P., Teixeira, P. P., Amazarray, C. R., Merello, E. N., Wayerbacher, L. F., Farenzena, L. P., Correia, P. E., Bertoluci, M. C., & Gerchman, F. (2023). Coconut oil: An overview of cardiometabolic effects and the public health burden of misinformation. *Archives of Endocrinology and Metabolism*, *67*(6), 000641.

Spronk, I., Kullen, C., Burdon, C., & O'Connor, H. (2014). Relationship between nutrition knowledge and dietary intake. *British Journal of Nutrition*, *111*(10), 1713–1726.

Sproston, N. R., & Ashworth, J. J. (2018). Role of C-reactive protein at sites of inflammation and infection. *Frontiers in Immunology*, *9*, 754.

Srour, B., Fezeu, L. K., Kesse-Guyot, E., Allès, B., Méjean, C., Andrianasolo, R. M., Chazelas, E., Deschasaux, M., Hercberg, S., Galan, P., Monteiro, C. A., Julia, C., & Touvier, M. (2019). Ultra-processed food intake and risk of cardiovascular disease: prospective cohort study (NutriNet-Santé). *BMJ (Clinical research ed.)*, *365*, l1451.

Stamler, J., Daviglus, M. L., Garside, D. B., Dyer, A. R., Greenland, P., & Neaton, J. D. (2000). Relationship of baseline serum cholesterol levels in 3 large cohorts of younger men to long-term coronary, cardiovascular, and all-cause mortality and to longevity. *Jama*, *284*(3), 311–318.

Starnes, T., Broxmeyer, H. E., Robertson, M. J., & Hromas, R. (2002). Cutting edge: IL-17D, a novel member of the IL-17 family, stimulates cytokine production and inhibits hemopoiesis. *The Journal of Immunology*, *169*(2), 642–646.

Statista. (2025) *Production volume of palm oil worldwide from 2012/13 to 2024/25.* Available at: https://www.statista.com/statistics/613471/palm-oil-production-volume-worldwide/

Steer, T., Carpenter, H., Tuohy, K., & Gibson, G. R. (2000). Perspectives on the role of the human gut microbiota and its modulation by pro-and prebiotics. *Nutrition Research Reviews*, *13*(2), 229–254.

Stein, C. J., & Colditz, G. A. (2004). Modifiable risk factors for cancer. *British Journal of Cancer*, *90*(2), 299–303.

Steinberg, D. (2013). In celebration of the 100th anniversary of the lipid hypothesis of atherosclerosis. *Journal of Lipid Research*, *54*(11), 2946–2949.

Steinberg, M. B., Schmelzer, A. C., Lin, P. N., & Garcia, G. (2010). Smoking as a chronic disease. *Current Cardiovascular Risk Reports*, *4*, 413–420.

Stern, M. C., Barnoya, J., Elder, J. P., & Gallegos-Carrillo, K. (2020). Diet, physical activity, obesity and related cancer risk: Strategies to reduce cancer burden in the Americas. *Salud Pública de México*, *61*, 448–455.

Sterne, J.A., Hernán, M.A., Reeves, B.C., Savović, J., Berkman, N.D., Viswanathan, M., Henry, D., Altman, D.G., Ansari, M.T., Boutron, I. and Carpenter, J.R., 2016. ROBINS-I: a tool for assessing risk of bias in non-randomised studies of interventions. *bmj*, *355*.

Sterne, J.A., Savović, J., Page, M.J., Elbers, R.G., Blencowe, N.S., Boutron, I., Cates, C.J., Cheng, H.Y., Corbett, M.S., Eldridge, S.M. and Emberson, J.R., 2019. RoB 2: a revised tool for assessing risk of bias in randomised trials. *bmj*, *366*.

Stewart, A. S., Pratt-Phillips, S., & Gonzalez, L. M. (2017). Alterations in intestinal permeability: The role of the "leaky gut" in health and disease. *Journal of Equine Veterinary Science*, *52*, 10–22.

Stojanov, S., Berlec, A., & Štrukelj, B. (2020). The influence of probiotics on the firmicutes/bacteroidetes ratio in the treatment of obesity and inflammatory bowel disease. *Microorganisms*, *8*(11), 1715.

Strable, M. S., & Ntambi, J. M. (2010). Genetic control of de novo lipogenesis: Role in diet-induced obesity. *Critical Reviews in Biochemistry and Molecular Biology*, *45*(3), 199–214.

Straczkowski, M., Kowalska, I., Nikolajuk, A., Dzienis-Straczkowska, S., Kinalska, I., Baranowski, M., Zendzian-Piotrowska, M., Brzezinska, Z., & Gorski, J. (2004). Relationship between insulin sensitivity and sphingomyelin signaling pathway in human skeletal muscle. *Diabetes*, *53*(5), 1215–1221.

Stuyvenberg. (1969). *Margarine: An economic, social and scientific history, 1869-1969* (J.H.). Liverpool University Press.

Subroto, E., & Nurannisa, R. L. (2020). The recent application of palm stearin in food industry: A review. *International Journal of Scientific & Technology Research*, *9*, 2593.

Suganami, T., Tanimoto-Koyama, K., Nishida, J., Itoh, M., Yuan, X., Mizuarai, S., Kotani, H., Yamaoka, S., Miyake, K., Aoe, S., & Kamei, Y. (2007). Role of the Toll-like receptor 4/NF-κB pathway in saturated fatty acid–induced inflammatory changes in the interaction between adipocytes and macrophages. *Arteriosclerosis, Thrombosis, and Vascular Biology*, *27*(1), 84–91.

Sulaiman, N. S., Sintang, M. D., Mantihal, S., Zaini, H. M., Munsu, E., Mamat, H., Kanagaratnam, S., Jahurul, M. H. A., & Pindi, W. (2022). Balancing functional and health benefits of food products formulated with palm oil as oil sources. *Heliyon*, *8*(10).

Šulinskaitė, K., Zagurskienė, D., & Blaževičienė, A. (2022). Patients' health literacy and health behaviour assessment in primary health care: Evidence from a cross-sectional survey. *BMC Primary Care*, *23*(1), 223.

Sun, G., Xia, H., Yang, Y., Ma, S., Zhou, H., Shu, G., Wang, S., Yang, X., Tang, H., Wang, F., & He, Y. (2018). Effects of palm olein and olive oil on serum lipids in a Chinese population: A randomized, double-blind, cross-over trial. *Asia Pacific Journal of Clinical Nutrition*, *27*(3), 572–580.

Sun, Y., Neelakantan, N., Wu, Y., Lote-Oke, R., Pan, A., & Dam, R. M. (2015). Palm oil consumption increases LDL cholesterol compared with vegetable oils low in saturated fat in a meta-analysis of clinical trials. *The Journal of Nutrition*, *145*(7), 1549–1558.

Sun, Y., Neelakantan, N., Wu, Y., Lote-Oke, R., Pan, A., & van Dam, R. M. (2015). Palm oil consumption increases LDL cholesterol compared with vegetable oils low in saturated fat in a meta-analysis of clinical trials. *The Journal of nutrition*, *145*(7), 1549-1558.

Sundram, K., French, M. A., & Clandinin, M. T. (2003). Exchanging partially hydrogenated fat for palmitic acid in the diet increases LDL-cholesterol and endogenous cholesterol

synthesis in normocholesterolemic women. *European Journal of Nutrition*, *42*(4), 188–194.

Sundram, K., Hayes, K. C., & Siru, O. H. (1994). Dietary palmitic acid results in lower serum cholesterol than does a lauric-myristic acid combination in normolipemic humans. *The American Journal of Clinical Nutrition*, *59*(4), 841–846.

Sundram, K., Hornstra, G., Houwelingen, A. C. V., & Kester, A. D. (1992). Replacement of dietary fat with palm oil: Effect on human serum lipids, lipoproteins and apolipoproteins. *British Journal of Nutrition*, *68*(3), 677–692.

Sundram, K., Ismail, A., Hayes, K. C., Jeyamalar, R., & Pathmanathan, R. (1997). Trans (elaidic) fatty acids adversely affect the lipoprotein profile relative to specific saturated fatty acids in humans. *The Journal of Nutrition*, *127*(3), 514–520.

Sundram, K., Karupaiah, T., & Hayes, K. C. (2007). Stearic acid-rich interesterified fat and trans-rich fat raise the LDL/HDL ratio and plasma glucose relative to palm olein in humans. *Nutrition & Metabolism*, *4*(1), 3.

Swaroop, J. J., Rajarajeswari, D., & Naidu, J. N. (2012). Association of TNF-α with insulin resistance in type 2 diabetes mellitus. *Indian Journal of Medical Research*, *135*(1), 127–130.

Syarifah-Noratiqah, S. B., Fairus, S., Zulfarina, M. S., Nasrullah, Z., Qodriyah, H. M. S., & Naina-Mohamed, I. (2020). The effects of palm oil on plasma and serum lipid parameters: A systematic review on animal intervention studies. *Frontiers in Veterinary Science*, *7*, 303.

Szabo, Z., Koczka, V., Marosvolgyi, T., Szabo, E., Frank, E., Polyak, E., Fekete, K., Erdelyi, A., Verzar, Z., & Figler, M. (2021). Possible biochemical processes underlying the positive health effects of plant-based diets—A narrative review. *Nutrients*, *13*(8), 2593.

Szabo, Z., Marosvölgyi, T., Szabo, E., Koczka, V., Verzar, Z., Figler, M., & Decsi, T. (2022). Effects of repeated heating on fatty acid composition of plant-based cooking oils. *Foods*, *11*(2), 192.

Tafti, Z. S. M., Moshiri, A., Marvasti, F. E., Tarashi, S., Khalili, S. F. S., Motahhary, A., Fateh, A., Vaziri, F., Badi, S. A., & Siadat, S. D. (2019). The effect of saturated and unsaturated fatty acids on the production of outer membrane vesicles from Bacteroides fragilis and Bacteroides thetaiotaomicron. *Gastroenterology and Hepatology from Bed to Bench*, *12*(2), 155.

Tan, C. H., Ariffin, A. A., Ghazali, H. M., Tan, C. P., Kuntom, A., & Choo, A. C. (2017). Changes in oxidation indices and minor components of low free fatty acid and freshly extracted crude palm oils under two different storage conditions. *Journal of Food Science and Technology*, *54*(7), 1757–1764.

Taylor, M. K., Sullivan, D. K., Ellerbeck, E. F., Gajewski, B. J., & Gibbs, H. D. (2019). Nutrition literacy predicts adherence to healthy/unhealthy diet patterns in adults with a nutrition-related chronic condition. *Public Health Nutrition*, *22*(12), 2157–2169.

Tchernof, A., & Després, J. P. (2013). Pathophysiology of Human Visceral Obesity: An Update. *Physiol Rev*, *93*, 359–404.

Teegala, S. M., Willett, W. C., & Mozaffarian, D. (2009). Consumption and health effects of trans fatty acids: A review. *Journal of AOAC International*, *92*(5), 1250–1257.

Temme, E. H., Mensink, R. P., & Hornstra, G. (1996). Comparison of the effects of diets enriched in lauric, palmitic, or oleic acids on serum lipids and lipoproteins in healthy women and men. *The American Journal of Clinical Nutrition*, *63*(6), 897–903.

Temme, E. H., Mensink, R. P., & Hornstra, G. (1997). Effects of medium chain fatty acids (MCFA), myristic acid, and oleic acid on serum lipoproteins in healthy subjects. *Journal of Lipid Research*, *38*(9), 1746–1754.

Teng, K. T., Nagapan, G., Cheng, H. M., & Nesaretnam, K. (2011). Palm olein and olive oil cause a higher increase in postprandial lipemia compared with lard but had no effect on plasma glucose, insulin and adipocytokines. *Lipids*, *46*(4), 381–388.

Teng, K. T., Voon, P. T., Cheng, H. M., & Nesaretnam, K. (2010). Effects of partially hydrogenated, semi-saturated, and high oleate vegetable oils on inflammatory markers and lipids. *Lipids*, *45*(5), 385–392.

Teng, S., Khong, K. W., & Ha, N. C. (2020). Palm oil and its environmental impacts: A big data analytics study. *Journal of Cleaner Production*, *274*, 122901.

Tennant, D., & Gosling, J. P. (2015). Modelling consumer intakes of vegetable oils and fats. *Food Additives & Contaminants: Part A*, *32*(9), 1397–1405.

Teres, S., Barceló-Coblijn, G., Benet, M., Alvarez, R., Bressani, R., Halver, J. E., & Escribá, P. V. (2008). Oleic acid content is responsible for the reduction in blood pressure induced by olive oil. *Proceedings of the National Academy of Sciences*, *105*(37), 13811–13816.

Tholstrup, T., Hjerpsted, J., & Raff, M. (2011). Palm olein increases plasma cholesterol moderately compared with olive oil in healthy individuals. *The American Journal of Clinical Nutrition*, *94*(6), 1426–1432.

Tholstrup, T., Marckmann, P., Jespersen, J., & Sandström, B. (1994). Fat high in stearic acid favorably affects blood lipids and factor VII coagulant activity in comparison with fats high in palmitic acid or high in myristic and lauric acids. *The American Journal of Clinical Nutrition*, *59*(2), 371–377.

Tholstrup, T., Marckmann, P., Jespersen, J., Vessby, B., Jart, A., & Sandström, B. (1994). Effect on blood lipids, coagulation, and fibrinolysis of a fat high in myristic acid and a fat high in palmitic acid. *The American Journal of Clinical Nutrition*, *60*(6), 919–925.

Tholstrup, T., Sandström, B., Bysted, A., & Hølmer, G. (2001). Effect of 6 dietary fatty acids on the postprandial lipid profile, plasma fatty acids, lipoprotein lipase, and cholesterol

ester transfer activities in healthy young men. *The American Journal of Clinical Nutrition*, *73*(2), 198–208.

Thursby, E., & Juge, N. (2017). Introduction to the human gut microbiota. *Biochemical Journal*, *474*(11), 1823–1836.

Tibble, J., Teahon, K., Thjodleifsson, B., Roseth, A., Sigthorsson, G., Bridger, S., Foster, R., Sherwood, R., Fagerhol, M., & Bjarnason, I. (2000). A simple method for assessing intestinal inflammation in Crohn's disease. *Gut*, *47*(4), 506–513.

Tilg, H., & Moschen, A. R. (2008). Inflammatory mechanisms in the regulation of insulin resistance. *Molecular medicine*, *14*, 222–231.

Tilg, H., Zmora, N., Adolph, T. E., & Elinav, E. (2020). The intestinal microbiota fuelling metabolic inflammation. *Nature Reviews Immunology*, *20*(1), 40–54.

Tishkowski, K., & Gupta, V. (2020). Erythrocyte sedimentation rate. *StatPearls Publishing, Treasure Island (FL).* Available at: https://europepmc.org/article/NBK/nbk557485

Tomarelli, R. M., Meyer, B. J., Weaber, J. R., & Bernhart, F. W. (1968). Effect of positional distribution on the absorption of the fatty acids of human milk and infant formulas. *The Journal of Nutrition*, *95*(4), 583–590.

Tomkin, G. H. (2014). Treatment of type 2 diabetes, lifestyle, GLP1 agonists and DPP4 inhibitors. *World Journal of Diabetes*, *5*(5), 636.

Traka, M. H., Plumb, J., Berry, R., Pinchen, H., & Finglas, P. M. (2020). Maintaining and updating food composition datasets for multiple users and novel technologies: Current challenges from a UK perspective. *Nutrition Bulletin*, *45*(2), 230–240.

Trautwein, E. A., & McKay, S. (2020). The role of specific components of a plant-based diet in management of dyslipidemia and the impact on cardiovascular risk. *Nutrients*, *12*(9), 2671.

Trevisan, M., Krogh, V., Freudenheim, J., Blake, A., Muti, P., Panico, S., Farinaro, E., Mancini, M., Menotti, A., & Ricci, G. (1990). Consumption of olive oil, butter, and vegetable oils and coronary heart disease risk factors. *Jama*, *263*(5), 688–692.

Truswell, A. S., Choudhury, N., & Roberts, C. K. (1999). Double blind comparison of plasma lipids in Healthy subjects eating potato crisps fried in palmolein of canola oil. *Journal of Food Technology in Africa*, *4*(2).

Tuccinardi, D., Mauro, A., Lattanzi, G., Rossini, G., Monte, L., Beato, I., Spiezia, C., Bravo, M., Watanabe, M., Soare, A., & Kyanvash, S. (2022). An extra virgin olive oil-enriched chocolate spread positively modulates insulin-resistance markers compared with a palm oil-enriched one in healthy young adults: A double-blind, cross-over, randomised controlled trial. *Diabetes/Metabolism Research and Reviews*, *38*(2), 3492.

Tuncay, C., & Ergoren, M. C. (2020). A systematic review of precision nutrition and Mediterranean Diet: A personalized nutrition approaches for prevention and management of obesity related disorders. *Clinical Nutrition ESPEN*, *38*, 61–64.

Turner-McGrievy, G., Mandes, T., & Crimarco, A. (2017). A plant-based diet for overweight and obesity prevention and treatment. *Journal of Geriatric Cardiology: JGC*, *14*(5), 369.

Tuso, P. J., Ismail, M. H., Ha, B. P., & Bartolotto, C. (2013). Nutritional update for physicians: Plant-based diets. *The Permanente Journal*, *17*(2), 61.

Tutunchi, H., Zolrahim, F., Nikbaf-Shandiz, M., Naeini, F., Ostadrahimi, A., Naghshi, S., Salek, R., & Najafipour, F. (2023). Effects of oleoylethanolamide supplementation on inflammatory biomarkers, oxidative stress and antioxidant parameters of obese patients with NAFLD on a calorie-restricted diet: A randomized controlled trial. *Frontiers in Pharmacology*, *14*, 1144550.

Tzeng, H. T., Chyuan, I. T., & Chen, W. Y. (2019). Shaping of innate immune response by fatty acid metabolite palmitate. *Cells*, *8*(12), 1633.

U.S Department of Agriculture (2018). *Vegetable oil, palm kernel.* Available at: https://fdc.nal.usda.gov/fdc-app.html#/food-details/171422/nutrients

UK Data Service. NatCen Social Research, University College London, Department of Epidemiology and Public Health. (2024). *Health Survey for England. 8th Release.* Available at: http://doi.org/10.5255/UKDA-Series-2000021

Unhapipatpong, C., Shantavasinkul, P. C., Kasemsup, V., Siriyotha, S., Warodomwichit, D., Maneesuwannarat, S., Vathesatogkit, P., Sritara, P., & Thakkinstian, A. (2021). Tropical oil consumption and cardiovascular disease: An umbrella review of systematic reviews and meta analyses. *Nutrients*, *13*(5), 1549.

United States Department of Agriculture. (2024). *Indonesia Palm Oil Production by Year.* Available at: https://www.indexmundi.com/agriculture/?country=id&commodity=palm-oil&graph=production

United States Department of Agriculture. (2024). *United Kingdom Palm Oil Imports by Year.* Available at: https://www.indexmundi.com/agriculture/?country=gb&commodity=palm-oil&graph=imports

United States National Toxicology Program Office of Health Assessment and Translation. (2019). *Handbook for Conducting a Literature-Based Health Assessment Using OHAT Approach for Systematic Review and Evidence Integration.* Available at: https://ntp.niehs.nih.gov/sites/default/files/ntp/ohat/pubs/handbookmarch2019_508.pdf

US Department of Agriculture (USDA). (2024). *Production - Palm Oil.* Available at: https://www.fas.usda.gov/data/production/commodity/4243000

Utarwuthipong, T., Komindr, S., Pakpeankitvatana, V., Songchitsomboon, S., & Thongmuang, N. (2009). Small dense low-density lipoprotein concentration and oxidative susceptibility changes after consumption of soybean oil, rice bran oil, palm oil and mixed rice bran/palm

oil in hypercholesterolaemic women. *Journal of International Medical Research*, *37*(1), 96–104.

Valdés-Ramos, R., & Benítez-Arciniega, A. D. (2007). Nutrition and immunity in cancer. *British Journal of Nutrition*, *98*(S1), 127–132.

Valk, R., Hammill, J., & Grip, J. (2022). Saturated fat: Villain and bogeyman in the development of cardiovascular disease? *European Journal of Preventive Cardiology*, *29*(18), 2312–2321.

Valsta, L. M., Tapanainen, H., & Männistö, S. (2005). Meat fats in nutrition. *Meat science*, *70*(3), 525–530.

Van Stuijvenberg, M. E., Dhansay, M. A., Lombard, C. J., Faber, M., & Benadé, A. J. S. (2001). The effect of a biscuit with red palm oil as a source of β-carotene on the vitamin A status of primary school children: a comparison with β-carotene from a synthetic source in a randomised controlled trial. *European journal of clinical nutrition*, *55*(8), 657-662.

Vanhaverbeke, M., Attard, R., Bartekova, M., Ben-Aicha, S., Brandenburger, T., Gonzalo-Calvo, D., Emanueli, C., Farrugia, R., Grillari, J., Hackl, M., Kalocayova, B., Martelli, F., Scholz, M., Wettinger, S. B., & Devaux, Y. (2022). Peripheral blood RNA biomarkers for cardiovascular disease from bench to bedside: A position paper from the EU-CardioRNA COST action CA17129. *Cardiovasc Res*, *29;118(16):3183-3197*.

Varela, M. L., Mogildea, M., Moreno, I., & Lopes, A. (2018). Acute inflammation and metabolism. *Inflammation*, *41*, 1115–1127.

Vazquez-Coto, D., Perez-Oliveira, S., Menéndez-González, M., Coto, E., & Álvarez, V. (2025). Assessing the association between common functional Nuclear Factor Kappa-b gene polymorphisms (NFKB1, NFKBIZ, NFKBIA) and Alzheimer´ s disease. *Behavioural Brain Research*, *476*, 115264.

Vega-López, S., Ausman, L. M., Jalbert, S. M., Erkkilä, A. T., & Lichtenstein, A. H. (2006). Palm and partially hydrogenated soybean oils adversely alter lipoprotein profiles compared with soybean and canola oils in moderately hyperlipidemic subjects. *The American Journal of Clinical Nutrition*, *84*(1), 54–62.

Vesga-Jiménez, D. J., Martin, C., Barreto, G. E., Aristizábal-Pachón, A. F., Pinzón, A., & González, J. (2022). Fatty acids: An insight into the pathogenesis of neurodegenerative diseases and therapeutic potential. *International Journal of Molecular Sciences*, *23*(5), 2577.

Vessby, B., Uusitupa, M., Hermansen, K., Riccardi, G., Rivellese, A. A., Tapsell, L. C., Nälsén, C., Berglund, L., Louheranta, A., Rasmussen, B. M., & Calvert, G. D. (2001). Substituting dietary saturated for monounsaturated fat impairs insulin sensitivity in healthy men and women: The KANWU Study. *Diabetologia*, *44*(3), 312–319.

Vilas-Boas, E. A., Almeida, D. C., Roma, L. P., Ortis, F., & Carpinelli, A. R. (2021). Lipotoxicity and β-cell failure in type 2 diabetes: Oxidative stress linked to NADPH oxidase and ER stress. *Cells*, *10*(12), 3328.

Visscher, B. B., Steunenberg, B., Heijmans, M., Hofstede, J. M., Devillé, W., Heide, I., & Rademakers, J. (2018). Evidence on the effectiveness of health literacy interventions in the EU: a systematic review. *BMC Public Health*, *18*, 1–12.

Vogler, M., Braun, Y., Smith, V. M., Westhoff, M. A., Pereira, R. S., Pieper, N. M., Anders, M., Callens, M., Vervliet, T., Abbas, M., & Macip, S. (2025). The BCL2 family: From apoptosis mechanisms to new advances in targeted therapy. *Signal Transduction and Targeted Therapy*, *10*(1), 91.

Voon, P. T., Lee, S. T., Ng, T. K. W., Ng, Y. T., Yong, X. S., Lee, V. K. M., & Ong, A. S. H. (2019). Intake of palm olein and lipid status in healthy adults: A meta-analysis. *Advances in Nutrition*, *10*(4), 647–659.

Voon, P. T., Ng, T. K. W., Lee, V. K. M., & Nesaretnam, K. (2011). Diets high in palmitic acid (16: 0), lauric and myristic acids (12: 0+ 14: 0), or oleic acid (18: 1) do not alter postprandial or fasting plasma homocysteine and inflammatory markers in healthy Malaysian adults. *The American Journal of Clinical Nutrition*, *94*(6), 1451–1457.

Wakil, S. J. (1961). Mechanism of fatty acid synthesis. *Journal of Lipid Research*, *2*(1), 1–24.

Walker, C. L. (1983). Implementing the NACNE report. III. The new British diet.

Wan Rosnani, A. I., Nor Aini Idris, N. A. I., & Noor Lida, H. M. D. (2008). Palm-based trans-free whipped toppings as an alternative to dairy cream.

Wan, J., Hu, S., Jacoby, J. J., Liu, J., Zhang, Y., & Yu, L. L. (2017). The impact of dietary sn-2 palmitic triacylglycerols in combination with docosahexaenoic acid or arachidonic acid on lipid metabolism and host faecal microbiota composition in Sprague Dawley rats. *Food & Function*, *8*(5), 1793–1802.

Wan, J., Hu, S., Ni, K., Chang, G., Sun, X., & Yu, L. (2016). Characterisation of fecal soap fatty acids, calcium contents, bacterial community and short-chain fatty acids in sprague dawley rats fed with different sn-2 palmitic triacylglycerols diets. *PLoS One*, *11*(10), 0164894.

Wanders, A. J., Zock, P. L., & Brouwer, I. A. (2017). Trans fat intake and its dietary sources in general populations worldwide: a systematic review. *Nutrients*, *9*(8), 840.

Wang, D., Karvonen-Gutierrez, C. A., Jackson, E. A., Elliott, M. R., Appelhans, B. M., Barinas-Mitchell, E., Bielak, L. F., Huang, M. H., & Baylin, A. (2020). Western dietary pattern derived by multiple statistical methods is prospectively associated with subclinical carotid atherosclerosis in midlife women. *The Journal of Nutrition*, *150*(3), 579–591.

Wang, L., Bravo-Ruiseco, G., Happe, R., He, T., Dijl, J. M., & Harmsen, H. J. (2021). The effect of calcium palmitate on bacteria associated with infant gut microbiota. *Microbiologyopen*, *10*(3), 1187.

Wang, L., Du, M., Wang, K., Khandpur, N., Rossato, S. L., Drouin-Chartier, J. P., Steele, E. M., Giovannucci, E., Song, M., & Zhang, F. F. (2022). Association of ultra-processed food consumption with colorectal cancer risk among men and women: Results from three prospective US cohort studies. *Bmj*, *378*(e068921), 1.

Wang, P. X., Deng, X. R., Zhang, C. H., & Yuan, H. J. (2020). Gut microbiota and metabolic syndrome. *Chinese Medical Journal*, *133*(7), 808–816.

Wang, T., Masedunskas, A., Willett, W. C., & Fontana, L. (2023). Vegetarian and vegan diets: Benefits and drawbacks. *European heart journal*, *44*(36), 3423–3439.

Wang, T., Yao, W., Li, J., He, Q., Shao, Y., & Huang, F. (2018). Acetyl-CoA from inflammation-induced fatty acids oxidation promotes hepatic malate-aspartate shuttle activity and glycolysis. *American Journal of Physiology-Endocrinology and Metabolism*, *315*(4), 496–510.

Wang, X., Ge, A., Cheng, M., Guo, F., Zhao, M., Zhou, X., Liu, L., & Yang, N. (2012). Increased hypothalamic inflammation associated with the susceptibility to obesity in rats exposed to high-fat diet. *Journal of Diabetes Research*.

Wang, X., Zhang, C., & Bao, N. (2023). Molecular mechanism of palmitic acid and its derivatives in tumor progression. *Frontiers in Oncology*, *13*, 1224125.

Wang, X., Zhong, Z., Chen, X., Hong, Z., Lin, W., Mu, X., Hu, X., & Zheng, H. (2021). High-fat diets with differential fatty acids induce obesity and perturb gut microbiota in honey bee. *International Journal of Molecular Sciences*, *22*(2), 834.

Wang, Z., Liu, D., Wang, F., Liu, S., Zhao, S., Ling, E. A., & Hao, A. (2012). Saturated fatty acids activate microglia via Toll-like receptor 4/NF-κB signalling. *British Journal of Nutrition*, *107*(2), 229–241.

Wareham, L. K., Liddelow, S. A., Temple, S., Benowitz, L. I., Polo, A., Wellington, C., Goldberg, J. L., He, Z., Duan, X., Bu, G., & Davis, A. A. (2022). Solving

neurodegeneration: Common mechanisms and strategies for new treatments. *Molecular neurodegeneration*, *17*(1), 23.

Wei, Y., Li, J., Li, J., Liu, C., Guo, X., Liu, Z., Zhang, L., Bao, S., Wu, X., Su, W., & Wang, X. (2024). Dietary long-chain fatty acids promote colitis by regulating palmitoylation of STAT3 through CD36-mediated endocytosis. *Cell Death & Disease*, *15*(1), 60.

Welch, R. W., & Mitchell, P. C. (2000). Food processing: A century of change. *British Medical Bulletin*, *56*(1), 1–17.

Wessely, S., Nickson, J., & Cox, B. (1990). Symptoms of low blood pressure: A population study. *British Medical Journal*, *301*(6748), 362–365.

Wieser, V., Moschen, A. R., & Tilg, H. (2013). Inflammation, cytokines and insulin resistance: A clinical perspective. *Archivum Immunologiae et Therapiae Experimentalis*, *61*, 119–125.

Wilcove, D. S., & Koh, L. P. (2010). Addressing the threats to biodiversity from oil-palm agriculture. *Biodiversity and Conservation*, *19*, 999–1007.

Wilcox, G. (2005). Insulin and insulin resistance. *Clinical Biochemist Reviews*, *26*(2), 19.

Willett, W. C. (2006). The Mediterranean diet: Science and practice. *Public Health Nutrition*, *9*(1a), 105–110.

Willett, W. C., Stampfer, M. J., Manson, J. E., Colditz, G. A., Speizer, F. E., Rosner, B. A., Hennekens, C. H., & Sampson, L. A. (1993). Intake of trans fatty acids and risk of coronary heart disease among women. *The Lancet*, *341*(8845), 581–585.

Wisdom, R., Johnson, R. S., & Moore, C. (1999). C-Jun regulates cell cycle progression and apoptosis by distinct mechanisms. *The EMBO Journal*, *18*(1), 188–197.

Wit, N., Derrien, M., Bosch-Vermeulen, H., Oosterink, E., Keshtkar, S., Duval, C., Bosch, J., Kleerebezem, M., Müller, M., & Meer, R. (2012). Saturated fat stimulates obesity and hepatic steatosis and affects gut microbiota composition by an enhanced overflow of

dietary fat to the distal intestine. *American Journal of Physiology-Gastrointestinal and Liver Physiology*, *303*(5), 589–599.

Wit, N., Derrien, M., Bosch-Vermeulen, H., Oosterink, E., Keshtkar, S., Duval, C., Bosch, J., Kleerebezem, M., Müller, M., & Meer, R. (2012). Saturated fat stimulates obesity and hepatic steatosis and affects gut microbiota composition by an enhanced overflow of dietary fat to the distal intestine. *American Journal of Physiology-Gastrointestinal and Liver Physiology*, *303*(5), 589–599.

Wlodarchak, N., & Xing, Y. (2016). PP2A as a master regulator of the cell cycle. *Critical Reviews in Biochemistry and Molecular Biology*, *51*(3), 162–184.

Wood, J. D., Richardson, R. I., Nute, G. R., Fisher, A. V., Campo, M. M., Kasapidou, E., Sheard, P. R., & Enser, M. (2004). Effects of fatty acids on meat quality: A review. *Meat Science*, *66*(1), 21–32.

Wood, R., Kubena, K., Tseng, S., Martin, G., & Crook, R. (1993). Effect of palm oil, margarine, butter, and sunflower oil on the serum lipids and lipoproteins of normocholesterolemic middle-aged men. *The Journal of Nutritional Biochemistry*, *4*(5), 286–297.

World Association of Zoos and Aquariums. (2025). *PalmOil Scan.* Available at: https://apps.apple.com/gb/app/palmoil-scan/id671945416

World Health Organization (2023). *Total fat intake for the prevention of unhealthy weight gain in adults and children: WHO guideline*. Available at: https://iris.who.int/handle/10665/375574

World Health Organization (WHO). (2006). *Definition and diagnosis of diabetes mellitus and intermediate hyperglycaemia.* Available at: https://www.who.int/publications/i/item/definition-and-diagnosis-of-diabetes-mellitus-and-intermediate-hyperglycaemia

World Health Organization (WHO). (2022). *Cancer: carcinogenicity of the consumption of red meat and processed meat.* Available at: https://www.who.int/news-room/questions-and-answers/item/cancer-carcinogenicity-of-the-consumption-of-red-meat-and-processed-meat

World Health Organization. (2022). *WHO European regional obesity report 2022*. Available at: https://www.who.int/europe/publications/i/item/9789289057738

World Health Organization. (2023). Saturated fatty acid and trans-fatty acid intake for adults and children: WHO guideline. In *Saturated fatty acid and trans-fatty acid intake for adults and children: WHO guideline*.

World Health Organization. (2023). Total fat intake for the prevention of unhealthy weight gain in adults and children: WHO guideline. In *Total fat intake for the prevention of unhealthy weight gain in adults and children: WHO guideline*.

World Heart Federation. (2023). *World Heart Report 2023: Full Report.* Available: https://world-heart-federation.org/resource/world-heart-report-2023/

Wu, D., Liu, J., Pang, X., Wang, S., Zhao, J., Zhang, X., & Feng, L. (2014). Palmitic acid exerts pro-inflammatory effects on vascular smooth muscle cells by inducing the expression of C-reactive protein, inducible nitric oxide synthase and tumor necrosis factor-α. *International Journal of Molecular Medicine*, *34*(6), 1706–1712.

Wu, S., Zhou, K., Misra-Hebert, A., Bena, J., & Kashyap, S. R. (2022). Impact of metabolic syndrome on severity of COVID-19 illness. *Metabolic Syndrome and Related Disorders*, *20*(4), 191–198.

Wu, W., Zhao, A., Liu, B., Ye, W. H., Su, H. W., Li, J., & Zhang, Y. M. (2021). Neurodevelopmental outcomes and gut bifidobacteria in term infants fed an infant formula containing high sn-2 palmitate: A cluster randomized clinical trial. *Nutrients*, *13*(2), 693.

Wuebben, E. L., & Rizzino, A. (2017). The dark side of SOX2: Cancer-a comprehensive overview. *Oncotarget*, *8*(27), 44917.

Xian, T. K., Omar, N. A., Ying, L. W., Hamzah, A., Raj, S., Jaarin, K., Othman, F., & Hussan, F. (2012). Reheated palm oil consumption and risk of atherosclerosis: Evidence at ultrastructural level. *Evidence-Based Complementary and Alternative Medicine*, *2012*(1), 828170.

Xie, T., Pei, Y., Shan, P., Xiao, Q., Zhou, F., Huang, L., & Wang, S. (2022). Identification of miRNA–mRNA Pairs in the Alzheimer's disease expression profile and explore the effect of miR-26a-5p/PTGS2 on amyloid-β induced neurotoxicity in Alzheimer's disease cell model. *Frontiers in Aging Neuroscience*, *14*, 909222.

Xin, F. Z., Zhao, Z. H., Zhang, R. N., Pan, Q., Gong, Z. Z., Sun, C., & Fan, J. G. (2020). Folic acid attenuates high-fat diet-induced steatohepatitis via deacetylase SIRT1-dependent restoration of PPARα. *World Journal of Gastroenterology*, *26*(18), 2203.

Xu, X., Xiao, L., Xiao, P., Yang, S., Chen, G., Liu, F., Kanwar, Y. S., & Sun, L. (2014). A glimpse of matrix metalloproteinases in diabetic nephropathy. *Current Medicinal Chemistry*, *21*(28), 3244–3260.

Yan, L., Hu, Y., D'Andrea, M. R., Towers, M. A., Wang, Y., Kesavan, P., DeWitte, M., Nakada, M. T., & Ahrendt, S. (2007). Association between high tumor CCL2 expression and poor survival in patients with resectable pancreatic cancer. *Journal of Clinical Oncology*, *25*(18_suppl), 4618–4618.

Yang, A. L., & McNabb-Baltar, J. (2020). Hypertriglyceridemia and acute pancreatitis. *Pancreatology*, *20*(5), 795–800.

Yang, C. L., Qiu, X., Lin, J. Y., Chen, X. Y., Zhang, Y. M., Hu, X. Y., Zhong, J. H., Tang, S., Li, X. Y., Xiang, B. D., & Zhang, Z. M. (2021). Potential role and clinical value of

PPP2CA in hepatocellular carcinoma. *Journal of Clinical and Translational Hepatology*, *9*(5), 661.

Yang, F., Zhang, S., Tian, M., Chen, J., Chen, F., & Guan, W. (2020). Different sources of high fat diet induces marked changes in gut microbiota of nursery pigs. *Frontiers in Microbiology*, *11*, 859.

Yang, M., Wei, D., Mo, C., Zhang, J., Wang, X., Han, X., Wang, Z., & Xiao, H. (2013). Saturated fatty acid palmitate-induced insulin resistance is accompanied with myotube loss and the impaired expression of health benefit myokine genes in C2C12 myotubes. *Lipids in Health and Disease*, *12*(1), 104.

Yao, D., Dong, M., Dai, C., & Wu, S. (2019). Inflammation and inflammatory cytokine contribute to the initiation and development of ulcerative colitis and its associated cancer. *Inflammatory Bowel Diseases*, *25*(10), 1595–1602.

Yao, M., Lien, E. L., Capeding, M. R., Fitzgerald, M., Ramanujam, K., Yuhas, R., Northington, R., Lebumfacil, J., Wang, L., & DeRusso, P. A. (2014). Effects of term infant formulas containing high sn-2 palmitate with and without oligofructose on stool composition, stool characteristics, and bifidogenicity. *Journal of Pediatric Gastroenterology and Nutrition*, *59*(4), 440–448.

Yao, Y., Cai, X., Ye, Y., Wang, F., Chen, F., & Zheng, C. (2021). The role of microbiota in infant health: From early life to adulthood. *Frontiers in Immunology*, *12*, 708472.

Yaron, S., Shachar, D., Abramas, L., Riskin, A., Bader, D., Litmanovitz, I., Bar-Yoseph, F., Cohen, T., Levi, L., Lifshitz, Y., & Shamir, R. (2013). Effect of high β-palmitate content in infant formula on the intestinal microbiota of term infants. *Journal of Pediatric Gastroenterology and Nutrition*, *56*(4), 376–381.

Yashodhara, B. M., Umakanth, S., Pappachan, J. M., Bhat, S. K., Kamath, R., & Choo, B. H. (2009). Omega-3 fatty acids: A comprehensive review of their role in health and disease. *Postgraduate Medical Journal*, *85*(1000), 84–90.

Ye, L., Mueller, O., Bagwell, J., Bagnat, M., Liddle, R. A., & Rawls, J. F. (2019). High fat diet induces microbiota-dependent silencing of enteroendocrine cells. *Elife*, *8*, 48479.

Ye, Z., Arumugam, V., Haugabrooks, E., Williamson, P., & Hendrich, S. (2015). Soluble dietary fiber (Fibersol-2) decreased hunger and increased satiety hormones in humans when ingested with a meal. *Nutrition Research*, *35*(5), 393–400.

Ye, Z., Cao, C., Li, Q., Xu, Y. J., & Liu, Y. (2020). Different dietary lipid consumption affects the serum lipid profiles, colonic short chain fatty acid composition and the gut health of Sprague Dawley rats. *Food Research International*, *132*, 109117.

Ye, Z., Xu, Y. J., & Liu, Y. (2021). Influence of different dietary oil consumption on nutrient malabsorption: An animal trial using Sprague Dawley rats. *Journal of Food Biochemistry*, *45*(4), 13695.

Yi, D. W., Jeong, D. W., Lee, S. Y., Son, S. M., & Kang, Y. H. (2012a). The association between apolipoprotein A-II and metabolic syndrome in Korean adults: A comparison study of apolipoprotein AI and apolipoprotein B. *Diabetes & Metabolism Journal*, *36*(1), 56.

Yi, D. W., Jeong, D. W., Lee, S. Y., Son, S. M., & Kang, Y. H. (2012b). The association between apolipoprotein A-II and metabolic syndrome in Korean adults: A comparison study of apolipoprotein AI and apolipoprotein B. *Diabetes & Metabolism Journal*, *36*(1), 56.

Yılmaz, B., & Ağagündüz, D. (2022). Fractionated palm oils: Emerging roles in the food industry and possible cardiovascular effects. *Critical Reviews in Food Science and Nutrition*, *62*(7), 1990–1998.

Yokoyama, Y., Levin, S. M., & Barnard, N. D. (2017). Association between plant-based diets and plasma lipids: A systematic review and meta-analysis. *Nutrition Reviews*, *75*(9), 683–698.

Yoon, H., Shaw, J. L., Haigis, M. C., & Greka, A. (2021). Lipid metabolism in sickness and in health: Emerging regulators of lipotoxicity. *Molecular Cell*, *81*(18), 3708–3730.

Yoshikawa, T., & Naito, Y. (2002). What is oxidative stress? *Japan Medical Association Journal*, *45*(7), 271–276.

Young, F. V. K. (1983). Palm kernel and coconut oils: Analytical characteristics, process technology and uses. *Journal of the American Oil Chemists' Society*, *60*(2Part2), 374–379.

Younossi, Z. M., Koenig, A. B., Abdelatif, D., Fazel, Y., Henry, L., & Wymer, M. (2016). Global epidemiology of nonalcoholic fatty liver disease—Meta-analytic assessment of prevalence, incidence, and outcomes. *Hepatology*, *64*(1), 73–84.

Younossi, Z. M., Stepanova, M., Younossi, Y., Golabi, P., Mishra, A., Rafiq, N., & Henry, L. (2020). Epidemiology of chronic liver diseases in the USA in the past three decades. *Gut*, *69*(3), 564–568.

Yu, D., Zhao, J., Wang, Y., Hu, J., Zhao, Q., Li, J., & Zhu, J. (2019). Upregulated histamine receptor H3 promotes tumor growth and metastasis in hepatocellular carcinoma. *Oncology Reports*, *41*(6), 3347–3354.

Yu, F., Huang, D., Kuang, Y., Dong, J., Han, Q., Zhou, J., & Teng, X. (2024). IRS1 promotes thyroid cancer metastasis through EMT and PI3K/AKT pathways. *Clinical Endocrinology*, *100*(3), 284–293.

Yuan, X., & Xu, D. (2019). Telomerase reverse transcriptase (TERT) in action: Cross-talking with epigenetics. *International journal of molecular sciences*, *20*(13), 3338.

Yue, G. H., Ye, B., & Suwanto, A. A. (2020). The unavoidable palm oil can be sustainable. *International Journal of Oil Palm*, *3*(2), 29–39.

Yuhas, R., Pramuk, K., & Lien, E. L. (2006). Human milk fatty acid composition from nine countries varies most in DHA. *Lipids*, *41*(9), 851–858.

Zafaryab, M., Fakhri, K. U., Khan, M. A., Hajela, K., & Rizvi, M. M. A. (2019). In vitro assessment of cytotoxic and apoptotic potential of palmitic acid for breast cancer treatment. *Int. J. Life Sci. Res*, *7*(1), 166–174.

Zainal, Z., Rahim, A. A., Radhakrishnan, A. K., Chang, S. K., & Khaza'ai, H. (2019). Investigation of the curative effects of palm vitamin E tocotrienols on autoimmune arthritis disease in vivo. *Scientific reports*, *9*(1), 1–11.

Zhang, H., Davies, K. J., & Forman, H. J. (2015). Oxidative stress response and Nrf2 signaling in aging. *Free Radical Biology and Medicine*, *88*, 314–336.

Zhang, J. M., & An, J. (2007). Cytokines, inflammation, and pain. *International Anesthesiology Clinics*, *45*(2), 27–37.

Zhang, J., Lu, Y., & Pienta, K. J. (2010). Multiple roles of chemokine (CC motif) ligand 2 in promoting prostate cancer growth. *Journal of the National Cancer Institute*, *102*(8), 522–528.

Zhang, J., Ping, W., Chunrong, W., Shou, C. X., & Keyou, G. (1997). Nonhypercholesterolemic Effects of a Palm Oil Diet in Chinese Adults123. *The Journal of Nutrition*, *127*(3), 509–513.

Zhang, K., Jiang, Y., Zeng, H., & Zhu, H. (2023). Application and risk prediction of thrombolytic therapy in cardio-cerebrovascular diseases: A review. *Thrombosis Journal*, *21*(1), 90.

Zhang, P., Yu, Y., Qin, Y., Zhou, Y., Tang, R., Wang, Q., Li, X., Wang, H., Weston-Green, K., Huang, X. F., & Zheng, K. (2019). Alterations to the microbiota–colon–brain axis in high-fat-diet-induced obese mice compared to diet-resistant mice. *The Journal of Nutritional Biochemistry*, *65*, 54–65.

Zhang, S., & Cui, W. (2014). Sox2, a key factor in the regulation of pluripotency and neural differentiation. *World Journal of Stem Cells*, *6*(3), 305.

Zhang, Y., Wang, S., Wang, C., Xiao, J., Zhang, S., & Zhou, H. (2019). High expression of FAM13A was associated with increasing the liver cirrhosis risk. *Molecular Genetics & Genomic Medicine*, *7*(3), 543.

Zhang, Z., Guan, B., Li, Y., He, Q., Li, X., & Zhou, L. (2021). Increased phosphorylated CREB1 protein correlates with poor prognosis in clear cell renal cell carcinoma. *Translational Andrology and Urology*, *10*(8), 3348.

Zhang, Z., Xing, X., Hensley, G., Chang, L. W., Liao, W., Abu-Amer, Y., & Sandell, L. J. (2010). Resistin induces expression of proinflammatory cytokines and chemokines in human articular chondrocytes via transcription and messenger RNA stabilization. *Arthritis & Rheumatism*, *62*(7), 1993–2003.

Zhao, H., Wu, L., Yan, G., Chen, Y., Zhou, M., Wu, Y., & Li, Y. (2021). Inflammation and tumor progression: Signaling pathways and targeted intervention. *Signal Transduction and Targeted Therapy*, *6*(1), 263.

Zhao, S., Wang, L., Hu, W., & Zheng, Y. (2023). Meet the meatless: Demand for new generation plant-based meat alternatives. *Applied Economic Perspectives and Policy*, *45*(1), 4–21.

Zhao, X., Zhang, W., Jiang, F., Chen, X., Chen, C., Wang, M., Chen, B., Cannon, R. D., Saffery, R., Han, T. L., & Zhang, H. (2024). Excessive palmitic acid disturbs macrophage α-ketoglutarate/succinate metabolism and causes adipose tissue insulin resistance associated with gestational diabetes mellitus. *Free Radical Biology and Medicine*, *222*, 424–436.

Zhao, Z., Shi, A., Wang, Q., & Zhou, J. (2019). High oleic acid peanut oil and extra virgin olive oil supplementation attenuate metabolic syndrome in rats by modulating the gut microbiota. *Nutrients*, *11*(12), 3005.

Zhao, Z., Wu, D., Gao, S., Zhou, D., Zeng, X., Yao, Y., Xu, Y., & Zeng, G. (2023). The association between dairy products consumption and prostate cancer risk: A systematic review and meta-analysis. *British Journal of Nutrition*, *129*(10), 1714–1731.

Zhao, Z., Zhong, L., Zhou, P., Deng, Y., Liu, G., Li, P., Zeng, J., Zhang, Y., Tang, X., & Zhang, M. (2024). Impact of Dietary Fatty Acid Composition on the Intestinal Microbiota and Fecal Metabolism of Rats Fed a High-Fructose/High-Fat Diet. *Nutrients*, *16*(21), 3774.

Zheng, D., Liwinski, T., & Elinav, E. (2020). Interaction between microbiota and immunity in health and disease. *Cell Research*, *30*(6), 492–506.

Zhou, B. R., Zhang, J. A., Zhang, Q., Permatasari, F., Xu, Y., Wu, D., Yin, Z. Q., & Luo, D. (2013). Palmitic Acid Induces Production of Proinflammatory Cytokines Interleukin-6, Interleukin-1β, and Tumor Necrosis Factor-α via a NF-κB-Dependent Mechanism in HaCaT Keratinocytes. *Mediators of Inflammation*, *2013*(1), 530429.

Zhou, H., & Chang, S. L. (2023). Meta-analysis of the effects of palmitic acid on microglia activation and neurodegeneration. *NeuroImmune Pharmacology and Therapeutics*, *2*(3), 281–291.

Zhou, L., Zhang, T., Zheng, M., Wang, S., Li, B., Hu, Z., Nie, Q., He, D., Hu, C., Zheng, J., & Lei, F. (2024). Physicochemical properties and flavor substances analyses of refined beef tallow with dry fractionation treatment. *Food Chemistry*, *460*, 140736.

Zhao, W., Wu, C., Li, S., & Chen, X. (2016). Adiponectin protects palmitic acid induced endothelial inflammation and insulin resistance via regulating ROS/IKKβ pathways. *Cytokine*, *88*, 167-176.

Zhou, Q., Fu, X., Dhital, S., Zhai, H., Huang, Q., & Zhang, B. (2021). In vitro fecal fermentation outcomes of starch-lipid complexes depend on starch assembles more than lipid type. *Food Hydrocolloids*, *120*, 106941.

Zhu, B., Zheng, S., Lin, K., Xu, X., Lv, L., Zhao, Z., & Shao, J. (2021). Effects of infant formula supplemented with prebiotics and OPO on infancy fecal microbiota: A pilot randomized clinical trial. *Frontiers in Cellular and Infection Microbiology*, *11*, 650407.

Zhu, G., Cheng, Z., Huang, Y., Zheng, W., Yang, S., Lin, C., & Ye, J. (2019). TRAF6 promotes the progression and growth of colorectal cancer through nuclear shuttle regulation NF-kB/c-jun signaling pathway. *Life Sciences*, *235*, 116831.

Zhu, M., Liu, X., Liu, W., Lu, Y., Cheng, J., & Chen, Y. (2021). β cell aging and age-related diabetes. *Aging (Albany NY*, *13*(5), 7691.

Zhu, S. R., Chong, F. F., & Xu, H. X. (2021). Cocoa flavanols intake and cognitive functions: A systematic review and meta-analysis of randomised controlled trials. *Journal of Nutritional Oncology*, *6*(1), 42–49.

Zhu, S., Jiao, W., Xu, Y., Hou, L., Li, H., Shao, J., Zhang, X., Wang, R., & Kong, D. (2021). Palmitic acid inhibits prostate cancer cell proliferation and metastasis by suppressing the PI3K/Akt pathway. *Life Sciences*, *286*, 120046.

Zhu, Y., Gao, X. M., Yang, J., Xu, D., Zhang, Y., Lu, M., Zhang, Z., Sheng, Y. Y., Li, J. H., Yu, X. X., & Zheng, Y. (2018). C-C chemokine receptor type 1 mediates osteopontin-promoted metastasis in hepatocellular carcinoma. *Cancer Science*, *109*(3), 710–723.

Ziemlińska, E., Sobocińska, J., Świątkowska, A., Hromada-Judycka, A., Traczyk, G., Malinowska, A., Świderska, B., Mietelska-Porowska, A., Ciesielska, A., & Kwiatkowska, K. (2021). Palm oil-rich diet affects murine liver proteome and S-palmitoylome. *International Journal of Molecular Sciences*, *22*(23), 13094.

Zock, P. L., Vries, J. H., & Katan, M. B. (1994). Impact of myristic acid versus palmitic acid on serum lipid and lipoprotein levels in healthy women and men. *Arteriosclerosis and Thrombosis: A Journal of Vascular Biology*, *14*(4), 567–575.

Zwierczyk, U., Sowada, C., & Duplaga, M. (2022). Eating choices—The roles of motivation and health literacy: A cross-sectional study. *Nutrients*, *14*(19), 4026.

Appendices

Appendix 1.1. Animal-based foods, plant-based foods and plant-based alternatives' fatty acid composition.

FAs [mg/g]	*Animal-based foods*			*Plant-based foods*			*Plant-based alternatives*		
	Beef Burger	Cheddar	Ice Cream	Olive oil	Peanut	Avocado	Burger	Cheese	Ice Cream
[1]Myristic	4.06	32.37	9.58	0.29	0.15	0.04	9.36	20.33	11.17
[1]Palmitic	37.82	80.53	14.34	119.25	48.12	19.32	7.65	13.12	14.40
[1]Stearic	27.65	30.05	10.89	38.42	11.49	0.53	2.50	3.41	8.67
[1]Oleic	37.36	52.92	13.27	843.64	159.28	68.74	10.82	9.54	15.15
[1]Linoleic	2.24	3.63	2.04	87.85	177.68	11.12	13.30	2.71	3.69
[1]α-Linolenic	0.72	1.76	0.12	8.19	0.32	0.98	0.18	0.06	0.30
[2]Saturated	**72.52**	**174.73**	**41.16**	166.30	86.48	20.31	**25.12**	**50.01**	**64.26**
[2]Unsaturated	67.62	65.92	15.71	**973.06**	**343.57**	**88.11**	24.47	12.49	19.26

[1]Predominant fatty acids. [2]Total (including fatty acids not displayed). Data compiled/sourced from Chapter 4.

Appendix 2.1. Prisma 2020 for Abstracts Checklist.

PRISMA 2020 for Abstracts Checklist

Section and Topic	Item #	Checklist item	Reported (Yes/No)
TITLE			
Title	1	Identify the report as a systematic review.	Yes
BACKGROUND			
Objectives	2	Provide an explicit statement of the main objective(s) or question(s) the review addresses.	Yes
METHODS			
Eligibility criteria	3	Specify the inclusion and exclusion criteria for the review.	Yes
Information sources	4	Specify the information sources (e.g. databases, registers) used to identify studies and the date when each was last searched.	Yes
Risk of bias	5	Specify the methods used to assess risk of bias in the included studies.	Yes
Synthesis of results	6	Specify the methods used to present and synthesise results.	Yes
RESULTS			
Included studies	7	Give the total number of included studies and participants and summarise relevant characteristics of studies.	Yes
Synthesis of results	8	Present results for main outcomes, preferably indicating the number of included studies and participants for each. If meta-analysis was done, report the summary estimate and confidence/credible interval. If comparing groups, indicate the direction of the effect (i.e. which group is favoured).	Yes
DISCUSSION			
Limitations of evidence	9	Provide a brief summary of the limitations of the evidence included in the review (e.g. study risk of bias, inconsistency and imprecision).	Yes
Interpretation	10	Provide a general interpretation of the results and important implications.	Yes
OTHER			

Section and Topic	Item #	Checklist item	Reported (Yes/No)
Funding	11	Specify the primary source of funding for the review.	N/A
Registration	12	Provide the register name and registration number.	Yes

From: Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 2021;372:n71. doi: 10.1136/bmj.n71. This work is licensed under CC BY 4.0. To view a copy of this license, visit https://creativecommons.org/licenses/by/4.0/

Appendix 2.2. Prisma 2020 for Systematic Reviews Checklist.

PRISMA 2020 Checklist

Section and Topic	Item #	Checklist item	Location where item is reported
TITLE			
Title	1	Identify the report as a systematic review.	Chapter title Thesis page 52
ABSTRACT			
Abstract	2	See the PRISMA 2020 for Abstracts checklist.	Chapter overview Thesis page 52
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of existing knowledge.	Introduction Thesis page 55
Objectives	4	Provide an explicit statement of the objective(s) or question(s) the review addresses.	Introduction Thesis page 55
METHODS			
Eligibility criteria	5	Specify the inclusion and exclusion criteria for the review and how studies were grouped for the syntheses.	Methodology Thesis pages 56, 57
Information sources	6	Specify all databases, registers, websites, organisations, reference lists and other sources searched or consulted to identify studies. Specify the date when each source was last searched or consulted.	Methodology Thesis page 56
Search strategy	7	Present the full search strategies for all databases, registers and websites, including any filters and limits used.	Methodology Thesis page 56
Selection process	8	Specify the methods used to decide whether a study met the inclusion criteria of the review, including how many reviewers screened each record and each report retrieved, whether they worked independently, and if applicable, details of automation tools used in the process.	Methodology Thesis pages 56, 57
Data collection process	9	Specify the methods used to collect data from reports, including how many reviewers collected data from each report, whether they worked independently, any processes for obtaining or confirming data from study investigators, and if applicable, details of automation tools used in the process.	Methodology Thesis page 56
Data items	10a	List and define all outcomes for which data were sought. Specify whether all results that were compatible with each outcome domain in each study were sought (e.g. for all measures, time points, analyses), and if not, the methods used to decide which results to collect.	Methodology Thesis pages 56, 57

Section and Topic	Item #	Checklist item	Location where item is reported
	10b	List and define all other variables for which data were sought (e.g. participant and intervention characteristics, funding sources). Describe any assumptions made about any missing or unclear information.	Methodology Thesis pages 56, 57
Study risk of bias assessment	11	Specify the methods used to assess risk of bias in the included studies, including details of the tool(s) used, how many reviewers assessed each study and whether they worked independently, and if applicable, details of automation tools used in the process.	Methodology Thesis pages 57, 58
Effect measures	12	Specify for each outcome the effect measure(s) (e.g. risk ratio, mean difference) used in the synthesis or presentation of results.	N/A
Synthesis methods	13a	Describe the processes used to decide which studies were eligible for each synthesis (e.g. tabulating the study intervention characteristics and comparing against the planned groups for each synthesis (item #5)).	Methodology Thesis pages 58, 59
	13b	Describe any methods required to prepare the data for presentation or synthesis, such as handling of missing summary statistics, or data conversions.	Methodology Thesis pages 58, 59
	13c	Describe any methods used to tabulate or visually display results of individual studies and syntheses.	Methodology Thesis pages 58, 59
	13d	Describe any methods used to synthesize results and provide a rationale for the choice(s). If meta-analysis was performed, describe the model(s), method(s) to identify the presence and extent of statistical heterogeneity, and software package(s) used.	Methodology, Summary of Findings Thesis pages 58, 59
	13e	Describe any methods used to explore possible causes of heterogeneity among study results (e.g. subgroup analysis, meta-regression).	N/A
	13f	Describe any sensitivity analyses conducted to assess robustness of the synthesized results.	N/A
Reporting bias assessment	14	Describe any methods used to assess risk of bias due to missing results in a synthesis (arising from reporting biases).	Methodology Thesis pages 57, 58
Certainty assessment	15	Describe any methods used to assess certainty (or confidence) in the body of evidence for an outcome.	N/A
RESULTS			
Study selection	16a	Describe the results of the search and selection process, from the number of records identified in the search to the number of studies included in the review, ideally using a flow diagram.	PRISMA flow diagram Thesis page 61
	16b	Cite studies that might appear to meet the inclusion criteria, but which were excluded, and explain why they were excluded.	Results Thesis pages 59, 61
Study characteristics	17	Cite each included study and present its characteristics.	Study characteristics tables Thesis pages 77 - 111
Risk of bias in studies	18	Present assessments of risk of bias for each included study.	Presence of bias Thesis pages 61 - 69

Section and Topic	Item #	Checklist item	Location where item is reported
Results of individual studies	19	For all outcomes, present, for each study: (a) summary statistics for each group (where appropriate) and (b) an effect estimate and its precision (e.g. confidence/credible interval), ideally using structured tables or plots.	N/A
Results of syntheses	20a	For each synthesis, briefly summarise the characteristics and risk of bias among contributing studies.	N/A
	20b	Present results of all statistical syntheses conducted. If meta-analysis was done, present for each the summary estimate and its precision (e.g. confidence/credible interval) and measures of statistical heterogeneity. If comparing groups, describe the direction of the effect.	N/A
	20c	Present results of all investigations of possible causes of heterogeneity among study results.	N/A
	20d	Present results of all sensitivity analyses conducted to assess the robustness of the synthesized results.	N/A
Reporting biases	21	Present assessments of risk of bias due to missing results (arising from reporting biases) for each synthesis assessed.	N/A
Certainty of evidence	22	Present assessments of certainty (or confidence) in the body of evidence for each outcome assessed.	N/A
DISCUSSION			
Discussion	23a	Provide a general interpretation of the results in the context of other evidence.	Discussion Thesis pages 112 - 116
	23b	Discuss any limitations of the evidence included in the review.	Discussion Thesis page 117
	23c	Discuss any limitations of the review processes used.	Discussion Thesis pages 117, 118
	23d	Discuss implications of the results for practice, policy, and future research.	Conclusions Thesis page 118
OTHER INFORMATION			
Registration and protocol	24a	Provide registration information for the review, including register name and registration number, or state that the review was not registered.	Validity statement Thesis page 55
	24b	Indicate where the review protocol can be accessed, or state that a protocol was not prepared.	Validity statement Thesis page 55
	24c	Describe and explain any amendments to information provided at registration or in the protocol.	N/A
Support	25	Describe sources of financial or non-financial support for the review, and the role of the funders or sponsors in the review.	Conflicting interests and funding statement Thesis page 118
Competing interests	26	Declare any competing interests of review authors.	Conflicting interests and funding statement Thesis page 118

Section and Topic	Item #	Checklist item	Location where item is reported
Availability of data, code and other materials	27	Report which of the following are publicly available and where they can be found: template data collection forms; data extracted from included studies; data used for all analyses; analytic code; any other materials used in the review.	N/A

From: Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 2021;372:n71. doi: 10.1136/bmj.n71. This work is licensed under CC BY 4.0. To view a copy of this license, visit https://creativecommons.org/licenses/by/4.0/

Appendix 3.1. Participant information sheet for the survey and food diary data collection.

Participant Information Sheet

University of Central Lancashire
UCLan

1. Title of Study
Palmitic acid (16:0) consumption in plant-based diets and its effect on metabolic health.

2. Document version
PIS_Exp2_v1 (16/11/2021); PIS_Exp2_v2 (28/02/2022)

3. Invitation Paragraph
You are being invited to take part in this study. Before you decide, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with friends, relatives and your GP if you wish. Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part.

Thank you for reading this.

4. What is the purpose of the study?
The widespread use of palm oil/palmitic acid, particularly in processed food products, suggests that palmitic acid (a saturated fatty acid) is consumed at an alarming rate. Growing evidence indicates that palmitic acid can induce inflammation associated with a range of chronic diseases. This warrants investigation of the health effects of palm oil/palmitic acid consumption particularly in plant-based dietary habits and omnivore diets. The purpose of this study is to assess the intake of palm oil/palmitic acid in a plant-based diet versus an omnivore diet.

5. Why have I been invited to take part?
The knowledge on palmitic acids' effects on human health is widely debated and its prevalence in plant-based diets is not well-documented. We hope to assess the health effect of consuming food containing palm oil/palmitic acid. In this study, we are expecting to collect data on your overall health and dietary habits, as well as knowledge on palm oil/palmitic acid. We welcome your participation in this study if you fulfil the following eligibility criteria-

1. No pre-existing disease conditions and should be in good health.
2. Be on your respective diet (plant-based/vegan or omnivore) for >3 months.
3. Must be aged 18+, any gender, any ethnicity.
4. Able to clearly understand/communicate (reading and writing) in English.
5. Must have access to a smartphone or computer with email functionality.
6. Must have access to a scale/balance (weight) and measuring tape (for height, waist and hip circumference), further guidance will be available on request
7. Must read and fully understood the procedure(s) and all information provided on the participant information sheet (PIS).
8. Must have read, fully understood and signed the participant consent form.

6. Do I have to take part?
It is entirely up to you whether to take part or not in this study. If you decide to take part you will be given this information sheet and be asked to sign a consent form. If you participate in this study, you are still free to withdraw at any stage without giving a reason.

7. What will happen if I take part?
As a participant in this study, you will need to have access to Microsoft Word and an email account:

1. Complete a questionnaire designed to collect some general information about you're your anthropometry, physical activity and lifestyle, dietary habits and awareness on palm oil.

2. Complete a diet diary stating about your dietary intake for a total of 12 days, including all meals, snacks and beverages (including alcohol):
    a. Week 1 - Monday, Wednesday, Friday and Saturday
    b. Week 6 - Monday, Wednesday, Friday and Saturday
    c. Week 12 - Monday, Wednesday, Friday and Saturday

3. It is expected that you have access to Microsoft Word in a computer or smartphone so that you can complete the questionnaire and diet diaries electronically. However, if you would like to receive a printed copy of the questionnaire/diet diaries, please do contact Thomas at THothersall1@uclan.ac.uk.

4. Following completion of the questionnaire and diet diaries, please send those electronically (as email attachment) to Thomas at THothersall1@uclan.ac.uk.

8. COVID-19

All participant interaction for this experiment will be held remotely via electronic media, with no face-to-face interactions. As such, there is no COVID-19 risk involved in this study.

9. How will my data be used?

The University processes personal data as part of its research and teaching activities in accordance with the lawful basis of 'public task', and in accordance with the University's purpose of "advancing education, learning and research for the public benefit".

Under UK data protection legislation, the University acts as the Data Controller for personal data collected as part of the University's research. The University privacy notice for research participants can be found on the attached link https://www.uclan.ac.uk/data_protection/privacy-notice-research-participants.php

Further information on how your data will be used can be found in the table below.

How will my data be collected?	Data will be collected through questionnaire(s) and diet diaries (via email).
How will my data be stored?	All digital data collected will be saved and stored on a password protected computer in Preston Campus of the University of Central Lancashire that has encryption and two-factor authentication facilities. The digital data will only be accessible by the lead researcher and supervisory team.
How long will my data be stored for?	All personal contact details will be retained for 28 days following results.
What measures are in place to protect the security and confidentiality of my data?	All data collected via email will be saved and stored, as well as password protected with two-factor authentication, via Microsoft OneDrive.
Will my data be anonymised?	*Yes.* All subjects will be assigned an identification number. This number will be used when discussing the information collected during the study and in any future publication of results.
How will my data be used?	This study involves collection of data relating to your nutritional intake, physical activity and sleeping habits and awareness and consumption of palm oil/palmitic acid.

	-Data collected will be analysed to assess the intake of palm oil/palmitic acid and its effect on metabolic health. -This study is expected to provide an estimation of palm oil/palmitic acid containing foods and these contribute to overweight/obesity etc.
Who will have access to my data?	Data collected during this research is purely for the scientific research and generation of information for public health purpose. The data will only be accessible by the lead researcher and supervisory team. No third-parties will have access to any data.
Will my data be archived for use in other research projects in the future?	Data collected in this study will provide the basis for further study of this PhD research. It is expected that following completion of the PhD project and publication of research papers, all data will be destroyed permanently.
How will my data be destroyed?	All personal contact details will be deleted on day 29 (following results).

10. Are there any risks in taking part?

It is unlikely that you might feel distress, discomfort or harm while participating in this study. However, in the unlikely event of any participants experiencing distress and/or any unhealthy behaviour/habit they may approach the lead researcher for further advice or directly approach the following for support-

Beat
0808 801 0677 (adult helpline)
0808 801 0711 (youthline)
0808 801 0811 (studentline)
beateatingdisorders.org.uk

11. Are there any benefits from taking part?

This study will explore palm oil/palmitic acid consumption in omnivore and plant-based diets and in addition may help to further our understanding of the role played by palm oil/palmitic acid in shaping human metabolic and gut health. There is no direct benefit for you to participate in this research. However, the conclusion drawn from this study is expected to help the health-conscious consumers whether to avoid excessive consumption of palm oil/palmitic acid containing food products.

12. Expenses and / or payments

No expense or payment will be made for participating in this research

13. What will happen to the results of the study?

The results of the study will be utilized to develop further follow up studies in this PhD project and is likely to provide scientific evidence on the health implication of consuming foods with palm oil/palmitic acid.

14. What will happen if I want to stop taking part?

If you change your mind, you can always withdraw at any time, however the time in which you do so has different effects on the data process.

Signing of the consent form (electronically) is considered as the basis for the participants willingness to participate in this study. However, if they wish, they may withdraw at various stages as follows-
After consent is given but prior to the start of data collection- No data will be collected from such participants.
During the data collection process- No data will be collected from such participants and any data already collected will be excluded in the study.
Participants have already supplied their response- The data will be excluded from the study until the stage of anonymization of the data. However, following the anonymization process when the tracing of the individual is not possible, the exclusion of the data will not be possible.

15. What if I am unhappy or if there is a problem?

*If you are unhappy, or if there is a problem, please feel free to let us know by contacting **Thomas Hothersall via thothersall1@uclan.ac.uk** and we will try to help. If you remain unhappy, or have a complaint which you feel you cannot come to us with, then please contact the Ethics, Integrity and Governance Unit at OfficerForEthics@uclan.ac.uk.*

The University strives to maintain the highest standards of rigour in the processing of your data. However, if you have any concerns about the way in which the University processes your personal data, it is important that you are aware of your right to lodge a complaint with the Information Commissioner's Office by calling 0303 123 1113.

Contact details of investigatory team

Researcher
Thomas Hothersall
Tel: 07500338638
Email: THothersall1@uclan.ac.uk

Director of Studies
Dr Bojlul Bahar
Email: bbahar@uclan.ac.uk

Supervisor(s)
Dr Stephanie Dillon
Email: sdillon@uclan.ac.uk

Dr Jonathan Sinclair
Email: jksinclair@uclan.ac.uk

Appendix 3.2. Participant consent form for the survey and food diary data collection.

University of Central Lancashire
UCLan

Participant Consent Form

Version number & date: Version 2.0, 8th February 2022
Research ethics approval number: HEALTH 0263 FR
Title of the research project: Palmitic acid (16:0) consumption in plant-based diets and its effect on metabolic health.
Name of researcher(s): Thomas Hothersall

Please initial box

1. I confirm that I have read and have understood the information sheet dated PIS_Exp2_v2 (28/02/2022) for the above study, or it has been read to me. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily. ☐
2. I understand that taking part in the study involves completion of a questionnaire designed to collect some general information about me, my anthropometry, physical activity and lifestyle, dietary habits and awareness on palm oil (over 12 weeks). ☐
3. I understand that my participation is voluntary and that I am free to stop taking part and can withdraw from the study at any time without giving any reason and without my rights being affected. In addition, I understand that I am free to decline to answer any particular question or questions. ☐

Choose appropriate option EITHER 4a or 4b

4. a) I understand that if I withdraw from this study data collected prior to my withdrawal will be retained but no further data will be collected.
   b) I understand that I can ask for access to the information I provide and I can request the destruction of that information if I wish at any time prior to completion and return of the documents to the researcher (Thomas Hothersall). I understand that following completion and return of the documents, I will no longer be able to request access to or withdrawal of the information I provide. ☐
5. I understand that the information I provide will be held securely and in line with data protection requirements at the University of Central Lancashire. ☐
6. I understand that signed consent forms and questionnaires, diet diaries will be retained in Microsoft OneDrive. Contact details will be held for 28 days following return of documents to the researcher, contact details will be deleted day 29. ☐

7. I agree to take part in the above study. ☐

Participant name	Date	Signature
Name of person taking consent	Date	Signature

Principal Investigator
Bojlul Bahar
DB204 Darwin Building
School of Sport & Health Sciences
University of Central Lancashire
Preston, Pr1 2HE
01 772 894914
bbahar@uclan.ac.uk

Student Investigator
Thomas Hothersall
07500338638
thothersall1@uclan.ac.uk

Open data and data sharing

I understand that the information I provide will be held securely and in line with data protection requirements at the University of Central Lancashire until it is fully anonymised and then deposited in Microsoft OneDrive for sharing and use by other authorised researchers to support other research in the future. ☐

I understand that other authorised researchers may use my words in publications, reports, webpages, and other research outputs, if their study has been approved by a research ethics committee, and they agree to preserve the confidentiality of the information as requested in this form. ☐

I understand that personal information collected about me that can identify me, such as my name or where I live, will not be shared beyond the study team. ☐

Risk to participants

I understand that taking part in the study has a potential risk of triggering distress that could potentially lead to unhealthy eating habits. I understand I can contact the lead researcher at any time, as well as the provided helpline below if need be. ☐

Beat
0808 801 0677 (adult helpline)
0808 801 0711 (youthline)
0808 801 0811 (studentline)
beateatingdisorders.org.uk

Confidentiality of the data

I understand that the research team will respect my confidentiality and I give permission for them to have access to my responses. ☐

I understand that confidentiality and anonymity will be maintained and it will not be possible to identify me in any reports, presentations or publications arising from the research will be numbered and anonymised. ☐

Disclosure of criminal activity and other confidentiality issues

I understand that the confidentiality of the information I provide will be safeguarded and won't be released without my consent unless I disclose information which raises ☐

concerns about the safety of myself or others. In such cases the researcher may be required to disclose my confidential information to the relevant authorities.

I agree that my information can be quoted in research outputs such as dissertation, scientific literature and research manuscripts as appropriate. ☐

I understand and agree that what I have said or written as part of this study will be used in reports, publications and other research outputs so that anything I have contributed to this project can be recognised. ☐

I agree that my real name can be used for communication with me for collection of data. ☐

Health-related findings in clinical research

I agree for my GP to be contacted if any unexpected results are found in relation to my health. ☐

I understood that once all results have been verified, I am allowed to contact via email by a member of the research team. This email will inform the aggregated group results of the study while no individual data will be available. ☐

I understood that if I fail to respond to the email within 28 days, it will be assumed that I don't wish to know the results and all my personal contact details will be deleted on day 29. ☐

Re-contacting participants for the purpose of inviting them to take part in future studies

I agree to being contacted at a later date and invited to take part in future relevant studies. I understand that I am only agreeing to receive information and I am under no obligation to take part in any future studies. I understand that if I do not consent to being contacted in the future it will not have any influence on my involvement in this particular research study. ☐

Appendix 3.3. Data protection checklist for the survey and intake study.

Legal and Governance: Data Protection Checklist

University of Central Lancashire UCLan

Data protection checklist: Teaching, research, knowledge transfer, consultancy and related activities

All activities which involve personal data of any kind, in any way, must comply with data protection legislation (the General Data Protection Regulation (GDPR) and Data Protection Act 2018 (DPA)). This checklist outlines the requirements of the data protection legislation and the measures you must take when processing personal data; it also provides a mechanism for recording the steps you will take to ensure the personal data you are using are safeguarded and the reputation of the University is upheld.

Ensuring personal data are processed transparently, fairly and lawfully with due regard for individuals' privacy and ensuring that personal data remain secure are paramount. Demonstrating that we have considered the requirements of data protection legislation when conducting our activities will provide assurances to students, employees, research participants and business partners that their personal data is protected at UCLan. Organisations can be fined up to €20 million or 4% of global annual turnover for breaches of the GDPR; therefore it is important that we get it right from the outset. If it is possible to use anonymised data so that individuals cannot be identified from it and still achieve your aims, this is always the preferred method of operating. Truly anonymised data (which cannot be reconstructed or linked to any other data you hold or may hold in the future to enable you to identify individuals from it) does not constitute personal data because it cannot be used to identify individuals.

Section 1: Does my activity involve personal data?

What is *personal data*?

Personal data is data relating to a living individual who can be identified from those data (or from those data and other information in our possession or likely to come into our possession).

Personal data can be factual (such as name, address, date of birth) or can be an opinion (such as a professional opinion as to the causes of an individual's behavioural problems or a response to a survey question). Voice recordings are personal data because the individual can be identified from his or her voice. Information can be personal data even if it does not include a person's name or other obvious identifiers. ***Example***: *a paragraph describing a specific event*

involving an individual or a set of characteristics relating to a particular individual may not include their name but would clearly identify them from the set of circumstances or characteristics being described or represented.

If you are unsure whether or not your activity involves personal data, please contact the Information Governance team to discuss on DPFOIA@uclan.ac.uk.

More information about personal data is available in the Data Protection Policy.

What is *processing*?

The GDPR is concerned with the processing of personal data. 'Processing' means any operation or set of operations which is performed on personal data or on sets of personal data, whether or not by automated means, such as collection, recording, organisation, structuring, storage, adaptation or alteration, retrieval, consultation, use, disclosure by transmission, dissemination or otherwise making available, alignment or combination, restriction, erasure or destruction.

Does your project involve personal data?	Yes	*Please provide details:* *Some partial identifying information will be collected regarding individuals' gender, age, ethnicity, lifestyle and anthropometry. This information will be anonymised, and no name and content of the participants will be recorded. The data collected on the dietary habits will have the personal identifiers. This data will be anonymised at a later stage.*
If yes, are you processing that data? If you have any kind of access to that data, including just holding it, you are likely to be processing it.	Yes	*Please provide details:* *Data will be stored on a password-encrypted device. All potential identifying personal information be numbered and randomise and handled as anonymous.*

If your proposed activity involves processing personal data, you must complete the remainder of this checklist, providing answers to each relevant question.

Section 2: Have I addressed all the key areas of data protection compliance?

This checklist will ensure that you have properly considered how you are processing the personal data you are collecting. You should be able to answer 'yes' to all questions or explain why the question is not applicable. You must

provide details to support your answers so that your proposal can be approved. If you are a researcher completing this form as part of your ethics application, you can complete parts of this checklist by referencing your answers on the relevant parts of your Ethics Application Form, if they cover the same detail.

Type of activity:	**Questionnaire/Intake Study**
Activity name/title:	**Palmitic acid (16:0) consumption in plant-based diets and its effect on metabolic health.**

Processing personal data transparently, fairly and lawfully
Data protection legislation requires us to process personal data transparently, fairly and lawfully. In practice, it means that you must: • have lawful grounds for collecting and using the personal data; • not use the data in ways that have unjustified adverse effects on the individuals concerned; • be transparent about how you intend to use the data, and give individuals appropriate *privacy notices* when collecting their personal data; • handle people's personal data only in ways they would reasonably expect; and make sure you do not do anything unlawful with the data.

1. Have you checked and confirmed that the intended uses of personal data (including special category data or criminal convictions data) have a lawful basis?	Yes	
2. What is the lawful basis for processing the personal data? *In almost all cases at UCLan, the lawful basis for processing personal data for research purposes will be that the processing is necessary for a task carried out in the public interest (Article 6(1)(e) GDPR). If you think there is an exceptional reason why this does not apply to your research, there are alternative*		*A task carried out in the public interest*

lawful bases from the GDPR which can be considered. You must contact the Information Governance team for advice, particularly if you are considering relying on consent as a lawful basis. Further information about lawful bases can be found here.		
3. Are you processing special category data **or criminal convictions data?**	No	*N/A*
4. If the answer to question 3 is 'yes', what is the lawful basis for processing this type of personal data? *If you are processing this type of data for research purposes and the processing is in the public interest, the lawful basis is likely to be Article 9(2)(j) GDPR by virtue of section 10 and Schedule 1(4) DPA.*		*N/A*
5. If the intended use of the personal data would or would be likely to have an adverse effect on one or more individuals, have you considered and documented why that adverse effect is justified?	Not applicable	*N/A*
6. Have you documented why you are collecting the specific items of information to demonstrate they are necessary for your stated purposes *e.g. if you are carrying out research into how students' music preferences affect their degree classification and also collecting participants' shoe sizes, can you show you have a legitimate need for this information, when they are not obviously linked?*	Yes	*Relevant information is included in the Ethics application.*

7. Have you written an appropriate privacy notice to provide to individuals at the point you collect their personal data? *A privacy notice is a legal requirement and tells individuals how you will process their data if they give it to you, among other things. Clear and detailed guidance about privacy notices and what they must contain is available here. If you are carrying out research, you should refer participants to the University's standard privacy notice for research participants, which must be supplemented by your participant information sheet.*	Yes	*Yes, all related and relevant information is provided on both the Participant Information Sheet(s) and Participant Consent Form(s).*

Consent
Consent to participate in a research study is not the same as relying on consent as a lawful basis on which to process personal data. Researchers are strongly discouraged from relying on consent as a lawful basis for processing personal data for research purposes. If you are relying on consent as the lawful basis for processing personal data, the GDPR requires that a certain standard of consent is obtained. Consent as a lawful basis to process personal data is only valid if it is freely given, fully informed, unambiguous and **capable of being withdrawn at any time**. If consent is withdrawn, you must cease all processing of personal data which is being carried out on the basis of that consent. For research purposes, this means that if a participant withdraws consent to process their personal data, all processing must cease and all personal data already collected about that participant must be deleted or destroyed. This may have significant implications for your research. The GDPR is clear that an indication of consent must be unambiguous and involve a clear affirmative action (an opt-in). A failure to respond to a notification that an activity will take place unless an individual opts out is not valid consent. The GDPR also requires granular consent options for distinct processing operations e.g. participants may consent to you processing their survey responses but they may choose not to consent to you taking photographs of them during focus groups. Consent should be separate from other terms and conditions. You must keep clear records to demonstrate consent has been obtained. You need to tell people about their right to withdraw and offer them easy ways to withdraw consent at any time.

Consent should only be used as a lawful basis for processing personal data if valid consent can be obtained; the consent can be withdrawn at any time; and withdrawal of consent will not impact your research. Note that where group recordings are made e.g. focus groups, it will be very difficult to remove one participant's personal data (their voice and opinions) from the recording if that participant withdraws consent to process their personal data. This is one example of why consent as a lawful basis for processing personal data will not be appropriate for many types of research.

You may still obtain participants' consent to participate in various aspects of your research, even if you do not rely on consent as a lawful basis for processing the personal data you subsequently collect. Participants may withdraw their consent to participate in any future aspects of the research but you may continue to process any personal data already collected **if** you have relied on a lawful basis for processing other than consent.

Further detailed information about obtaining valid consent under the GDPR is available here.

8. Have you checked and confirmed that consent is necessary and is the most appropriate basis for your processing? *Note that this question relates to consent as a lawful basis for processing personal data and **not** consent to participate in your research.*	N/A	
9. If you are processing special category data or criminal convictions data on the basis of consent, have you planned to obtain individuals' explicit consent? *As the standard for consent is so high under the GDPR, there is not likely to be a significant difference between consent and explicit consent.*	N/A	
10. If you are relying on individuals' consent as the lawful basis for using their personal data, have you developed a process for managing the withdrawal of consent at any point during or after the research?	N/A	

11. If you are relying on consent as a lawful basis to process personal data, have you ensured that the individual understands their rights and is capable of giving consent? This should be assessed on a case-by-case basis. *If you are processing personal data about children or those with reduced capacity, consider whether or not you need to obtain consent from parents, guardians or legal representatives. You must consider how to provide clear consent statements to those whose first language is not English.*	N/A	

Security
The GDPR requires you to process personal data securely. This means that you must have appropriate security measures in place to protect the personal data you hold from being lost, damaged, destroyed or disclosed inappropriately. Security measures include technical security measures to keep electronic information secure; physical security measures to keep hard-copy information secure; and processes and procedures around the management of, and access to, the information. Organisations can be fined up to €20 million for breaches of security involving personal data. The GDPR requires us to implement appropriate technical and organisational measures to ensure a level of security appropriate to the risk. It is important that any personal data you collect or use during your activities remains secure until it is securely destroyed, which includes ensuring that only those who are authorised to access and use the data can do so. **For further guidance on information security, please see the** Information Governance pages **of the UCLan intranet and the LIS** IT Security Policy**. You may also find the information on the** ICO website **useful.**

12. If you are intending to publish information which could identify individuals, have you made those individuals aware that this will happen via your privacy notice and obtained their consent, if appropriate?	Not applicable	*N/A*

13. Will papers, files, audio visual recordings, CDs, USB (memory) sticks, microfiche or other media which contain personal data be kept in locked cabinets, cupboards, drawers etc. when the offices are vacated or if they are stored off UCLan premises?	Yes	*Data will be kept on password protected computers in the UCLan Network Computer.*
14. Do all individuals who will have access to or be using the personal data understand that it must not be provided to any unauthorised person (which includes disclosing information to family members or other representatives of data subjects, unless the data subject has given consent for us to do this)?	Yes	*All members of the supervisory team and researcher are aware of this.*
15. Do all individuals who will have access to or be using the personal data understand their responsibilities under data protection legislation and have they received data protection training? *A GDPR e-learning course is available to staff and students via Blackboard.*	Yes	*All members of the supervisory team have undertaken GDPR training as appropriate.*
16. Do you have appropriate procedures in place to ensure the security of the personal data if it is removed from or collected and stored away from UCLan offices for any reason? *You must provide details of how you will ensure personal data remains secure if you remove it from UCLan premises or collect and store it away from UCLan premises. Please note that electronic data must only be removed if it is stored on encrypted devices or media e.g.*	Yes	*All digital data collected will be saved and stored on a password protected computer in Preston Campus of the University of Central Lancashire that has encryption and two-factor authentication facilities. The digital data will only be accessible by the lead researcher and supervisory team. All personal contact details will be retained for 28 days following results. All data collected via email will be saved and stored, as well as password protected with two-factor authentication, via Microsoft OneDrive.*

an encrypted disc or USB stick, an encrypted laptop or Dictaphone etc. Alternatively it can be accessed remotely via a secure connection. If an unencrypted device containing personal data is lost or stolen, it is likely to lead to a substantial fine for a breach of data protection legislation. Non-electronic records must be rigorously safeguarded at all times and not left unattended or in view of unauthorised people. Laptops, USB sticks and other devices, papers or any other form of personal data must not be left in cars.		
17. Will the personal data be stored on the UCLan network, or in an Office 365 account provided by UCLan, in a secure location with restricted access, to prevent unauthorised parties who have no right or need accessing the data?	Yes	Stated in the Ethics form
18. Are all individuals who will have access to or use the personal data aware that personal information should only be stored on the UCLan network or in cloud storage provided by UCLan and should only be stored on equipment owned or leased by UCLan, unless exceptional circumstances apply, after taking advice from LIS? *Storage under such exceptional circumstances must include the use of appropriate security measures. No personal information should be stored on any removable media e.g. USB sticks, CDs or devices e.g. laptops, smartphones, Dictaphones unless they are encrypted. BitLocker is the*	Yes	

recommended tool for encrypting external devices; guidance can be found here. UCLan makes available Office 365 cloud storage for staff and students, including OneDrive for Business. This is the only approved cloud storage facility.		
19. Are all individuals who will have access to or use the personal data aware that any information accessed via remote working methods such as Outlook Web App, UCLan Global or similar must be treated securely in line with relevant legislation and all University guidelines? *UCLan business information, including personal data, should not be stored on personal, non-UCLan equipment or devices unless exceptional circumstances apply, after taking guidance from LIS, even where personal equipment and devices are used to access information remotely. See the BYOD Policy for further information.*	Yes	
20. Are all individuals who will have access to or use the personal data aware that email is not a secure method of communication and can easily be sent to the wrong recipient and do they know how to encrypt documents so that they can be attached to an email and sent securely? *N.B. Encryption passwords must be provided separately and never included in the same email as the encrypted attachment. Guidance is available here. Users should consider whether or not email is the most appropriate method. Consider using*	Yes	

UCLan's OneDrive for Business instead.		
21. Are all individuals who will have access to or use the personal data aware that all non-electronic material which contains personal data and has been authorised for disposal must be disposed of via the University's confidential waste service (including handwritten notes, computer print-outs etc.)?	Yes	
22. Are all individuals who will have access to or use the personal data aware that any paper documents, electronic media or hardware which has been designated for disposal must be kept in a secure location until it has been appropriately destroyed and any information it contains is no longer accessible or recoverable? *Electronic media and hardware must be disposed of in line with LIS guidelines and procedures.*	Yes	
23. Data protection legislation prohibits the transfer of personal data to countries or territories outside the European Economic Area (EEA) unless certain conditions can be met. Can you confirm that if you intend to transfer personal data outside the EEA, you have taken advice from the Information Governance team to ensure you can do so in compliance with data protection legislation? *Transfers includes via email, websites and by virtue of using*	N/A	*N/A*

'cloud' providers which store your data on their servers based overseas, as well as physically holding the information overseas. For detailed guidance see Sending personal data outside the UK.		

Third parties acting on behalf of UCLan
Under some circumstances, it will be necessary or desirable to work with organisations external to UCLan, such as charities, research organisations, private companies, other public sector organisations, contractors, service providers or any other types of third parties. If a third party is acting on our behalf e.g. providing a service for us or on our behalf and that activity involves the third party accessing, collecting or otherwise processing personal data, they are a *data processor* under the GDPR. Guidance on the use of data processors, including a checklist to ensure their use is appropriate, is available here.

24. Are you using a data processor?	No	*If no, then you do not need to complete the questions regarding data processors below.*
25. If you are using a data processor or you need help deciding if the proposed arrangement does involve a data processor, have you read the guidance and, if necessary, taken advice from the Information Governance team to ensure all data protection requirements around the use of data processors have been satisfied?	N/A	*Please provide details:*
26. **If you are using a data processor, have you taken advice on information security from the**	N/A	*Please provide details:*

Information Governance team and the Information Security team in LIS? *It is a legal requirement to ensure a data processor is capable of putting in place appropriate technical and organisational security measures to protect personal data. If your data processor is based outside the EEA, additional measures must be in place.*		
27. If you are using a data processor, have you taken advice from the contracts team in Legal and Governance or from Procurement (as appropriate) to ensure you have appropriate contractual arrangements in place to cover the use of a data processor? *It is a legal requirement to have GDPR-compliant contracts in place with data processors.*	N/A	*Please provide details:*

Once this form has been completed, it should be attached to your Ethics Application Form (if applicable) and submitted as directed. If your activity does not require further or any ethical approval, this form should be retained with your project documentation as a record of your considerations and data protection compliance. If you require any further advice or guidance to help you complete this checklist, please contact the Information Governance team: DPFOIA@uclan.ac.uk. Members of staff can also find a variety of guidance documents and FAQs on the Information Governance intranet pages.

Appendix 3.4a. Recruitment poster for the survey and food diary data collection.

Appendix 3.4b. Recruitment social media post for the survey and food diary data collection.

Appendix 3.5. Participant information and palm oil awareness survey/questionnaire.

Questionnaire

Thank you for taking the time to provide this information. This questionnaire is designed to collect some general information about your overall health and dietary habits, as well as your knowledge on palm oil/palmitic acid. If you require any clarification, please contact Thomas at thothersall1@uclan.ac.uk. *Once completed, please return questionnaires to the provided email.*

Today's date ……

Please answer by clicking the box as appropriate.

A. Information about you

1.	Gender	Male	☐
		Female	☐
		Non-binary	☐
		Prefer not to say	☐
2.	Age (year)	18-25	☐
		25-34	☐
		35+	☐
3.	Your ethnicity	White	☐
		Mixed	☐
		Asian British	☐
		African British	☐
		Other British	☐
		Prefer not to say	☐

B. Anthropometry *(This includes Height, Weight, Waist Circumference and Hip Circumference)*

1. Your height is

…… Metre …… cm OR

…… Feet …….. inch

2. Your weight is

……. Kilogram (kg) OR

……...Pounds (lb) OR

……...Stone (st)

3. Your waist circumference is (measurement across the belly button)

…… cm OR

…… inch

4. Your hip circumference is (measurement across the hip)

…… cm OR

…… inch

C. **Physical Activity and Lifestyle** *(This includes Exercise, Profession and General Lifestyle. Please provide additional information in the space provided, if appropriate)*

1. How would you describe the level of your daily physical activity (in hr)?

Highly active (4 h or more) ☐
Moderately active (2-3 h) ☐
Sedentary (less than 1 h) ☐

2. Select the main type of physical activity/Exercise you perform on daily basis

Walking ☐
Running ☐
Cycling ☐
Swimming ☐
Weight training ☐
Others (please specify)
……

3. In a typical evening, you go to sleep at

Weekdays		*Free days*	
6:00 to 8:59 pm	☐	6:00 to 8:59 pm	☐

9:00 to 11:59 pm	☐	9:00 to 11:59 pm	☐
0:00 to 2:59 am	☐	0:00 to 2:59 am	☐
3:00 to 5:59 am	☐	3:00 to 5:59 am	☐
6:00 am to 5:59 pm	☐	6:00 am to 5:59 pm	☐

4. In a typical night, how many hours do you sleep?

Weekdays		*Free days*	
Less than 4 h	☐	Less than 4h	☐
4 to 8 h	☐	4 to 8 h	☐
8 to 9 h	☐	8 to 9 h	☐
9 h or more	☐	9 h or more	☐

D. **Dietary Habit** *(This includes information relating to your dietary habits and nutritional health)*

1. Based on your dietary habit, you are

Plant-based/vegan	☐
Vegetarian	☐
Pescatarian	☐
Flexitarian	☐
Omnivore	☐
Other (please specify)	……

2. You are on your present dietary habit since

Less than 3 months	☐
1 to 2 years	☐
2 to 5 years	☐
More than 5 years	☐
Since birth	☐

3. Which of the following describe your nutritional health?

I enjoy sound nutritional health	☐
I have nutritional deficiency	☐
I take dietary supplement	☐
I take medication	☐
I'm not sure (please explain)	☐

……

……

E. **Awareness on Palm Oil** *(This includes information relating to your awareness of palm oil and its health effect)*

1. Have you heard of palm oil/palm kernel oil/palmitic acid?

   Yes ☐
   No ☐

2. In relation to the presence of palm oil/palm kernel oil in food, please select as appropriate

   Foods I consume **rarely** contain palm oil or its derivatives ☐
   Foods I consume **don't** contain palm oil or its derivatives ☐
   I **avoid** foods that contain palm oil or its derivatives ☐
   I **don't mind** about the presence of palm oil or its derivatives in food ☐

3. In relation to the health effect of palm oil and its derivatives, please select as appropriate

   Palm oil is healthy ☐
   Palm oil is unhealthy ☐
   I don't know if healthy or unhealthy ☐

4. If you are to avoid the foods containing palm oil and its derivatives, which of the following would motivate you?

   Health reason (s) ☐
   Ethical reason (s) ☐
   Environmental reason (s) ☐
   Economic reason (s) ☐

Addition information about your dietary habit(s)/nutritional health (optional)

……

……

……

……

……

……

……

Appendix 3.6. Participant food/diet diary.

4-Day Diet Diary

University of Central Lancashire
UCLan

Instructions

1. Please use this diary to fill out the following diet diaries (a total of 4 days, over one week), including all meals, snacks and beverages (including alcohol):
    a. Week 1 - Monday, Wednesday, Friday and Saturday

2. Each page is a new day and diary, please fill out the date (for example, Jan 3rd 2022) and print name or sign at the bottom of each page once completed.

3. Please include as much relevant information as possible, from brand and cooking method, to serving size. If unsure of exact serving size, please provide a good guess/estimate*. Full list of what to include:
    a. Food or drink item.
    b. Brand name(s).
    c. Serving size (if you have access to a scale, *weigh food before cooking):*
        i. e.g., g, ml, tsp, tbsp.
        ii. e.g., small, medium, large.
    d. Additional info (optional), e.g.:
        i. Meat: fresh, frozen, fat included, fat cut off.
        ii. Vegetables/Fruit: fresh, frozen, tinned.
        iii. Cup of tea (with milk); fried in 1tbsp of olive oil; drizzled in BBQ sauce

**Example:*

Fresh Banana

Small Medium Large

4. More accurate examples of serving sizes (in grams), as well as a 1-day diet diary example are provided on the following pages.

5. If you are buying store-bought food (i.e., 'meal deals'), eating out or ordering a takeaway - please describe as accurately as possible.

Serving size example(s)

Fresh Broccoli - Steamed

50g

100g

150g

200g

250g

White Rice - Microwaved

50g

100g

150g

200g

250g

Frozen Oven Chips - Oven-baked

68g (Frozen)
50g (Cooked)

136g (Frozen)
100g (Cooked)

204g (Frozen)
150g (Cooked)

272g (Frozen)
200g (Cooked)

340g (Frozen)
250g (Cooked)

Pasta - Pan-boiled

22.2g (Uncooked)
50g (Cooked)

44.4g (Uncooked)
100g (Cooked)

66.6g (Uncooked)
150g (Cooked)

88.8g (Uncooked)
200g (Cooked)

111.1g (Uncooked)
250g (Cooked)

Diet diary example

Meal	Food or drink	Brand	Amount or size	Additional info (optional)
Early morning meal or breakfast	Avocado Wholemeal bread Orange juice	Tesco Warburtons Tropicana	1 x medium 2 x slices 250ml	Fresh/uncooked Toasted -
Mid-morning	Apple Coffee Oat milk Pineapple	Tesco Nescafé Oatly Del Monte	1 x medium 2 x tsp 100ml 2 x slices/chunks	Fresh Kettle-boiled Kettle-boiled Fresh/tinned
Lunch	Sweet potato Spinach Mayonnaise Green tea	Tesco Tesco Hellman's Twinings	150g 80g 2 x tbsp 1 x teabag	Oven-baked Pan-fried (no oil) - Kettle-boiled
Afternoon	Bananas Cashew nuts Protein shake (choc.) Falafel Cookies	Tesco Aasani Bulk Cauldron Oreo	2 x small 30g 60g (2 x scoop) 2 x 3 x	Fresh - - Uncooked -
Evening meal or dinner	Green curry paste Tofu Red pepper Babycorn Sugar snap peas Spring onion Red chilli peppers Tenderstem broccoli Jasmine rice	Blue Dragon Tofoo Tesco Tesco Tesco Tesco Tesco Tesco Uncle Ben's	90g 125g 1 x large 60g 50g 25g 30g 50g 125g (half packet)	Stir-fried (no oil) Air-fried Oven-roasted Stir-fried (no oil) Stir-fried (no oil) Stir-fried (no oil) Stir-fried (no oil) Oven-roasted Microwaved
Supper	Porridge Oat milk Blueberries	Quaker Oatly Tesco	75g 150ml 50g	Microwaved Microwaved Fresh

Today's date Jan 3rd 2022 **Sign/print name** Thomas Hothersall

Week 1 - Monday

Meal	Food or drink	Brand	Amount or size	Additional info (optional)
Early morning meal or breakfast				
Mid-morning				
Lunch				
Afternoon				
Evening meal or dinner				
Supper				

Today's date …… **Sign/print name** ……

Week 1 - Wednesday

Meal	Food or drink	Brand	Amount or size	Additional info (optional)
Early morning meal or breakfast				
Mid-morning				
Lunch				
Afternoon				
Evening meal or dinner				
Supper				

Today's date …… **Sign/print name** ……

Week 1 - Friday

Meal	Food or drink	Brand	Amount or size	Additional info (optional)
Early morning meal or breakfast				
Mid-morning				
Lunch				
Afternoon				
Evening meal or dinner				
Supper				

Today's date …… **Sign/print name** ……

Week 1 - Saturday

Meal	Food or drink	Brand	Amount or size	Additional info (optional)
Early morning meal or breakfast				
Mid-morning				
Lunch				
Afternoon				
Evening meal or dinner				
Supper				

Today's date …… **Sign/print name** ……

Appendix 3.7a. Respondent dietary intake (4-day mean) of calories and carbohydrates.

ID	Dietary habit	Energy (kcal)		Carbohydrates (g)					
		Daily Intake	Target Intake	Total	Fibre	Sugars	Free Sugars	Calories	%
1	Pescatarian	1403.93	2136.36	149.04	21.32	42.59	0.94	596.18	42.46%
2	Omnivore	1650.21	2092.20	170.53	22.90	50.22	2.47	682.12	41.34%
3	Omnivore	2245.01	2740.80	245.59	22.25	64.14	0.00	982.34	43.76%
4	Vegetarian	1215.03	1887.36	149.73	18.87	41.48	11.37	598.91	49.29%
5	Flexitarian	2144.10	2486.01	217.18	27.89	111.83	4.05	868.73	40.52%
6	Omnivore	1937.03	2353.85	189.80	15.48	48.85	2.54	759.21	39.19%
7	Plant-based/vegan	2180.66	2543.55	250.68	40.88	79.39	3.81	1002.71	45.98%
8	Omnivore	2362.97	2383.90	300.32	21.13	167.33	11.25	1201.26	50.84%
9	Omnivore	1642.66	2023.28	155.35	16.41	57.06	32.69	621.40	37.83%
10	Omnivore	2369.65	2707.64	190.78	17.54	55.78	13.54	763.12	32.20%
11	Vegetarian	2147.96	2140.55	194.59	31.07	72.41	7.31	778.35	36.24%
12	Flexitarian	2212.04	2652.05	238.29	40.46	63.91	6.66	953.15	43.09%
13	Omnivore	2419.40	3876.93	204.93	10.94	47.73	0.00	819.73	33.88%
14	Other: Paleo	2545.08	3392.95	152.70	39.74	78.75	14.09	610.80	24.00%
15	Flexitarian	2766.36	2596.15	269.82	37.69	65.92	4.99	1079.28	39.01%
16	Plant-based/vegan	2251.01	2103.35	225.20	35.11	82.74	1.28	900.79	40.02%
17	Omnivore	2158.67	2496.08	195.23	31.61	99.42	0.65	780.92	36.18%
18	Other: Dairy free	2042.47	2063.17	239.43	19.84	76.87	0.98	957.74	46.89%
19	Omnivore	1845.51	3062.80	178.17	11.63	52.34	0.26	712.69	38.62%
20	Omnivore	1845.51	2427.31	178.17	11.63	52.34	0.26	712.69	38.62%

	21	Flexitarian	2515.41	2162.25	307.29	25.53	98.78	25.48	1229.17	48.87%	
	22	Flexitarian	1528.61	1909.78	183.55	18.25	76.23	20.07	734.20	48.03%	
	23	Vegetarian	1353.59	2232.12	175.12	19.74	42.25	1.26	700.50	51.75%	
	24	Omnivore	1821.56	2213.64	206.72	20.42	65.03	23.27	826.88	45.39%	
	25	Omnivore	1050.31	2211.00	123.26	18.58	63.04	2.46	493.05	46.94%	
	26	Flexitarian	1388.25	1603.42	200.89	22.02	52.68	4.33	803.55	57.88%	
	27	Plant-based/vegan	1799.66	3213.28	195.46	31.59	51.40	1.45	781.84	43.44%	
	28	Pescatarian	1788.15	2534.25	167.73	24.97	89.56	1.19	670.92	37.52%	
	29	Plant-based/vegan	1474.11	1881.00	191.51	26.79	64.30	1.31	766.03	51.97%	
	30	Plant-based/vegan	1830.99	2503.25	229.82	32.89	66.15	4.30	919.28	50.21%	
	31	Plant-based/vegan	1935.04	2245.10	233.89	62.28	111.31	1.30	935.57	48.35%	
	32	Flexitarian	1641.77	2627.87	219.56	36.11	53.95	0.94	878.25	53.49%	
	33	Vegetarian	2319.54	2294.00	278.50	27.35	55.16	11.37	1113.99	48.03%	
	34	Omnivore	1507.02	2515.37	114.21	10.48	27.93	4.78	456.85	30.32%	
	35	Omnivore	1355.56	1925.10	97.86	7.82	26.32	4.78	391.45	28.88%	
	36	Omnivore	1708.80	1841.84	142.58	12.37	42.16	5.58	570.31	33.37%	
	37	Omnivore	1699.61	2152.15	168.55	16.21	41.00	17.19	674.19	39.67%	
	38	Omnivore	2359.32	2687.70	219.52	15.37	49.04	5.58	878.09	37.22%	
	39	Omnivore	1973.29	2783.80	245.76	18.49	53.72	19.51	983.04	49.82%	
	40	Omnivore	2400.43	3306.60	220.71	15.51	54.71	3.17	882.85	36.78%	
	41	Plant-based/vegan	2050.45	2366.85	226.27	31.93	85.84	7.64	905.07	44.14%	
	42	Omnivore	1690.33	2621.52	127.80	12.62	41.64	13.85	511.19	30.24%	
	43	Plant-based/vegan	2437.66	2512.91	263.02	40.65	95.02	13.17	1052.10	43.16%	
	44	Plant-based/vegan	1864.25	2053.75	247.23	33.96	103.96	43.26	988.92	53.05%	
	45	Flexitarian	1647.05	2631.90	168.87	23.92	52.05	3.72	675.49	41.01%	
	46	Omnivore	1241.83	1923.24	165.62	18.16	43.78	23.20	662.47	53.35%	

	47	Plant-based/vegan	2391.25	2140.20	215.68	63.44	133.64	1.37	862.72	36.08%	
	48	Omnivore	2102.54	2859.75	260.47	25.44	134.94	0.54	1041.86	49.55%	
	49	Omnivore	1772.08	3268.91	160.77	19.06	63.19	3.50	643.08	36.29%	
	50	Omnivore	1650.31	1965.25	163.06	23.81	85.66	3.77	652.23	39.52%	
	51	Plant-based/vegan	2586.85	2273.22	334.72	58.14	154.47	24.64	1338.88	51.76%	
	52	Plant-based/vegan	1080.47	2173.10	132.44	28.74	39.53	12.17	529.76	49.03%	
	53	Omnivore	1685.65	2343.74	189.03	15.10	47.47	4.36	756.13	44.86%	
	54	Omnivore	1493.31	1990.67	142.05	13.42	41.37	2.28	568.21	38.05%	
	55	Omnivore	1555.89	1666.44	173.23	11.05	57.66	8.51	692.94	44.54%	
	56	Omnivore	2158.81	1879.19	215.90	18.08	59.33	30.19	863.61	40.00%	
	57	Vegetarian	1715.53	1931.61	253.78	22.22	97.08	43.80	1015.11	59.17%	
	58	Vegetarian	1763.65	1954.73	276.25	17.99	114.51	36.96	1105.02	62.66%	
	59	Omnivore	1414.03	2671.09	130.76	21.52	42.37	5.90	523.04	36.99%	
	60	Omnivore	1551.44	3110.54	116.39	13.89	26.00	5.19	465.58	30.01%	
	61	Vegetarian	1300.10	2358.32	149.48	25.80	60.10	28.36	597.93	45.99%	
	62	Omnivore	1589.89	2085.09	212.93	21.87	72.00	13.94	851.71	53.57%	

Appendix 3.7b. Respondent dietary intake (4-day mean) of fats.

ID	Dietary habit	Fats (g)								
		Total	Saturated	% Sat./Fat	SFA Calories	Trans-fatty acids	Monounsaturated	Polyunsaturated	Calories	%
1	Pescatarian	49.93	15.01	30.06%	135.08	0.19	11.91	5.97	449.41	32.01%
2	Omnivore	69.74	20.04	28.74%	180.38	0.92	18.72	6.87	627.66	38.04%
3	Omnivore	99.72	15.45	15.49%	139.06	0.36	14.09	11.86	897.50	39.98%
4	Vegetarian	44.46	17.34	39.01%	156.07	0.86	10.85	5.05	400.10	32.93%
5	Flexitarian	102.79	31.73	30.87%	285.56	0.46	21.72	15.51	925.11	43.15%
6	Omnivore	78.36	30.19	38.53%	271.71	1.72	10.17	2.47	705.27	36.41%
7	Plant-based/vegan	89.32	21.33	23.88%	192.00	0.00	5.33	9.63	803.90	36.86%
8	Omnivore	83.54	24.10	28.85%	216.92	0.19	15.51	4.02	751.89	31.82%
9	Omnivore	68.98	23.83	34.55%	214.49	0.60	14.71	8.46	620.82	37.79%
10	Omnivore	118.93	48.69	40.94%	438.20	1.88	29.92	5.91	1070.39	45.17%
11	Vegetarian	102.46	34.31	33.48%	308.75	0.31	18.53	7.75	922.18	42.93%
12	Flexitarian	84.46	22.81	27.01%	205.30	0.22	10.70	5.36	760.16	34.36%
13	Omnivore	51.54	16.51	32.03%	148.59	0.39	8.97	8.53	463.90	19.17%
14	Other: Paleo	131.83	51.04	38.71%	459.33	0.94	23.52	6.24	1186.50	46.62%
15	Flexitarian	153.53	41.80	27.23%	376.19	0.17	18.84	40.93	1381.78	49.95%
16	Plant-based/vegan	122.92	22.03	17.93%	198.31	0.10	16.58	14.74	1106.25	49.14%
17	Omnivore	86.37	22.47	26.01%	202.21	0.17	12.11	6.28	777.30	36.01%
18	Other: Dairy free	85.93	31.16	36.27%	280.46	0.02	3.56	1.75	773.35	37.86%
19	Omnivore	95.66	36.42	38.07%	327.79	0.66	12.27	4.64	860.93	46.65%
20	Omnivore	95.66	36.42	38.07%	327.79	0.66	12.27	4.64	860.93	46.65%
21	Flexitarian	84.85	28.66	33.77%	257.90	0.28	4.91	2.60	763.64	30.36%

22	Flexitarian	73.37	29.47	40.17%	265.26	0.16	11.70	11.91	660.35	43.20%	
23	Vegetarian	47.72	14.11	29.56%	126.97	0.33	10.29	5.83	429.51	31.73%	
24	Omnivore	77.70	29.59	38.09%	266.33	0.30	20.61	8.26	699.29	38.39%	
25	Omnivore	41.52	11.48	27.64%	103.28	0.05	8.87	6.57	373.65	35.58%	
26	Flexitarian	46.69	13.81	29.58%	124.30	0.01	2.28	1.54	420.17	30.27%	
27	Plant-based/vegan	80.34	25.05	31.18%	225.46	0.89	6.55	2.82	723.10	40.18%	
28	Pescatarian	98.73	31.03	31.43%	279.25	0.03	15.72	7.61	888.60	49.69%	
29	Plant-based/vegan	60.13	18.13	30.16%	163.21	0.01	4.92	3.97	541.20	36.71%	
30	Plant-based/vegan	75.29	16.63	22.08%	149.64	0.00	6.53	10.24	677.59	37.01%	
31	Plant-based/vegan	75.82	13.42	17.70%	120.81	0.03	6.27	17.53	682.37	35.26%	
32	Flexitarian	66.21	33.89	51.19%	305.03	0.12	9.29	14.20	595.85	36.29%	
33	Vegetarian	96.83	30.35	31.34%	273.12	0.80	22.75	11.07	871.49	37.57%	
34	Omnivore	80.05	21.56	26.94%	194.08	1.25	20.75	21.04	720.46	47.81%	
35	Omnivore	72.67	17.96	24.71%	161.64	1.24	20.70	21.04	654.05	48.25%	
36	Omnivore	68.73	20.30	29.54%	182.74	0.85	22.16	14.40	618.57	36.20%	
37	Omnivore	65.83	17.02	25.85%	153.15	0.56	28.46	11.99	592.49	34.86%	
38	Omnivore	82.27	26.63	32.37%	239.65	1.01	27.54	12.61	740.42	31.38%	
39	Omnivore	75.83	27.10	35.74%	243.91	0.91	11.98	9.32	682.49	34.59%	
40	Omnivore	132.33	48.05	36.31%	432.46	2.13	46.36	15.04	1190.94	49.61%	
41	Plant-based/vegan	93.75	27.78	29.63%	250.02	0.05	14.57	11.17	843.71	41.15%	
42	Omnivore	80.66	32.54	40.35%	292.89	0.80	23.76	8.56	725.91	42.94%	
43	Plant-based/vegan	107.83	19.38	17.97%	174.39	0.17	10.93	13.54	970.44	39.81%	
44	Plant-based/vegan	69.74	13.06	18.72%	117.52	0.12	15.36	9.71	627.64	33.67%	
45	Flexitarian	73.37	27.42	37.37%	246.78	0.68	17.00	7.15	660.31	40.09%	
46	Omnivore	38.00	8.28	21.78%	74.50	0.09	11.41	5.51	341.99	27.54%	
47	Plant-based/vegan	135.05	64.71	47.91%	582.35	0.00	21.43	6.73	1215.43	50.83%	

48	Omnivore	74.86	31.12	41.57%	280.05	0.38	6.83	3.14	673.73	32.04%	
49	Omnivore	64.60	18.73	28.99%	168.55	0.34	8.49	6.32	581.39	32.81%	
50	Omnivore	59.40	19.12	32.18%	172.05	0.86	19.23	11.52	534.61	32.39%	
51	Plant-based/vegan	100.23	15.69	15.65%	141.22	0.50	18.04	16.28	902.07	34.87%	
52	Plant-based/vegan	33.73	9.00	26.68%	81.00	0.05	4.92	4.93	303.58	28.10%	
53	Omnivore	69.13	24.19	34.99%	217.68	0.14	3.79	2.68	622.14	36.91%	
54	Omnivore	63.90	23.34	36.52%	210.04	1.35	22.14	9.93	575.09	38.51%	
55	Omnivore	66.42	16.20	24.39%	145.78	0.47	21.32	10.57	597.75	38.42%	
56	Omnivore	98.28	30.09	30.61%	270.78	0.68	36.17	15.68	884.49	40.97%	
57	Vegetarian	56.84	18.37	32.32%	165.33	0.34	15.52	6.82	511.53	29.82%	
58	Vegetarian	44.08	13.34	30.26%	120.04	0.30	12.44	5.95	396.68	22.49%	
59	Omnivore	52.47	22.08	42.07%	198.70	0.86	18.05	6.93	472.26	33.40%	
60	Omnivore	65.12	26.53	40.74%	238.76	1.51	20.03	8.29	586.10	37.78%	
61	Vegetarian	50.38	14.71	29.19%	132.35	0.54	16.21	11.35	453.38	34.87%	
62	Omnivore	54.70	22.28	40.73%	200.49	0.81	14.66	5.92	492.27	30.96%	

Appendix 3.7c. Respondent dietary intake (4-day mean) of protein and vitamins A, D, E, K.

ID	Dietary habit	Protein (g)			Vitamin(s)			
		Total	Calories	%	A (ug)	D (ug)	E (mg)	K (ug)
1	Pescatarian	49.79	199.17	14.19%	764.57	3.13	5.90	77.22
2	Omnivore	70.86	283.44	17.18%	156.98	2.62	6.68	6.46
3	Omnivore	91.13	364.52	16.24%	228.13	0.38	2.90	1.70
4	Vegetarian	54.41	217.65	17.91%	896.48	2.28	4.31	119.37
5	Flexitarian	77.80	311.20	14.51%	854.37	5.53	9.89	507.48
6	Omnivore	82.15	328.61	16.96%	114.48	0.69	1.15	0.28
7	Plant-based/vegan	93.70	374.79	17.19%	762.09	1.31	4.12	24.17
8	Omnivore	102.98	411.90	17.43%	830.62	2.93	10.27	3.13
9	Omnivore	79.63	318.52	19.39%	564.80	3.59	5.00	56.98
10	Omnivore	134.01	536.05	22.62%	11049.76	7.20	5.93	43.79
11	Vegetarian	98.10	392.41	18.27%	765.02	12.13	9.07	26.33
12	Flexitarian	102.08	408.30	18.46%	130.37	1.39	3.26	20.28
13	Omnivore	258.78	1035.12	42.78%	84.31	1.11	2.75	26.74
14	Other: Paleo	185.78	743.12	29.20%	7710.58	3.47	5.23	11.92
15	Flexitarian	85.42	341.68	12.35%	332.13	0.15	33.85	6.53
16	Plant-based/vegan	60.91	243.63	10.82%	407.02	2.14	13.67	60.91
17	Omnivore	151.95	607.79	28.16%	1649.77	0.04	6.68	74.47
18	Other: Dairy free	75.89	303.55	14.86%	58.66	1.50	7.65	0.60
19	Omnivore	68.05	272.22	14.75%	265.53	0.42	1.76	38.96
20	Omnivore	68.05	272.22	14.75%	265.53	0.42	1.76	38.96
21	Flexitarian	95.42	381.67	15.17%	103.43	1.05	0.87	8.38

	22	Flexitarian	33.60	134.41	8.79%	1486.94	2.33	13.69	129.22	
	23	Vegetarian	55.97	223.90	16.54%	290.08	1.23	4.16	64.55	
	24	Omnivore	73.96	295.84	16.24%	957.47	3.90	9.03	26.92	
	25	Omnivore	45.31	181.25	17.26%	704.67	0.05	5.47	59.50	
	26	Flexitarian	41.32	165.26	11.90%	431.26	1.95	3.54	25.76	
	27	Plant-based/vegan	74.19	296.78	16.49%	258.56	1.13	5.56	1.15	
	28	Pescatarian	40.04	160.14	8.96%	656.49	2.29	5.82	7.16	
	29	Plant-based/vegan	42.00	168.01	11.40%	701.42	0.75	4.28	12.15	
	30	Plant-based/vegan	59.07	236.27	12.90%	181.64	6.64	4.72	11.95	
	31	Plant-based/vegan	80.55	322.20	16.65%	621.96	1.53	6.58	88.25	
	32	Flexitarian	44.04	176.18	10.73%	189.84	0.12	11.07	54.28	
	33	Vegetarian	84.02	336.08	14.49%	233.50	1.20	9.42	32.62	
	34	Omnivore	58.87	235.49	15.63%	630.37	0.68	10.33	98.74	
	35	Omnivore	53.98	215.91	15.93%	627.87	0.68	10.32	98.74	
	36	Omnivore	63.83	255.33	14.94%	1508.37	2.63	6.02	107.56	
	37	Omnivore	57.58	230.32	13.55%	699.84	2.30	10.08	56.19	
	38	Omnivore	71.33	285.34	12.09%	1571.58	2.48	7.11	102.97	
	39	Omnivore	77.04	308.16	15.62%	337.34	1.04	6.52	1.84	
	40	Omnivore	81.69	326.75	13.61%	772.84	1.91	15.80	24.39	
	41	Plant-based/vegan	75.54	302.17	14.74%	1387.30	1.35	5.67	113.44	
	42	Omnivore	113.31	453.22	26.81%	495.45	4.66	8.39	18.42	
	43	Plant-based/vegan	103.88	415.53	17.05%	489.31	2.92	10.79	24.63	
	44	Plant-based/vegan	62.84	251.35	13.48%	1238.47	1.13	7.99	177.76	
	45	Flexitarian	79.20	316.80	19.23%	788.88	3.60	5.25	97.10	
	46	Omnivore	59.95	239.81	19.31%	189.12	1.48	4.22	18.81	
	47	Plant-based/vegan	79.29	317.15	13.26%	3560.28	5.94	22.75	412.08	

	48	Omnivore	96.98	387.92	18.45%	632.89	0.38	2.19	14.60	
	49	Omnivore	115.24	460.97	26.01%	578.90	2.45	3.13	107.33	
	50	Omnivore	96.87	387.50	23.48%	950.71	7.19	9.75	35.60	
	51	Plant-based/vegan	68.62	274.49	10.61%	1530.36	4.75	9.53	117.00	
	52	Plant-based/vegan	48.17	192.69	17.83%	265.05	0.54	4.80	289.30	
	53	Omnivore	76.97	307.88	18.26%	46.53	6.33	2.66	0.13	
	54	Omnivore	85.71	342.83	22.96%	244.50	0.78	3.08	17.20	
	55	Omnivore	66.29	265.15	17.04%	569.72	0.07	4.71	44.62	
	56	Omnivore	102.89	411.57	19.06%	314.78	2.92	6.46	18.01	
	57	Vegetarian	47.54	190.16	11.08%	832.37	0.92	7.15	15.52	
	58	Vegetarian	66.12	264.49	15.00%	805.20	0.70	5.69	9.98	
	59	Omnivore	82.62	330.47	23.37%	1481.00	1.37	8.27	55.27	
	60	Omnivore	87.03	348.13	22.44%	387.15	2.39	4.42	27.86	
	61	Vegetarian	62.10	248.39	19.11%	576.78	5.13	11.00	17.13	
	62	Omnivore	61.45	245.81	15.46%	916.54	1.91	6.67	117.06	

Appendix 3.8. Respondent dietary intake (mean) per dietary habit.

Variable	Dietary Habit	*n*	Mean	Std. Error	*P-value*
Energy (kcal)					
	Omnivore	30	1820.28	67.33	
	Flexitarian	9	1943.76	160.81	
Daily intake (kcal)	Vegetarian	7	1687.91	162.05	**0.521**
	Plant-based/vegan	12	1990.20	123.08	
	Other	4	1944.91	239.26	
	Omnivore	29	2421.32	83.16	
	Flexitarian	9	2306.85	127.10	
Target intake (kcal)	Vegetarian	7	2114.10	71.84	**0.406**
	Plant-based/vegan	12	2334.13	98.89	
	Other	4	2531.68	305.17	
Carbohydrates (g)					
	Omnivore	30	181.18	8.64	
	Flexitarian	9	219.55	15.46	
Total	Vegetarian	7	211.06	21.68	**0.030**
	Plant-based/vegan	12	228.83	13.82	
	Other	4	177.23	21.13	
	Omnivore	30	16.93	0.94	
	Flexitarian	9	28.31	2.62	
Fibre	Vegetarian	7	23.29	1.86	**<0.001**
	Plant-based/vegan	11	38.45	3.50	
	Other	4	26.47	4.55	
	Omnivore	29	55.72	4.08	
	Flexitarian	9	69.51	7.40	
Sugars	Vegetarian	7	69.00	10.48	**0.008**
	Plant-based/vegan	12	88.98	9.60	
	Other	4	71.94	10.18	
	Omnivore	30	8.76	1.68	
	Flexitarian	9	8.08	2.86	
Free sugars	Vegetarian	5	11.93	4.50	**0.830**
	Plant-based/vegan	12	9.64	3.68	
	Other	4	4.30	3.26	
	Omnivore	30	724.74	34.57	
Calories	Flexitarian	9	878.22	61.82	**0.030**
	Vegetarian	7	844.26	86.72	

		Plant-based/vegan	12	915.31	55.28	
		Other	4	708.91	84.51	
		Omnivore	30	40	1	
		Flexitarian	9	46	2	
	Macronutrient (%)	Vegetarian	7	50	3	**0.002**
		Plant-based/vegan	12	46	2	
		Other	4	38	5	
	Fats (g)					
		Omnivore	30	75.44	3.77	
		Flexitarian	9	83.89	10.09	
	Total	Vegetarian	7	63.25	9.55	**0.214**
		Plant-based/vegan	12	87.01	7.99	
		Other	4	91.61	16.93	
		Omnivore	30	24.94	1.69	
		Flexitarian	9	27.74	2.72	
	Saturated	Vegetarian	7	20.36	3.19	**0.038**
		Plant-based/vegan	11	18.32	1.67	
		Other	4	32.06	7.38	
		Omnivore	30	33	1	
		Flexitarian	9	34	3	
	Saturated (% of Total)	Vegetarian	7	32	1	**<0.001**
		Plant-based/vegan	11	23	2	
		Other	4	34	2	
		Omnivore	30	224.48	15.21	
		Flexitarian	9	249.63	24.49	
	Saturated (kcal)	Vegetarian	7	183.23	28.72	**0.038**
		Plant-based/vegan	11	164.87	15.04	
		Other	4	288.53	66.38	
		Omnivore	29	0.73	0.09	
		Flexitarian	9	0.34	0.10	
	Trans-fats	Vegetarian	7	0.50	0.09	**0.001**
		Plant-based/vegan	12	0.16	0.08	
		Other	4	0.30	0.22	
		Omnivore	29	17.14	1.41	
		Flexitarian	9	12.80	2.23	
	Monounsaturated	Vegetarian	7	15.23	1.69	**0.113**
		Plant-based/vegan	12	10.95	1.72	
		Other	4	13.68	4.15	
		Omnivore	30	9.07	0.88	
	Polyunsaturated	Flexitarian	8	8.14	1.84	**0.436**

		Vegetarian	7	7.69	0.96	
		Plant-based/vegan	12	10.11	1.40	
		Other	4	5.39	1.27	
		Omnivore	30	678.97	33.92	
		Flexitarian	9	755.00	90.79	
	Calories	Vegetarian	7	569.27	85.99	**0.214**
		Plant-based/vegan	12	783.11	71.89	
		Other	4	824.47	152.33	
		Omnivore	30	37	1	
		Flexitarian	9	38	2	
	Macronutrient (%)	Vegetarian	7	33	2	**0.321**
		Plant-based/vegan	12	39	2	
		Other	4	42	4	
	Protein (g)					
		Omnivore	28	80.53	3.91	
		Flexitarian	9	69.97	8.23	
	Total	Vegetarian	7	66.89	6.80	**0.158**
		Plant-based/vegan	12	70.73	5.12	
		Other	3	55.24	10.70	
		Omnivore	28	322.13	15.63	
		Flexitarian	9	279.88	32.93	
	Calories	Vegetarian	7	267.58	27.19	**0.158**
		Plant-based/vegan	12	282.92	20.49	
		Other	3	220.95	42.81	
		Omnivore	28	18	1	
		Flexitarian	9	14	1	
	Macronutrient (%)	Vegetarian	7	16	1	**0.002**
		Plant-based/vegan	12	14	1	
		Other	3	13	2	
	Vitamin(s)					
		Omnivore	29	642.10	83.99	
		Flexitarian	9	497.13	154.73	
	Vitamin A (ug)	Vegetarian	7	628.49	101.94	**0.822**
		Plant-based/vegan	11	713.02	142.79	
		Other	3	493.24	219.52	
		Omnivore	30	2.15	0.37	
		Flexitarian	9	2.08	0.57	
	Vitamin D (ug)	Vegetarian	6	1.91	0.68	**0.954**
		Plant-based/vegan	12	2.51	0.61	

		Other	4	2.60	0.44		
		Omnivore	30	6.09	0.62		
		Flexitarian	8	6.78	1.56		
	Vitamin E (mg)	Vegetarian	7	7.26	1.01	**0.874**	
		Plant-based/vegan	11	7.06	0.93		
		Other	4	6.15	0.52		
		Omnivore	30	44.56	6.65		
		Flexitarian	8	43.50	16.46		
	Vitamin K (ug)	Vegetarian	7	40.79	14.77	**0.590**	
		Plant-based/vegan	10	63.14	18.64		
		Other	4	24.23	17.82		

Appendix 4.1. Protocol for FAME preparation for gas chromatography analysis.

University College Dublin – School of Agriculture and Food Science		
Document Type: AM-IFH-001	**Modified:** 11/07/2023	**Page Number:** Page 1 of 7
Title: MICROWAVE-ASSISTED PREPARATION OF FATTY ACID METHYL ESTERS FOR GC ANALYSIS		

1. Purpose

The purpose of this Analytical Method (AM) is to outline the microwave-assisted preparation of fatty acid methyl esters (FAMEs) for gas chromatographic (GC) analysis.

2. Equipment & materials

Equipment	Materials
Analytical Balance (min. 3 decimals)	KOH (solid)
Top-loading balance (2 decimals)	Methanol
Stir plate	Acetyl Chloride
Fume hood	Pentane
Microwave	Chloroform*
GC with FID	C23:0-Methyl ester (IS)
	C23:0, free acid (IS)*
	NaCl
	Na2SO4 anhydrous
	DI water

NOTE 1: Chloroform is only required when C23:0, free acid is used as internal standard.
NOTE 2: The microwave emits a strong magnetic field. Persons with pacemakers are advised against the use of the equipment.

3. Analytical method

3.1. Solutions

NOTE: Except for the saturated NaCl-Solution all solution need to be prepared in fume hood!

- **2.5% KOH in Methanol Solution (1L)**
  1. Weigh 25 g of KOH using a top-loading balance.
  2. Measure 1L of methanol using a graduated cylinder.
  3. Transfer approx. 700 mL of methanol to a 1L Duran bottle containing a stir bar.
  4. In the fume hood, stir the methanol on the stir plate and slowly add the KOH.
  5. Transfer the rest of the methanol in the Duran bottle and stir until KOH is fully dissolved.
  6. Once the solution has cooled down to room temperature, label 2.5% KOH in Methanol Solution.

- **5% Acetyl Chloride in Methanol Solution (500ml)**
  1. In the fume hood, measure 475 ml of methanol using a graduated cylinder and transfer to a 1L Duran bottle containing a stir bar.
  2. Prepare a big container with ice and place Duran bottle containing methanol in it. Let it cool down for 10 min before proceeding with step 4.
  3. Using a graduated cylinder, measure 25ml of acetyl chloride.
  4. Slowly add the acetyl chloride into the Duran bottle containing methanol.

NOTE: Add acetyl chloride very SLOWLY, very exothermic reaction!

NOTE: The Duran bottle must remain placed in an ice bath during the addition of acetyl chloride as a lot of heat is produced during the mixing process.

5. Bring the solution to room temperature and swirl gently using a stir plate.
6. Label the solution as 5% Acetyl Chloride in Methanol Solution.

- **Saturated NaCl-Solution (500ml)**
  1. Transfer 500 mL DI water to a 1 L Duran flask containing a stir bar.
  2. While stirring, slowly add NaCl until the added NaCl no longer dissolves (approximately 180 g of added NaCl); label the flask Saturated NaCl Solution.

3.2. Internal Standard

- **Internal Standard Solution (C23:0-Methyl Ester)**
  1. Accurately weigh 0.1 g of IS (C23:0-Methyl Ester) directly into a 10 mL volumetric flask using an analytical balance and record the weight.
  2. Dissolve the IS in a small amount of heptane (about 7 mL) and shake the flask well.
  3. Make the solution to volume, mix well and label the flask IS Solution (concentration: 10 mg/mL).

- **Internal Standard Solution (C23:0, Free Acid)**

NOTE: This is only required when it is preferred to use the free fatty acid as Internal Standard rather than the FAME.

1. Accurately weigh 0.1 g of IS (C23:0, Free Acid) directly into a 10 mL volumetric flask using an analytical balance and record the weight.
2. Dissolve the IS in a small amount of chloroform (about 7 mL). Place it in water bath at 40°C and shake the flask well.

NOTE: Ensure IS is completely dissolved.

3. Make the solution to volume, mix well and label the flask IS Solution (concentration: 10 mg/mL).

3.3. Sample Preparation

1. Add a 10 mm stir bar to each MW Xpress vessels (clean and dry). Label vessels using laundry marker.
2. Accurately weigh out the sample as per Table 1 and Table 2 on an analytical balance and record the sample weight.

Table 1. Sample types and corresponding weight required for analysis (Original SOP)

Sample type	Lipid content	Example	Sample Weight
Wet solid	Very High (80%)	Meat fat, butter	0.1- 0.2g
	High (50%)	Walnuts	0.3 - 0.5 g
	Average (25%)	Pepperoni, cheese	0.5 – 0.7 g
	Low (5%-10%)	Lean Meat, yogurt	0.8- 1 g
	Low	Crab	3 g

Dry solid	Average	Milk powder	0.5 g
	Low	Grains	1.5 g
	Very low	Bread	2 g
Liquid	Average	Milk	1 ml
	High	Oil	3 drops (record weight)

NOTE 1: Addition of reagents for the saponification and esterification steps must be performed in the fume hood.
NOTE 2: Wear protective glasses.

3. For the **saponification**, add 10 mL **2.5% KOH in Methanol Solution** as well as 100 µL **IS Solution** to each sample and close the Xpress vessels with the key provided.

NOTE 1: Once sample preparation is complete, the concentration of the IS is 0.1 mg/ml.
NOTE 2: Confirm that all vessels are well closed and wiped dry on the outside as leaks/presence of solvent may damage the instrument. In addition, the instrument is fitted with a solvent sensor that will shut down the instrument if a leak/solvent is detected.

4. Load the carousel of the microwave so that the samples are well distributed with the carousel.
5. Switch on the microwave and add the carousel into the chamber; close the door.
6. On the touch screen, select *Classic Methods* → ***Saponification***; once the instrument has loaded the method, confirm the selection with Start.

NOTE 1: The instrument is setup to run the following sequence: heating to 130°C over 4 minutes with a hold at 130 °C for a further 4 minutes. If temperature was not hold for 4 minutes, run programme again.
NOTE 2: The sequence is set up to heat up to 20 vessels per run. If more than 20 vessels are loading into the carousel the temperature of 130 °C may not be reached. Hence, it is recommended to divide more than 20 vessels between 2 runs.

7. Once the programme is completed, remove the samples and cool in an ice bath for 5 minutes; if required, print the temperature profile of the saponification by selecting the "Printer Icon" on the touch screen.
8. For the **esterification**, add 15 mL **5% Acetyl Chloride in Methanol Solution** to each sample and close the Xpress vessels with the key provided.

NOTE: Confirm that all vessels are well closed and wiped dry on the outside as leaks/presence of solvent may damage the instrument. In addition, the instrument is fitted with a solvent sensor that will shut down the instrument if a leak/solvent is detected.

9. Load the carousel of the microwave so that the samples are well distributed with the carousel.
10. On the touch screen, select *Classic Methods* → ***Esterification***; once the instrument has loaded the method, confirm the selection with *Start*.

NOTE 1: The instrument is setup to run the following sequence: heating to 120 °C over 4 minutes with a hold at 120 °C for a further 2 minutes.
NOTE 2: The sequence is set up to heat up to 20 vessels per run. If more than 20 vessels are loading into the carousel the temperature of 130 °C may not be reached. Hence, it is recommended to divide more than 20 vessels between 2 runs.

11. Once the programme is completed, remove the samples and cool in an ice bath for 5 minutes; if required, print the temperature profile of the saponification by selecting the "Printer Icon" on the

touch screen.

NOTE: Once the programme is complete, the MW start the "cool down". You may stop the cool down once the temperature is 80C or below.

12. To extract the FAMEs, add 10 mL pentane to each vessel, refit the cap and turn the vessel 10 times.
13. Add approximately 20 mL of **Saturated NaCl Solution** to each sample, refit the cap, turn the vessel 10 times and allow the aqueous (bottom) and organic (top) layers to separate.

NOTE 1: Addition of Saturated NaCl Solution must be performed carefully due to the possible creation of a lot of gas resulting in the sample bubbling up.
NOTE 2: Depending on the sample matrix, this may take up to 30 min.

14. Remove approximately 1 mL from the pentane layer and transfer to a GC vial containing about 0.2 g anhydrous Na2SO4.

NOTE: It may be necessary to dilute samples. To determine whether this is required, the samples first need to be analysed and then re-analysed following dilution.

15. Empty all wash and waste vials on the GC and refill the wash vials with heptane.
16. Prepare one GC vial containing pentane with about 0.2 g anhydrous Na2SO4 to run as blank.

3.3 Sample Analysis

17. Analyse the samples using the proposed sequence given in Table 2 according to the chromatographic conditions given in Table 3. An example chromatogram is provided in Figure 1 while the elution order of the FAMEs and their retention time as well as relative retention time in relation to the internal standard is given in Table 4.

Table 2. Proposed sequence of analysis.

Solution	# of injections
Blank (heptane)	2
FAME Supelco standard for ID	1
PUFA standard for ID	1
Sample	1

Table 3. Chromatographic conditions for the analysis of FAMEs.

Parameter	Set point
GC	Agilent Technologies 7890B
Autosampler	Agilent Technologies 7693
Column	CP-Sil 88 (or equivalent) Length: 100 m; Internal diameter: 0.25 mm; film thickness: 0.2 µm.
Carrier gas type	Hydrogen
Carrier gas flow	1.25 ml/min (constant)
Injection temperature	250 °C

Injection volume	0.5 µl
Split ratio	10:1
Detector type	FID
Detector temperature	270 °C
Temperature gradient	

Rate	Temperature	Hold
	80 °C	N/A
6.2 °C/min	220 °C	3.2 min
6.3 °C/min	240 °C	6.5 min

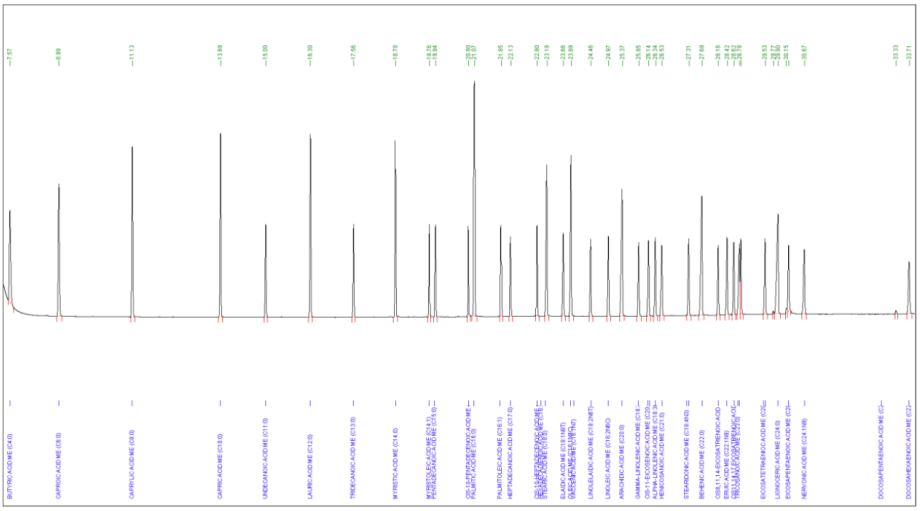


Figure 1. Example Chromatogram of the FAME standard obtained using the chromatographic conditions given in Table 3.

Table 4. Elution order of the FAMEs under the given chromatographic conditions.

Component	Lipid Name	RT [min]	RRT [min]
Butryic Acid Methyl Ester	C4:0	7.57	0.263
Caproic Acid Methyl Ester	C6:0	8.99	0.313
Caprylic Acid Methyl Ester	C8:0	11.13	0.387
Capric Acid Methyl Ester	C10:0	13.69	0.477
Undecanoic Acid Methyl Ester	C11:0	15.00	0.522
Lauric Acid Methyl Ester	C12:0	16.30	0.557
Tridecanoic Acid Methyl Ester	C13:0	17.56	0.611
Myristic Acid Methyl Ester	C14:0	18.78	0.654
Myristoleic Acid Methyl Ester	C14:1	19.76	0.688
Pentadecanoic Acid Methyl Ester	C15:0	19.94	0.694
cis-10-Pentadecenoic Acid Methyl Ester	C15:1	20.90	0.727
Palmitic Acid Methyl Ester	C16:0	21.07	0.733
Palmitoleic Acid Methyl Ester	C16:1n7	21.85	0.761
Heptadecanoic Acid Methyl Ester	C17:0	22.13	0.770
cis-10-Heptadecenoic Acid Methyl Ester	C17:1	22.90	0.797
Hexadecadienoic Acid Methyl Ester	C16:2n4	23.01	0.801
Stearic Acid Methyl Ester	C18:0	23.18	0.807
Elaidic Acid Methyl Ester	C18:1n9t	23.66	0.824
Oleic Acid Methyl Ester	C18:1n9c	23.89	0.832
Vaccenic Acid Methyl Ester	C18:1n7	23.96	0.834
Linolelaidic Acid Methyl Ester	C18:2n6t	24.46	0.851
Linoleic Acid Methyl Ester	C18:2n6c	24.97	0.869
Arachidic Acid Methyl Ester	C20:0	25.37	0.883
γ-Linolenic Acid Methyl Ester	C18:3n6	25.85	0.900
cis-11-Eicosenoic Acid Methyl Ester	C20:1n9	26.14	0.910
cis-8,11,14-OctadecatrienoicAcid Methyl Ester	C18:3n4	26.17	0.911
α-Linolenic Acid Methyl Ester	C18:3n3	26.34	0.917
Henicosanoic Acid Methyl Ester	C21:0	26.53	0.923
Stearidonic Acid Methyl Ester	C18:4n3	27.25	0.948
cis-11,14-Eicosadienoic Acid Methyl Ester	C20:2	27.31	0.951
Behenic Acid Methyl Ester	C22:0	27.69	0.964
cis-8,11,14-Eicosatrienoic Acid Methyl Ester	C20:3n6	28.16	0.980
Erucic Acid Methyl Ester	C22:1n9	28.42	0.989
cis-11,14,17-Eicosatrienoic Acid Methyl Ester	C20:3n3	28.62	0.996
Tricosanoic Acid Methyl Ester	C23:0	28.73	
Arachidonic Acid Methyl Ester	C20:4n6	28.81	1.003
cis-8,11,14,17-Eicosatetraenoic Acid Methyl Ester	C20:4n3	29.48	1.026
cis-13,16-Docosadienoic Acid Methyl Ester	C22:2	29.53	1.028
Lignoceric Acid Methyl Ester	C24:0	29.90	1.041
cis-5,8,11,14,17-Eicosapentaenoic Acid Methyl Ester	C20:5n3	30.21	1.052
Nervonic Acid Methyl Ester	C24:1n9	30.67	1.068
cis-7,10,13,16,19-Docosapentaenoic Acid Methyl Ester	C22:5n3	32.91	1.145
cis-4,7,10,13,16,19-Docosahexaenoic Acid Methyl Ester	C22:6n3	33.71	1.173

3.4 Sample calculation

1. Calculate the content of each fatty acid according to the following equation with an explanation of all variables given in Table 5.

$$\frac{PeakArea(FAME)}{PeakArea(ISTD)} \times \frac{Weight(ISTD)}{WeightSample} \times ISTDpurity \times 10 \times 0.96 = Content$$

Table 5. Equation variables

Variable	Explanation
PeakAreaSample	Peak Area obtained for FAME of interest
PeakAreaISTD	Peak Area obtained for the internal standard
WeightISTD	Weight of the internal standard in g
WeightSample	Weight of the sample in g
ISTDpurity	Purity of the internal standard as given on the certificate of analysis in decimal notation, e.g. 0.99 for 99% purity
10	Dilution factor
0.96*	Conversion factor for the internal standard which is already a FAME
Content	Content of the fatty acid in the sample in mg/g

Appendix 4.2. Dry matter sampling protocol.

Dry matter analysis protocol:

1. The oven was required to remain at a temperature of 105°C throughout.
2. Porcelain crucible containers were cleaned, dried within the oven for 1-2h, then placed inside a desiccator to absorb moisture and cool for 5-10min or until temperature reached ≤ 25°C.
3. Duplicates of each sample (79; a total of 158) were extracted (using a micro spoon) from the remaining food content within the 100g/ml bottles previously prepared for the fatty acid analysis.
4. The prepared (clean, dried, empty) crucible weight(s) were noted and crucible was numerically identified.
5. 2g of food sample was transferred to their identifying crucible and placed inside the oven overnight (for approximately 16h) before repeating the moisture absorption/cooling desiccator protocol.
6. The weight(s) of each sample in their respective crucible were recorded, before subtracting the original empty crucible weight to obtain sample dry weight.

Appendix 4.3. Dry weight of food items within the dataset.

Food sample	Dry weight (g)	Food sample	Dry weight (g)
Mr Hughs Extra Virgin Rapeseed Oil	0.999	Stork Margarine	0.722
KTC 100% Pure Coconut Oil	0.998	Ghana Fresh Palm Nut Cream Concentrate	0.693
Carotino Red Palm Oil + Rapeseed Oil	0.998	Ghana Taste Palm Fruit Pulp Extract	0.686
Napolina Extra Virgin Olive Oil	0.998	Heinz Vegan Mayonnaise	0.668
KTC 100% Pure Unrefined Palm Oil	0.997	Jacksons Palm Oil Free Multigrain Bread	0.648
Mystic Moments Palm Kernel Oil	0.997	Warburtons The Big 21 Seeds + Grains Bread	0.616
Tesco Sunflower Oil	0.997	Creamfields Cheddar Cheese	0.616
Tesco Sesame Oil	0.996	Tesco Goats Cheese	0.616
Tropical Sun Pure Palm Oil	0.996	Tesco Olive Spread	0.615
Tesco Tahini	0.990	Dairypak Butter	0.606
Nomo Vegan Chocolate Bar	0.986	Plant Menu Chicken Burger	0.568
Cadbury Dairy Milk Chocolate	0.985	Sacla Pesto (Basil)	0.530
Pip and Nut Almond Butter	0.981	Dairy Milk Ice Cream	0.509
Meridian Peanut Butter	0.980	Violife Vegan Cheese	0.497
Tesco Walnuts	0.979	Milano Margherita Pizza	0.492
Tesco Peanut Butter	0.977	Plant-It Chicken-free Vegan Nuggets	0.475
Nairn's Oatcakes	0.976	Tesco Feta Cheese	0.468
Eat Real Veggie Straws	0.975	Heinz Vegan Salad Cream	0.457
Tesco Brazil Nuts	0.975	Wicked Kitchen Vegan Ice Cream	0.455
Walkers Ready Salted Crisps	0.973	Tesco Beef Burger	0.445
Lizi's Granola	0.970	Better Naked Vegan Bacon Rashers	0.444
Lindt 85% Cocoa Dark Chocolate	0.963	Vemondo Vegan Burger	0.443
Tesco Peanuts	0.963	Plant Chef Vegan Pie	0.441
Tesco Cashew Nuts	0.956	Tesco Houmous	0.432
Thai Boy Palm Sugar	0.955	Tesco Pork Sausages	0.420
Tesco Almonds	0.941	Goodfella's Vegan Pizza	0.377
Wicked Kitchen Vegan Chocolate Cookie	0.923	Elmlea Double Cream	0.367
Trek Protein Flapjack	0.918	Elmlea Vegan Double Cream	0.357
Oggs Vegan Brownie Bites	0.908	Henry Denny & Sons Vegan Mince	0.342

Tesco Sunflower Seeds	0.906	Tesco Beef Mince (10%)	0.336
Nakd Cocoa Orange Fruit + Nut Bar	0.846	Better Naked Bacon Rashers	0.325
Naturli Vegan 'Palm-oil-free' Spread	0.794	Tesco Coleslaw	0.312
Flora Vegan Butter	0.792	Philadelphia Vegan Soft Cheese Spread	0.299
Tesco Parmesan Cheese	0.751	Tesco Avocado	0.246
Ghana Best Palm Nut Soup	0.730	Tesco Greek Yoghurt	0.182

Appendix 4.4. Fatty acid composition of edible oils (dry matter content).

FAs [mg/g]	Olive Oil	Coconut Oil	Sesame Oil	Red Palm & Rapeseed Oil	Sunflower Oil	Rapeseed Oil
C4:0	nd	nd	nd	nd	nd	nd
C6:0	nd	nd	nd	nd	nd	nd
C8:0	nd	nd	nd	0.15	nd	nd
C10:0	nd	56.97	nd	0.21	nd	0.09
C11:0	nd	0.26	nd	nd	nd	nd
C12:0	nd	472.39	nd	1.40	nd	0.11
C13:0	nd	0.37	nd	nd	nd	nd
C14:0	0.29	195.19	0.45	3.78	0.79	0.51
C15:0	0.04	0.13	nd	0.29	0.14	0.18
C16:0	**119.50**	**101.11**	**97.93**	**135.56**	**59.85**	**41.34**
C17:0	0.95	0.16	0.58	0.77	0.40	0.54
C18:0	38.50	30.25	51.87	25.09	33.31	13.97
C20:0	4.92	1.00	5.84	5.43	2.48	5.11
C21:0	0.33	nd	0.20	0.25	0.08	0.17
C22:0	1.38	nd	2.52	2.54	7.54	3.07
C24:0	0.68	nd	1.46	1.39	2.62	1.16
C14:1	nd	nd	nd	nd	nd	nd
C15:1n5c	nd	nd	nd	nd	nd	nd
C16:1n7c	7.99	0.20	2.07	2.17	0.96	1.91
C17:1n7c	nd	nd	nd	nd	nd	nd
C18:1n7c	21.20	1.07	nd	nd	nd	nd

	C18:1n9t	nd	0.57	1.03	nd	nd	nd
	C18:1n9c	845.36	70.67	418.19	557.95	288.78	563.73
	C20:1n9c	3.15	0.47	2.31	12.96	1.81	12.65
	C22:1n9c	nd	nd	0.05	0.47	0.13	2.14
	C24:1n9c	nd	nd	nd	1.31	0.22	1.80
	C18:2n6t	nd	nd	0.17	nd	0.04	nd
	C18:2n6c	88.03	18.21	458.07	188.22	584.81	196.22
	C18:3n3	8.21	0.11	3.69	63.15	1.68	101.80
	C18:3n6c	nd	nd	nd	nd	nd	nd
	C20:2	0.55	nd	0.75	1.34	0.23	1.45
	C20:3n3c	nd	nd	nd	nd	nd	0.08
	C20:3n6c	nd	nd	nd	nd	nd	nd
	C20:4n6	nd	nd	nd	nd	nd	nd
	C20:5n3c	nd	0.09	nd	nd	nd	nd
	C22:2n6c	nd	nd	nd	nd	nd	nd
	C22:5n3c	nd	nd	nd	nd	nd	nd
	C22:6n3c	nd	nd	nd	nd	nd	nd
	SFAs	166.60	857.82	160.84	176.86	107.21	66.24
	MUFAs	877.70	72.99	423.66	574.87	291.90	582.23
	PUFAs	96.79	18.41	462.69	252.71	586.76	299.55
	Data is presented as milligram (mg) total fatty acid (FA) content per 1 gram (g) dried food. 'nd' indicates there were no FAs determined/detected. SFAs = saturated fatty acids. MUFAs = mono-unsaturated fatty acids. PUFAs = poly-unsaturated fatty acids.						

Appendix 4.5. Fatty acid composition of dairy-based foods (dry matter content).

FAs [mg/g]	Butter	Margarine	Cheddar	Feta	Parmesan	Goat Cheese	Greek Yoghurt	Ice Cream	Double Cream	Coleslaw
C4:0	nd	nd	1.43	0.64	0.71	0.48	0.38	0.03	nd	0.07
C6:0	nd	0.30	5.69	4.89	4.83	5.04	1.70	0.50	nd	0.41
C8:0	nd	4.15	4.64	6.26	3.92	7.74	1.32	5.43	nd	0.33
C10:0	6.18	4.18	12.41	21.92	10.67	33.19	3.07	5.00	13.70	0.88
C11:0	0.12	0.06	0.32	0.28	0.33	0.57	0.05	0.02	0.11	nd
C12:0	20.55	58.18	16.33	13.92	14.10	21.03	4.26	nd	115.32	1.21
C13:0	0.15	0.08	0.19	0.40	0.54	0.22	0.12	0.05	0.13	0.06
C14:0	28.58	23.64	52.50	33.55	47.93	46.24	14.47	18.78	47.32	4.07
C15:0	2.54	0.34	5.80	2.92	5.71	4.41	1.38	0.19	0.13	0.49
C16:0	**125.27**	**200.66**	**130.62**	**80.77**	**134.76**	**127.47**	**46.11**	**28.11**	**36.50**	**30.38**
C17:0	1.65	0.63	3.53	2.53	3.60	3.31	1.18	0.22	0.23	0.49
C18:0	31.23	26.12	48.74	29.07	46.19	30.94	20.17	21.34	61.45	10.88
C20:0	2.44	4.07	0.66	0.72	0.76	0.67	0.29	0.66	1.83	2.42
C21:0	1.92	nd	nd	1.35	1.58	1.63	0.79	0.08	nd	0.13
C22:0	nd	nd	0.28	0.30	0.34	0.18	0.13	0.14	0.01	1.28
C24:0	nd	nd	0.21	0.11	0.22	0.07	0.08	0.11	nd	0.47
C14:1	2.53	nd	4.53	0.50	3.95	1.04	1.02	0.13	0.06	0.33
C15:1n5c	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C16:1n7c	4.86	1.35	6.90	2.31	6.31	3.64	2.61	0.34	0.42	1.58
C17:1n7c	nd	nd	nd	nd	nd	nd	nd	nd	0.03	nd
C18:1n7c	10.26	16.35	0.45	0.60	1.08	0.82	nd	nd	3.75	14.71

	C18:1n9t	nd	nd	0.03	0.03	0.06	0.03	0.48	nd	nd	nd
	C18:1n9c	249.15	406.31	85.84	61.74	88.75	82.07	33.67	26.00	85.83	234.95
	C20:1n9c	4.19	7.41	0.23	0.22	nd	nd	nd	nd	1.12	nd
	C22:1n9c	nd	nd	0.13	0.03	0.05	0.04	nd	nd	nd	0.53
	C24:1n9c	nd	nd	0.12	0.04	0.03	0.06	nd	nd	nd	0.82
	C18:2n6t	nd	nd	0.04	0.02	nd	nd	nd	nd	nd	nd
	C18:2n6c	76.01	123.43	5.89	7.71	11.42	9.32	2.21	3.99	26.46	92.64
	C18:3n3	30.72	41.01	2.86	1.83	2.63	1.38	0.38	0.24	8.65	43.26
	C18:3n6c	0.86	0.90	0.15	0.22	0.25	0.19	0.44	nd	0.48	nd
	C20:2	nd	nd	0.22	0.12	0.21	0.16	0.09	0.03	nd	0.54
	C20:3n3c	nd	0.02	0.06	0.03	0.04	0.03	nd	nd	nd	0.04
	C20:3n6c	0.04	nd	0.36	0.10	0.57	0.11	0.08	0.01	nd	0.04
	C20:4n6	nd	nd	0.40	0.74	0.77	0.69	0.17	0.02	nd	0.14
	C20:5n3c	0.17	nd	0.35	0.17	0.23	0.16	0.21	0.00	nd	0.01
	C22:2n6c	nd	nd	0.21	nd	nd	nd	nd	nd	nd	nd
	C22:5n3c	nd	nd	0.53	0.39	nd	nd	nd	nd	nd	nd
	C22:6n3c	nd	nd	0.04	0.39	nd	nd	nd	nd	nd	nd
	SFAs	220.63	322.42	283.35	199.62	276.17	283.20	95.51	80.67	276.75	53.56
	MUFAs	270.99	431.42	98.23	65.48	100.23	87.69	37.78	26.47	91.22	252.92
	PUFAs	107.80	165.37	11.11	11.71	16.13	12.03	3.58	4.30	35.59	136.67
	Data is presented as milligram (mg) total fatty acid (FA) content per 1 gram (g) dried food. 'nd' indicates there were no FAs determined/detected. SFAs = saturated fatty acids. MUFAs = mono-unsaturated fatty acids. PUFAs = poly-unsaturated fatty acids.										

Appendix 4.6. Fatty acid composition of nut/seed-based food items (dry matter content).

FAs [mg/g]	Brazil Nut	Cashew	Almond	Peanut	Walnut	Peanut Butter	PO Free Peanut Butter	Almond Butter	Sunflower Seed(s)	Tahini
C4:0	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C6:0	nd	nd	nd	nd	nd	0.02	nd	nd	nd	nd
C8:0	nd	nd	nd	nd	nd	nd	nd	nd	0.01	nd
C10:0	nd	nd	nd	nd	nd	nd	nd	nd	0.02	nd
C11:0	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C12:0	0.03	nd	0.03	nd	0.02	0.05	0.01	0.03	0.17	0.03
C13:0	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C14:0	0.32	0.12	0.22	0.16	0.10	0.25	0.10	0.27	0.48	0.17
C15:0	0.08	0.06	0.07	0.06	0.06	0.04	0.03	0.07	0.10	0.03
C16:0	**89.02**	**42.59**	**30.15**	**50.03**	**24.09**	**32.72**	**29.38**	**35.94**	**38.98**	**84.78**
C17:0	0.56	0.77	0.42	0.49	0.23	0.45	0.46	0.41	0.31	0.51
C18:0	62.37	39.68	8.73	11.94	10.42	7.53	10.27	7.60	23.65	55.25
C20:0	1.65	2.79	0.49	6.14	nd	3.78	5.03	0.45	1.81	6.05
C21:0	0.08	0.03	0.12	0.11	nd	0.08	0.12	0.12	0.09	0.13
C22:0	0.43	0.60	0.15	13.86	nd	10.46	11.35	0.15	5.41	1.30
C24:0	0.20	0.73	0.09	7.14	nd	7.46	6.62	0.09	1.92	0.82
C14:1	nd	nd	0.01	nd	nd	nd	nd	nd	nd	nd
C15:1n5c	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C16:1n7c	2.01	1.45	2.25	0.17	0.25	0.23	0.24	2.37	0.70	1.07
C17:1n7c	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C18:1n7c	nd	0.74	5.99	nd	nd	1.84	1.62	6.67	3.69	6.87

C18:1n9t	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C18:1n9c	186.92	266.59	325.46	165.60	52.76	277.46	267.36	338.34	263.13	367.91
C20:1n9c	0.43	0.83	0.47	5.38	0.90	10.32	7.26	0.46	1.03	1.70
C22:1n9c	0.02	nd	nd	0.40	nd	1.31	0.72	nd	0.02	0.02
C24:1n9c	0.08	nd	0.04	0.24	nd	0.21	0.12	0.15	0.27	0.01
C18:2n6t	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C18:2n6c	243.34	81.01	117.64	184.73	227.79	31.80	58.04	154.49	0.03	378.07
C18:3n3	0.54	0.72	0.16	0.34	53.39	0.38	0.24	0.15	0.28	2.87
C18:3n6c	nd	nd	nd	nd	0.25	0.02	nd	nd	nd	nd
C20:2	nd	nd	nd	0.33	nd	nd	nd	nd	nd	nd
C20:3n3c	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C20:3n6c	nd	nd	nd	0.01	nd	0.04	0.01	nd	nd	nd
C20:4n6	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C20:5n3c	nd	nd	nd	0.01	nd	nd	nd	0.02	0.02	nd
C22:2n6c	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C22:5n3c	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C22:6n3c	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
SFAs	154.72	87.37	40.47	89.92	34.93	62.84	63.36	45.14	72.94	149.07
MUFAs	189.46	269.62	334.21	171.79	53.91	291.37	277.32	347.99	268.83	377.58
PUFAs	243.89	81.72	117.80	185.41	281.43	32.23	58.29	154.66	0.33	380.94

Data is presented as milligram (mg) total fatty acid (FA) content per 1 gram (g) dried food.

'nd' indicates there were no FAs determined/detected.

SFAs = saturated fatty acids.

MUFAs = mono-unsaturated fatty acids.

PUFAs = poly-unsaturated fatty acids.

Appendix 4.7. Fatty acid composition of animal-origin foods (dry matter content).

FAs [mg/g]	Beef Mince	Beef Burger	Pork Sausage	Bacon Rasher
C4:0	nd	nd	nd	nd
C6:0	nd	nd	nd	nd
C8:0	nd	nd	0.02	0.02
C10:0	nd	nd	0.18	0.15
C11:0	nd	nd	nd	nd
C12:0	0.19	0.22	0.20	0.17
C13:0	0.04	0.08	nd	nd
C14:0	5.51	9.23	2.92	2.00
C15:0	1.09	1.87	0.12	0.11
C16:0	**52.04**	**86.04**	**53.25**	**38.48**
C17:0	2.48	4.06	0.73	0.58
C18:0	37.99	62.89	29.21	18.49
C20:0	nd	0.57	0.42	0.29
C21:0	nd	nd	0.09	0.06
C22:0	0.08	nd	0.04	0.04
C24:0	nd	nd	0.03	0.04
C14:1	1.08	2.05	0.04	0.05
C15:1n5c	0.06	0.03	nd	0.04
C16:1n7c	6.66	11.30	5.10	5.66
C17:1n7c	0.01	nd	nd	nd
C18:1n7c	2.55	3.47	nd	nd
C18:1n9t	nd	nd	nd	nd

	C18:1n9c	74.99	129.27	85.92	65.79
	C20:1n9c	nd	0.68	1.79	1.11
	C22:1n9c	nd	nd	0.06	0.06
	C24:1n9c	nd	nd	nd	nd
	C18:2n6t	0.01	0.02	nd	nd
	C18:2n6c	3.70	5.09	31.86	26.63
	C18:3n3	1.01	1.64	2.39	1.82
	C18:3n6c	0.10	0.14	0.13	0.14
	C20:2	nd	nd	1.45	1.02
	C20:3n3c	nd	nd	0.39	0.24
	C20:3n6c	nd	nd	0.28	0.31
	C20:4n6	nd	nd	0.94	1.46
	C20:5n3c	0.18	0.15	0.05	0.06
	C22:2n6c	nd	nd	0.05	0.05
	C22:5n3c	nd	nd	0.26	0.28
	C22:6n3c	nd	nd	0.07	0.10
	SFAs	99.41	164.97	87.23	60.44
	MUFAs	85.35	146.79	92.93	72.70
	PUFAs	5.00	7.04	37.87	32.10
	Data is presented as milligram (mg) total fatty acid (FA) content per 1 gram (g) dried food. 'nd' indicates there were no FAs determined/detected. SFAs = saturated fatty acids. MUFAs = mono-unsaturated fatty acids. PUFAs = poly-unsaturated fatty acids.				

Appendix 4.8a. Fatty acid composition of plant-based/vegan food alternatives (dry matter content).

FAs [mg/g]	Mayonnaise (v)	Salad Cream (v)	Chocolate Cookie (v)	Chocolate (v)	Butter (v)	Ice Cream (v)	Mince (v)	'Chicken' Nuggets (v)	Soft Cheese (v)
C4:0	nd	nd	nd	nd	nd	nd	nd	nd	nd
C6:0	nd	0.07	0.03	0.06	nd	nd	nd	nd	nd
C8:0	nd	nd	nd	nd	nd	nd	nd	nd	nd
C10:0	0.05	0.05	0.05	nd	nd	6.54	nd	nd	24.35
C11:0	nd	nd	nd	0.06	0.10	0.07	0.00	0.01	0.12
C12:0	0.08	0.07	0.52	0.05	113.40	58.81	9.18	0.03	0.02
C13:0	nd	nd	nd	nd	0.14	0.05	0.02	nd	0.16
C14:0	0.43	0.34	2.44	0.36	41.09	24.70	4.22	0.18	91.16
C15:0	0.14	0.14	0.16	0.10	0.17	0.05	0.10	0.04	0.07
C16:0	**30.15**	**24.92**	**114.31**	**90.07**	**123.32**	**31.83**	**17.82**	**13.55**	**51.81**
C17:0	0.52	1.11	1.16	1.46	0.32	0.81	0.25	0.15	0.11
C18:0	13.24	9.99	24.87	133.51	19.06	19.17	38.91	5.76	16.14
C20:0	nd	3.38	1.67	4.27	nd	nd	2.37	0.69	nd
C21:0	0.21	0.13	0.02	0.07	nd	nd	nd	nd	nd
C22:0	2.21	1.86	0.44	0.77	nd	nd	nd	nd	nd
C24:0	0.88	0.84	0.34	0.44	nd	nd	nd	nd	nd
C14:1	nd	nd	nd	nd	nd	nd	nd	nd	nd
C15:1n5c	nd	nd	nd	nd	0.02	nd	nd	nd	nd
C16:1n7c	1.48	1.21	0.57	0.89	0.44	0.15	0.44	0.25	0.26
C17:1n7c	nd	nd	nd	nd	nd	nd	nd	nd	nd
C18:1n7c	nd	16.53	3.52	1.31	2.39	nd	5.28	2.89	1.04

C18:1n9t	nd	nd	0.10	nd	nd	0.09	nd	nd	0.12
C18:1n9c	405.59	330.35	134.34	129.68	133.70	33.49	132.34	60.64	61.16
C20:1n9c	8.84	9.42	1.25	0.33	nd	nd	2.78	nd	nd
C22:1n9c	0.75	8.69	0.06	nd	nd	nd	nd	nd	nd
C24:1n9c	1.31	1.40	0.13	nd	nd	nd	nd	nd	nd
C18:2n6t	nd	nd	nd	nd	nd	nd	0.03	0.04	nd
C18:2n6c	150.80	107.88	38.39	13.61	51.67	8.16	49.17	83.83	18.22
C18:3n3	72.38	46.05	4.93	0.66	1.47	0.65	15.03	6.57	0.06
C18:3n6c	nd	nd	0.23	nd	0.12	0.03	0.47	0.33	nd
C20:2	1.08	0.99	nd	nd	nd	nd	nd	nd	nd
C20:3n3c	0.06	0.07	nd	nd	nd	nd	nd	nd	nd
C20:3n6c	nd	nd	nd	nd	nd	nd	nd	nd	nd
C20:4n6	nd	nd	nd	nd	nd	nd	nd	nd	nd
C20:5n3c	nd	nd	nd	nd	nd	nd	nd	0.02	nd
C22:2n6c	nd	nd	nd	nd	nd	nd	nd	nd	nd
C22:5n3c	nd	nd	nd	nd	nd	nd	nd	nd	nd
C22:6n3c	nd	nd	nd	nd	nd	nd	nd	nd	nd
SFAs	47.92	42.89	146.00	231.21	297.60	142.04	72.87	20.42	183.94
MUFAs	417.96	367.59	139.96	132.22	136.55	33.73	140.84	63.78	62.58
PUFAs	224.32	154.98	43.54	14.27	53.26	8.84	64.70	90.79	18.28

Data is presented as milligram (mg) total fatty acid (FA) content per 1 gram (g) dried food.

'nd' indicates there were no FAs determined/detected.

SFAs = saturated fatty acids.

MUFAs = mono-unsaturated fatty acids.

PUFAs = poly-unsaturated fatty acids.

Appendix 4.8b. Fatty acid composition of plant-based/vegan food alternatives (dry matter content).

FAs [mg/g]	Pie (v)	Burger (v)	Cheese (v)	'Chicken' Breast (v)	Brownie (v)	Bacon Rasher (v)	Pizza (v)	PO Free Spread (v)	Double Cream (v)
C4:0	nd	nd	nd	nd	nd	nd	nd	nd	nd
C6:0	0.03	0.39	0.87	nd	0.04	nd	nd	0.39	0.15
C8:0	0.00	5.90	13.34	0.06	0.38	0.05	nd	6.21	3.61
C10:0	nd	5.23	11.60	0.04	0.46	0.06	nd	5.73	5.73
C11:0	0.03	0.03	0.06	nd	nd	nd	nd	0.03	0.08
C12:0	0.34	nd	nd	0.21	6.18	0.06	0.04	47.79	nd
C13:0	nd	0.04	0.07	nd	0.01	nd	nd	0.04	0.14
C14:0	1.72	21.28	40.85	0.97	3.92	0.16	0.08	20.01	48.95
C15:0	0.12	0.03	0.04	0.10	0.07	0.07	0.03	0.11	0.17
C16:0	**78.52**	**17.37**	**26.36**	**51.60**	**53.48**	**11.58**	**5.27**	**32.10**	**111.65**
C17:0	0.24	0.05	0.04	0.20	0.17	0.14	0.06	0.33	0.31
C18:0	7.74	5.68	6.85	0.04	11.31	3.86	1.45	73.78	16.72
C20:0	nd	0.28	0.25	1.21	0.82	1.16	0.30	4.17	1.82
C21:0	nd	nd	nd	0.06	nd	nd	nd	nd	nd
C22:0	nd	0.40	0.07	1.54	0.26	0.62	0.26	1.31	0.81
C24:0	nd	0.40	0.10	0.59	0.16	0.32	0.12	0.66	0.41
C14:1	nd	nd	nd	nd	nd	nd	nd	nd	nd
C15:1n5c	nd	0.02	nd	nd	nd	nd	0.01	nd	nd
C16:1n7c	0.30	0.08	0.06	0.42	0.27	0.57	0.16	0.82	0.55
C17:1n7c	nd	nd	nd	nd	nd	nd	nd	nd	nd
C18:1n7c	1.87	nd	nd	nd	nd	6.57	nd	12.00	8.54

C18:1n9t	nd	nd	0.28	nd	nd	nd	0.03	nd	nd
C18:1n9c	68.77	24.59	19.17	112.58	67.70	114.48	26.90	276.25	161.37
C20:1n9c	nd	0.19	nd	nd	nd	nd	nd	nd	nd
C22:1n9c	nd	0.02	nd	0.25	0.14	0.24	0.15	1.74	0.70
C24:1n9c	nd	0.02	nd	0.14	0.08	0.29	0.08	0.61	0.38
C18:2n6t	0.01	0.01	nd	nd	nd	nd	nd	nd	nd
C18:2n6c	25.56	30.22	5.45	122.58	20.40	43.60	21.78	81.98	49.32
C18:3n3	2.49	0.41	0.11	6.81	4.02	13.61	3.69	27.05	18.33
C18:3n6c	0.11	nd	nd	nd	nd	nd	0.15	nd	nd
C20:2	0.02	0.03	0.02	0.23	0.14	0.24	0.06	0.80	0.35
C20:3n3c	nd	nd	nd	nd	nd	0.01	nd	0.02	0.01
C20:3n6c	nd	nd	nd	nd	nd	nd	nd	nd	nd
C20:4n6	nd	nd	nd	nd	nd	nd	nd	nd	nd
C20:5n3c	0.01	nd	nd	nd	nd	nd	nd	nd	nd
C22:2n6c	nd	nd	nd	nd	nd	nd	nd	nd	nd
C22:5n3c	nd	nd	nd	nd	nd	nd	nd	nd	nd
C22:6n3c	nd	nd	nd	nd	nd	nd	nd	nd	nd
SFAs	88.73	57.07	100.50	56.63	77.26	18.06	7.61	192.66	190.54
MUFAs	70.94	24.92	19.51	113.39	68.20	122.16	27.33	291.41	171.54
PUFAs	28.20	30.67	5.59	129.62	24.56	57.45	25.69	109.85	68.01

Data is presented as milligram (mg) total fatty acid (FA) content per 1 gram (g) dried food.

'nd' indicates there were no FAs determined/detected.

SFAs = saturated fatty acids.

MUFAs = mono-unsaturated fatty acids.

PUFAs = poly-unsaturated fatty acids.

Appendix 4.9. Fatty acid composition of miscellaneous/snack food items (dry matter content).

FAs [mg/g]	Ready Salted Crisps	Vegetable Straw Crisps	Milk Chocolate	Dark Chocolate	Fruit & Nut Bar	Protein Flapjack	Oatcake	Seeds & Grains Bread	PO Free Bread	Pizza	Granola	Avocado	Pesto	Olive Spread
C4:0	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C6:0	nd	nd	nd	nd	0.14	0.09	nd	0.02	0.03	nd	nd	nd	nd	0.14
C8:0	0.02	nd	0.61	nd	0.05	nd	nd	0.01	0.01	nd	nd	nd	nd	1.22
C10:0	0.01	0.03	1.57	nd	0.02	1.09	0.03	nd	nd	nd	3.23	nd	0.84	1.31
C11:0	nd	nd	0.10	nd	0.02	0.06	nd	0.02	0.04	0.07	0.03	nd	0.04	nd
C12:0	0.02	0.04	2.28	0.06	0.04	23.48	0.34	0.03	0.02	2.27	26.48	0.02	1.15	16.08
C13:0	nd	nd	0.07	nd	0.02	0.04	nd	nd	nd	0.10	0.02	nd	0.08	0.03
C14:0	0.15	0.17	7.76	0.55	0.08	11.12	1.68	0.09	0.06	7.08	10.34	0.17	4.17	8.27
C15:0	0.05	0.08	0.86	0.20	0.05	0.08	0.09	0.05	0.03	0.79	0.05	0.03	0.53	nd
C16:0	**12.86**	**15.71**	**92.58**	**165.63**	**21.56**	**54.15**	**79.49**	**10.42**	**6.88**	**22.54**	**25.40**	**78.25**	**53.71**	**139.11**
C17:0	0.14	0.23	2.36	1.85	0.37	0.45	0.22	0.09	0.07	0.49	0.23	0.09	0.56	0.56
C18:0	8.58	6.28	79.94	241.74	19.87	8.73	7.56	3.78	2.82	7.74	8.86	2.16	26.59	22.61
C20:0	1.17	1.96	2.58	7.70	1.34	0.90	0.70	0.28	0.12	nd	0.94	0.26	1.89	3.19
C21:0	0.11	0.25	0.35	0.11	0.02	0.02	0.02	0.03	nd	nd	0.04	0.21	0.22	0.17
C22:0	2.50	1.23	0.49	1.34	0.32	0.43	0.17	0.24	0.09	nd	0.60	0.78	4.68	1.25
C24:0	0.93	0.42	0.31	0.79	0.38	0.23	0.23	0.17	0.08	nd	0.36	0.24	1.73	0.65
C14:1	nd	nd	0.67	nd	0.01	nd	nd	nd	nd	0.61	nd	nd	0.35	0.07
C15:1n5c	nd	nd	nd	nd	0.06	0.01	nd	nd	nd	nd	nd	nd	nd	0.25
C16:1n7c	0.43	0.67	1.77	1.55	0.72	0.32	0.38	0.30	0.20	1.20	0.49	28.32	1.76	2.77
C17:1n7c	nd	nd	nd	nd	0.02	nd	nd	nd	nd	nd	nd	nd	nd	nd

C18:1n7c	3.67	9.88	0.10	2.20	0.92	3.27	1.77	0.88	0.42	0.23	3.96	nd	4.46	15.64
C18:1n9t	nd	nd	nd	nd	nd	0.15	nd	0.49	0.02	0.03	nd	nd	nd	nd
C18:1n9c	244.74	200.08	98.10	208.86	126.12	80.39	94.23	18.64	7.10	22.04	119.91	278.46	230.37	385.49
C20:1n9c	1.93	5.86	0.32	0.31	0.44	1.64	1.03	0.42	0.22	nd	2.08	0.96	1.13	4.02
C22:1n9c	0.33	1.83	0.01	nd	0.01	nd	0.07	0.11	0.04	nd	0.27	0.04	0.04	0.56
C24:1n9c	0.18	0.69	0.03	nd	nd	0.24	0.16	0.15	0.09	nd	0.30	nd	0.25	0.59
C18:2n6t	0.01	0.04	nd	nd	0.01	nd	0.02	0.02	nd	nd	nd	nd	0.08	nd
C18:2n6c	27.63	60.97	11.40	18.84	39.07	38.88	47.50	29.09	16.25	8.04	71.01	45.04	311.73	84.85
C18:3n3	5.88	24.08	1.16	1.17	0.47	7.60	0.96	3.69	1.08	1.48	13.79	3.98	2.19	24.81
C18:3n6c	nd	nd	nd	nd	nd	0.36	0.05	0.05	0.02	0.09	0.39	nd	0.07	nd
C20:2	nd	0.65	nd	nd	nd	nd	nd	0.07	0.04	nd	nd	0.14	nd	0.45
C20:3n3c	nd	0.02	nd	nd	nd	nd	nd	nd	nd	nd	0.01	nd	nd	nd
C20:3n6c	nd	nd	0.06	nd	nd	nd	nd	nd	nd	nd	nd	nd	0.04	nd
C20:4n6	nd	nd	0.08	nd	nd	nd	nd	nd	nd	nd	nd	nd	0.06	nd
C20:5n3c	nd	nd	0.05	nd	nd	nd	0.01	0.01	nd	0.04	0.01	nd	nd	nd
C22:2n6c	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
C22:5n3c	nd	nd	0.06	nd	nd	nd	nd	nd	nd	nd	nd	nd	0.02	nd
C22:6n3c	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
SFAs	26.55	26.40	191.85	419.97	44.28	100.88	90.52	15.24	10.25	41.07	76.56	82.22	96.19	194.58
MUFAs	251.26	219.01	100.99	212.93	128.30	86.02	97.64	20.98	8.08	24.10	127.02	307.78	238.36	409.38
PUFAs	33.52	85.76	12.80	20.01	39.54	46.84	48.54	32.92	17.39	9.65	85.20	49.15	314.18	110.11

Data is presented as milligram (mg) total fatty acid (FA) content per 1 gram (g) dried food.

'nd' indicates there were no FAs determined/detected.

SFAs = saturated fatty acids.

MUFAs = mono-unsaturated fatty acids.

PUFAs = poly-unsaturated fatty acids.

Appendix 4.10. Fatty acid composition of palm-based foods (dry matter content).

FAs [mg/g]	Palm Sugar	Unrefined Palm Oil	Pure Palm Oil	Palm Kernel Oil	Palm Nut Cream	Palm Nut Soup	Palm Fruit Pulp
C4:0	nd	nd	nd	nd	nd	nd	nd
C6:0	0.04	nd	nd	nd	nd	nd	nd
C8:0	nd	nd	nd	nd	nd	nd	nd
C10:0	nd	0.16	0.11	19.99	nd	nd	nd
C11:0	0.05	nd	nd	0.26	nd	nd	nd
C12:0	0.01	1.56	2.53	293.21	1.79	2.61	1.65
C13:0	nd	nd	nd	0.34	nd	nd	nd
C14:0	0.00	7.16	10.96	103.76	5.38	5.34	5.44
C15:0	nd	0.51	0.55	0.10	0.26	0.28	0.27
C16:0	**0.10**	**421.92**	**470.00**	**57.04**	**256.79**	**246.74**	**245.46**
C17:0	1.88	1.02	1.18	0.16	0.62	0.59	0.61
C18:0	0.03	57.88	55.64	16.99	35.14	32.94	29.62
C20:0	nd	4.33	4.80	nd	2.74	2.58	2.41
C21:0	nd	nd	nd	nd	nd	nd	nd
C22:0	nd	nd	nd	nd	nd	nd	nd
C24:0	nd	nd	nd	nd	nd	nd	nd
C14:1	nd	nd	nd	nd	nd	nd	nd
C15:1n5c	0.01	nd	nd	nd	nd	nd	nd
C16:1n7c	nd	1.37	1.49	0.14	0.70	0.67	0.86
C17:1n7c	nd	nd	nd	nd	nd	nd	nd
C18:1n7c	nd	6.09	6.69	1.01	3.07	2.67	3.37

	C18:1n9t	nd	nd	nd	0.16	nd	nd	nd
	C18:1n9c	0.13	433.47	412.47	110.62	246.02	235.18	235.85
	C20:1n9c	nd	1.51	1.61	nd	0.91	0.92	0.83
	C22:1n9c	nd	nd	nd	nd	nd	nd	nd
	C24:1n9c	nd	nd	nd	nd	nd	nd	nd
	C18:2n6t	nd	nd	nd	nd	nd	0.02	nd
	C18:2n6c	0.06	107.22	109.03	17.49	62.89	57.44	62.43
	C18:3n3	0.00	3.27	3.38	0.40	1.86	1.65	1.91
	C18:3n6c	nd	nd	nd	0.02	0.01	0.02	0.02
	C20:2	nd	nd	nd	nd	nd	nd	nd
	C20:3n3c	nd	nd	nd	nd	nd	nd	nd
	C20:3n6c	nd	nd	nd	nd	nd	nd	nd
	C20:4n6	nd	nd	nd	nd	nd	nd	nd
	C20:5n3c	0.01	nd	nd	nd	nd	nd	nd
	C22:2n6c	nd	nd	nd	nd	nd	nd	nd
	C22:5n3c	nd	nd	nd	nd	nd	nd	nd
	C22:6n3c	nd	nd	nd	nd	nd	nd	nd
	SFAs	2.12	494.55	545.78	491.85	302.73	291.08	285.45
	MUFAs	0.14	442.44	422.26	111.94	250.71	239.43	240.91
	PUFAs	0.08	110.49	112.40	17.91	64.76	59.13	64.35
	Data is presented as milligram (mg) total fatty acid (FA) content per 1 gram (g) dried food. 'nd' indicates there were no FAs determined/detected. SFAs = saturated fatty acids. MUFAs = mono-unsaturated fatty acids. PUFAs = poly-unsaturated fatty acids.							

Appendix 4.11. Comparison of fresh weight (mg/g) of PA within food items between Chapter 4 and McCance and Widdowson's (CoFID) dataset.

Food item	Chapter 4		CoFID	
	C16:0 Palmitic acid ↓	%	C16:0 Palmitic acid	%
Pure Palm Oil	467.838	46.78%	418.000	41.80%
Unrefined Palm Oil	420.528	42.05%	nd	
Palm Nut Soup	181.110	18.11%	nd	
Palm Nut Cream	176.367	17.64%	nd	
85% Dark Chocolate	160.410	16.04%	272.000	27.20%
Palm Fruit Pulp Extract	150.946	15.09%	nd	
Margarine	150.882	15.09%	208.100	20.81%
Red Palm Oil + Rapeseed Oil	134.880	13.49%	nd	
Extra Virgin Olive Oil	119.253	11.93%	101.000	10.10%
Parmesan Cheese	101.545	10.15%	80.900	8.09%
Coconut Oil	100.844	10.08%	84.000	8.40%
Vegan Choc Cookie	98.047	9.80%	nd	
Sesame Oil	97.645	9.76%	86.000	8.60%
Vegan Butter	97.402	9.74%	nd	
Milk Chocolate	91.140	9.11%	85.300	8.53%
Vegan Chocolate	88.652	8.87%	nd	
Brazil Nuts	86.583	8.66%	nd	
Olive Spread	85.372	8.54%	91.400	9.14%
Tahini	84.040	8.40%	nd	
Cheddar Cheese	80.532	8.05%	86.800	8.68%
Goats Cheese	78.677	7.87%	67.900	6.79%
Oatcakes	77.536	7.75%	47.900	4.79%
Butter	75.920	7.59%	220.400	22.04%
Sunflower Oil	59.628	5.96%	62.000	6.20%
Palm Kernel Oil	56.934	5.69%	nd	
Protein Flapjack	49.537	4.95%	nd	
Vegan Brownie Bites	48.635	4.86%	nd	
Peanuts	48.118	4.81%	107.000	10.70%
Extra V Rapeseed Oil	41.263	4.13%	42.000	4.20%
Cashew Nuts	40.721	4.07%	nd	
Vegan Double Cream	40.166	4.02%	nd	
Beef Burger	37.820	3.78%	60.500	6.05%
Feta Cheese	36.787	3.68%	nd	
Sunflower Seeds	36.195	3.62%	nd	

Almond Butter	35.282	3.53%	nd	
Vegan Pie	34.763	3.48%	nd	
Peanut Butter	31.931	3.19%	69.300	6.93%
Vegan Chicken Burger	29.213	2.92%	nd	
PO Free Peanut Butter	28.807	2.88%	nd	
Almonds	28.368	2.84%	30.800	3.08%
Pesto	27.980	2.80%	37.000	3.70%
PO Free Vegan Spread	25.707	2.57%	nd	
Granola	24.621	2.46%	27.200	2.72%
Walnuts	23.416	2.34%	nd	
Pork Sausages	23.153	2.32%	nd	
Vegan Mayonnaise	20.169	2.02%	nd	
Avocado	19.317	1.93%	nd	
Fruit & Nut Bar	18.244	1.82%	nd	
Beef Mince (10%)	17.103	1.71%	37.200	3.72%
Vegan Soft Cheese	15.701	1.57%	nd	
Veggie Straw Crisps	15.305	1.53%	nd	
Vegan Ice Cream	14.402	1.44%	23.700	2.37%
Chocolate Ice Cream	14.342	1.43%	37.600	3.76%
Double Cream	13.571	1.36%	30.600	3.06%
Vegan Cheese	13.117	1.31%	nd	
Ready Salted Crisps	12.524	1.25%	nd	
Bacon Rashers	12.300	1.23%	51.000	5.10%
Vegan Salad Cream	11.494	1.15%	nd	
Margherita Pizza	11.129	1.11%	19.600	1.96%
Houmous	10.512	1.05%	nd	
Coleslaw	9.456	0.95%	9.100	0.91%
Greek Yoghurt	8.337	0.83%	31.200	3.12%
Vegan Burger	7.645	0.76%	nd	
Seeds & Grains Bread	6.454	0.65%	nd	
Vegan Chicken Nuggets	6.432	0.64%	nd	
Vegan Mince	6.138	0.61%	nd	
Vegan Bacon Rashers	5.210	0.52%	nd	
Palm Oil-Free Multi Grain Bread	4.470	0.45%	nd	
Vegan Pizza	1.987	0.20%	nd	
Palm Sugar	0.099	0.01%	nd	

nd = no data; no entry/no value present within the dataset. Food items are exact or closest comparable.

Appendix 4.12a. Source information of failed food samples.

	Food item	Brand/Supplier	Size	Source
Failed samples (9)	Chia Seeds	Bulk	500g	Tesco Superstore, Penwortham, Preston, England
	Mayonnaise	Tesco	350ml	Tesco Superstore, Penwortham, Preston, England
	Vegan Pesto	Tesco	190g	Tesco Superstore, Penwortham, Preston, England
	Coconut Milk	Blue Dragon	400ml	Tesco Superstore, Penwortham, Preston, England
	Organic Palm Oil	The Soapery	500g	Amazon.co.uk
	Zomi Palm Oil	Africa's Finest	500ml	JB Supplies Ltd, City Centre, Preston, England
	Soft Cheese Spread	Philadelphia	165g	Tesco Superstore, Dundrum, Dublin, Ireland
	Vegan Sausage Rolls	Golden Bake	330g	Tesco Superstore, Dundrum, Dublin, Ireland
	Vegan Sausages	Henry Denny & Sons	304g	Tesco Superstore, Dundrum, Dublin, Ireland

Appendix 4.12b. Sample preparation requirements of failed food samples.

	Food item	Fat Content (g/100g)	To Weigh	Sample a Weight (g)	Sample b Weight (g)	Sample weight (mean)
Failed samples (9)	Chia Seeds	31.0	0.5g	0.4920	0.5033	0.4977
	Mayonnaise	73.2	0.2g	0.2124	0.2132	0.2128
	Vegan Pesto	36.8	0.5g	0.5371	0.5634	0.5503
	Coconut Milk	15.0	0.8g	0.8004	0.7923	0.7964
	Organic Palm Oil	100.0	0.08g	0.0894	0.1008	0.0951
	Zomi Palm Oil	98.2	0.08g	0.0895	0.0936	0.0916
	Soft Cheese Spread	21.0	0.5g	0.5347	0.4934	0.5141
	Vegan Sausage Rolls	16.5	0.6g	0.7377	0.6169	0.6773
	Vegan Sausages	6.0	1g	0.9671	1.2214	1.0943

Appendix 5.1. Participant information sheet for the biomarkers assessment.

University of Central Lancashire
UCLan

Participant Information Sheet

1. Title of Study:
Palmitic acid (16:0) consumption in plant-based diets and its effect on metabolic health.

2. Document version
PIS Thomas Hothersall v3 (5/6/2023)

3. Invitation Paragraph
You are being invited to take part in this study. Before you decide, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with friends, relatives and your GP if you wish. Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part.

Thank you for reading this.

4. What is the purpose of the study?
The purpose of this study is to assess the status of metabolic and gut health in participants with different dietary palm oil/palmitic acid intake levels. Specifically, we would like to assess the metabolic health indicators/biomarkers associated with the body composition and inflammatory/oxidative stress in the participants with various levels of dietary palmitic acid intake. In addition, we would like to assess the status of gut microbial diversity in the participants with various levels of dietary palmitic acid intake.

5. Why have I been invited to take part?
The knowledge on palmitic acids' effects on human health is widely debated and its prevalence in plant-based diets is not well-documented. We hope to assess the health effect of consuming food containing palm oil/palmitic acid. We believe that your participation would help us answering a number of question relating to the health effect of palm oil/palmitic acid. In this study, we are expecting to collect a blood sample and faecal sample to analyse the effects on human health. We welcome your participation in this study if you fulfil the following eligibility criteria-

9. Must have previously (or before samples to be taken) provided a food diary (1 week) to determine PO intake.
10. Must declare taking any medication (that alters microbiome status or blood).
11. No pre-existing disease conditions and should be in good health - or if so, must declare.
12. Be on your respective diet (plant-based/vegan or omnivore) for >3 months.
13. Must be aged 18+, any gender, any ethnicity.
14. Able to clearly understand/communicate (reading and writing) in English.
15. Must read and fully understood the procedure(s) and all information provided on the participant information sheet (PIS).
16. Must have read, fully understood and signed the participant consent form.

6. Do I have to take part?
It is entirely up to you whether to take part or not in this study. If you decide to take part you will be given this information sheet and be asked to sign a consent form. If you participate in this study, you are still free to withdraw at any stage without giving a reason.

7. What will happen if I take part?

As a participant in this study, you will need to:

1. Visit the Nutrition Suite (Darwin Building) at the University of Central Lancashire on one occasion.
2. Anthropometry (body weight, height, skinfold, waist circumference) and body composition (Bioimpedance) measurements will be performed during this visit.
3. A trained member of the research team will collect one venous blood sample (10 ml) following approved standard procedure.
4. A single faecal sample of 1-2 grams will be collected. This will be done using the already provided pre-approved stool sampling container and each participant will bring in a sample taken prior to this visit.

8. How will my data be used?

The University processes personal data as part of its research and teaching activities in accordance with the lawful basis of 'public task', and in accordance with the University's purpose of "advancing education, learning and research for the public benefit".

Under UK data protection legislation, the University acts as the Data Controller for personal data collected as part of the University's research. The University privacy notice for research participants can be found on the attached link https://www.uclan.ac.uk/data_protection/privacy-notice-research-participants.php

Further information on how your data will be used can be found in the table below.

How will my data be collected?	The data on your body measurement (anthropometric measurement) will be performed by a researcher and the data on gene expression and microbial profile will be generated from analysing your blood and faecal samples following standard procedure.
How will my data be stored?	All digital data collected will be saved and stored on a password protected computer in Preston Campus of the University of Central Lancashire that has encryption and two-factor authentication facilities. The digital data will only be accessible by the lead researcher and supervisory team. The identification details of the sample(s) will be stored on a password-protected computer in the University of Central Lancashire Preston Campus with encryption and two-factor authentication facilities via Microsoft OneDrive.
How long will my data be stored for?	All personal contact details will be retained for 28 days following results. All personal identification details will be deleted on day 29 following the invitation of the participants for the debriefing process. All electronic data will be retained for a maximum of 7 years or until the completion of the publications from this study, whichever is earlier.

What measures are in place to protect the security and confidentiality of my data?	All data collected via email will be saved and stored, as well as password protected with two-factor authentication, via Microsoft OneDrive.
Will my data be anonymised?	*Yes.* All subjects will be assigned an identification number. This number will be used when discussing the information collected during the study and in any future publication of results. Following the collection of the blood and faecal samples, an alpha numeric code will be assigned to each sample.
How will my data be used?	This study involves collection of blood and faecal samples. -Data collected will be analysed to assess the intake of palm oil/palmitic acid and its effect on metabolic health.
Who will have access to my data?	Data collected during this research is purely for the scientific research and generation of information for public health purpose. The data will only be accessible by the lead researcher and supervisory team. No third-parties will have access to any data.
Will my data be archived for use in other research projects in the future?	Data collected in this study will provide the basis for further study of this PhD research. It is expected that following completion of the PhD project and publication of research papers, all data will be destroyed permanently.
How will my data be destroyed?	All personal contact details will be deleted on day 29 (following results).

9. Are there any risks in taking part?

Participants may encounter minor discomfort during/after blood sampling which could include bruising. There is also a small risk of infection when blood is taken. Participants may find providing faecal samples uncomfortable. However, procedure information will be provided beforehand and care will be taken to minimise any of such discomfort. Any allergies, medication, illnesses should be discussed prior to accepting participation. Participants will be advised to visit their GP, in the event of any severe pain and/pr discomfort.

10. Are there any benefits from taking part?

This study proposes that the prevalence of palm oil/palmitic acid in the typical western diet may be harmful to human metabolic and gut microbiome health. This study will highlight the differences in the health effects of different levels of palm oil and palmitic acid consumption.

11. Expenses and / or payments

No expense or payment will be made for participating in this research.
All participants will be provided a gift card (£10/each) for completing this study.

12. What will happen to the results of the study?

The results of the study will be analysed in unison and develop other studies within this PhD project and is likely to provide scientific evidence on the health implication of consuming foods with palm oil/palmitic acid.

13. What will happen if I want to stop taking part?

If you change your mind, you can always withdraw at any time, however the time in which you do so has different effects on the data process.

Signing of the consent form is considered as the basis for the participants willingness to participate in this study. However, if they wish, they may withdraw at various stages as follows-
After consent is given but prior to the start of data collection - No data will be collected from such participants.
During the data collection process - No data will be collected from such participants and any data already collected will be excluded in the study.
Participants have already supplied their samples - Following the anonymization process when the tracing of the individual is not possible, the exclusion of the data will not be possible.

14. What if I am unhappy or if there is a problem?

If you are unhappy, or if there is a problem, please feel free to let us know by contacting ***Thomas Hothersall via thothersall1@uclan.ac.uk*** *and we will try to help. If you remain unhappy, or have a complaint which you feel you cannot come to us with, then please contact the Ethics, Integrity and Governance Unit at* OfficerForEthics@uclan.ac.uk.

The University strives to maintain the highest standards of rigour in the processing of your data. However, if you have any concerns about the way in which the University processes your personal data, it is important that you are aware of your right to lodge a complaint with the Information Commissioner's Office by calling 0303 123 1113.

Contact details of investigatory team

Researcher
Thomas Hothersall
Tel: 07500338638
Email: THothersall1@uclan.ac.uk

Director of Studies
Dr Bojlul Bahar
Email: bbahar@uclan.ac.uk

Supervisor(s)
Dr Stephanie Dillon
Email: sdillon@uclan.ac.uk

Dr Jonathan Sinclair
Email: JKSinclair@uclan.ac.uk

Appendix 5.2. Participant consent form for the biomarkers assessment.

University of Central Lancashire
UCLan

Participant Consent Form

Version number & date: Version 1.1, 5th June 2023
Research ethics approval number: HEALTH 01004
Title of the research project: Palmitic acid (16:0) consumption in plant-based diets and its effect on metabolic health.
Name of researcher(s): Thomas Hothersall

Please initial box

8. I confirm that I have read and have understood the information sheet (**PIS Thomas Hothersall v2 (20/01/2023**) for the above study, or it has been read to me. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily. ☐
9. I understand that taking part in the study involves the taking one blood sample (10 ml), collection of a faecal sample (1-2 gm) on one occasion only. I am aware that my RNA (Messenger RNA) will be anonymized and analysed for the purpose of gene expression analysis that would enable better understanding of the biochemical signalling processes associated with metabolic health. ☐
10. I understand that my participation is voluntary and that I am free to stop taking part and can withdraw from the study at any time without giving any reason and without my rights being affected. In addition, I understand that I am free to decline to answer any particular question or questions. ☐
11. I understand that I can request for deleting all information that I have supplied at any time prior to the visit at the Darwin Suite (UCLan). I understand that following completion of the visit and sampling, I will no longer be able to request access to or withdrawal of the information I provide. ☐
12. I understand that the information I provide will be held securely and in line with data protection requirements at the University of Central Lancashire.
13. I understand that signed consent forms will be retained in Microsoft OneDrive. Contact details will be held for 28 days following return of documents to the researcher, contact details will be deleted day 29. ☐

14. I agree to take part in the above study. ☐

Participant name	Date	Signature
Name of person taking consent	Date	Signature

Principal Investigator
Bojlul Bahar
School of Sport & Health Sciences
01772894914
bbahar@uclan.ac.uk

Student Investigator
Thomas Hothersall
07500338638
thothersall1@uclan.ac.uk

Open data and data sharing

I understand that the information I provide will be held securely and in line with data protection requirements at the University of Central Lancashire until it is fully anonymised and then deposited in Microsoft OneDrive for sharing and use by other authorised researchers to support other research in the future. ☐

I understand that other authorised researchers may use my words in publications, reports, webpages, and other research outputs, if their study has been approved by a research ethics committee, and they agree to preserve the confidentiality of the information as requested in this form. ☐

I understand that personal information collected about me that can identify me, such as my name or where I live, will not be shared beyond the study team. ☐

Risk to participants

I understand that taking part in the study may induce discomfort during/after blood sampling which could include bruising. Any allergies I have will be discussed prior to participation. ☐

Confidentiality of the data

I understand that the research team will respect my confidentiality and I give permission for them to have access to my responses. ☐

I understand that confidentiality and anonymity will be maintained and it will not be possible to identify me in any reports, presentations or publications arising from the research will be numbered and anonymised. ☐

Disclosure of criminal activity and other confidentiality issues

I understand that the confidentiality of the information I provide will be safeguarded and won't be released without my consent unless I disclose information which raises concerns about the safety of myself or others. In such cases the researcher may be required to disclose my confidential information to the relevant authorities. ☐

Health-related findings in clinical research

I agree for my GP to be contacted if any unexpected results are found in relation to my health. ☐

Appendix 5.3. Safety/COSHH form for the biomarkers assessment.

UCLan SHE Detailed Work Instruction

School/Service	**Assessors Name(s)**	**Job Title/Position**	☎
School of Sport and Health Sciences **University of Central Lancashire**	Thomas Hothersall	PhD Research Student	07500338638

Collection of blood and faecal samples from human subjects. Involves collection, handling and processing (centrifugation, quantitative real-time PCR & freezing). None of the participants are diagnosed as having any infectious disease.

Substances (used or produced as by-products or wastes)	**Quantity**	**Hazard Class**	**Workplace exposure limit** *(See HSE EH40/2005. If no limit, leave this column blank)*	**Exposure Route(s)**	**Frequency and Duration of Exposure**	**Known Health Effects:**
RNA Later	500ml	Non-identified (from MSDS)	N/A	N/A (from MSDS)	4 hours, once per week	N/A (from MSDS)
Insulin Quantikine ELISA Kit		Irritant	N/A	Splashed on skin, Eyes	4 hours, once per week	Causes serious skin irritation and serious eye irritation (from MSDS)
Sigma-Aldrich GENELUTE STOOL DNA ISOLATION KIT		Non-identified	N/A	N/A (from MSDS)	4 hours, once per week	N/A (from MSDS)
STREPTAVIDIN BEAD BUFFER		Toxic Health hazard Dangerous for the environment (from MSDS)	N/A	Ingestion, skin contact, inhalation	4 hours, once per week	Toxic if swallowed Toxic in contact with skin Causes skin irritation Causes damage to organs

						Toxic to aquatic life with long lasting effects (from MSDS)
10mM dNTP Mix		Non-identified (from MSDS)	N/A	N/A (from MSDS)	4 hours, once per week	N/A (from MSDS)
2X HYBRIDIZATION BUFFER		Toxic Health Hazard Dangerous for the environment (from MSDS)	N/A	Ingestion, skin contact, inhalation	4 hours, once per week	Toxic if swallowed Toxic in contact with skin Causes skin irritation Causes damage to organs Toxic to aquatic life with long lasting effects (from MSDS)
Water, nuclease-free		Non-identified (from MSDS)	N/A	N/A (from MSDS)	4 hours, once per week	N/A (from MSDS)
Oligo(dT)18 Primer		Non-identified (from MSDS)	N/A	N/A (from MSDS)	4 hours, once per week	N/A (from MSDS)
RevertAid H Minus M-MuLV Reverse Transcriptase		Non-identified (from MSDS)	10 mg/m^3 TWA (mist)	N/A (from MSDS)	4 hours, once per week	N/A (from MSDS)
RNA BINDING BEADS		Non-identified (from MSDS)	0.1 mg/m^3	N/A (from MSDS)	4 hours, once per week	N/A (from MSDS)
RNA BINDING BUFFER		Irritant (from MSDS)	N/A	N/A (from MSDS)	4 hours, once per week	Acute oral toxicity Acute dermal toxicity Acute Inhalation toxicity- Dusts and Mists Skin corrosion/irritation (from MSDS)
RNA WASH SOLUTION CONC.		Non-identified (from MSDS)	N/A	Ingestion, skin contact, inhalation, eye contact	4 hours, once per week	May cause eye irritation with susceptible persons

						May cause skin irritation in susceptible persons. May be harmful by inhalation. May be harmful if swallowed. (from MSDS)
RiboLock RNase Inhibitor		Non-identified (from MSDS)	N/A	Eyes, Skin, Inhalation, Ingestion	4 hours, once per week	N/A (from MSDS)
Streptavidin Magnetic Beads		Non-identified (from MSDS)	10 mg/m^3 TWA (mist)	N/A (from MSDS)	4 hours, once per week	May cause eye irritation with susceptible persons May cause skin irritation in susceptible persons. May be harmful by inhalation. May be harmful if swallowed. (from MSDS)
FG,SYBR GREEN PCR MASTER MIX		Non-identified (from MSDS)	N/A	Eyes, Skin, Inhalation, Ingestion	4 hours, once per week	N/A (from MSDS)
Human Blood Sample		Potentially Biohazard		Skin contact, Inhalation	During processing of the sample (once per week)	Potential infection with biological agents
Human Stool Sample		Potentially Biohazard		Skin contact, Inhalation	During processing of the sample (once per week)	Potential infection with biological agents
Results of Relevant Health Surveillance				**Results of Exposure Monitoring**		
None				None		

Control Measures				
☐ Elimination	☐ Substitution	☐ Reduction	☐ Isolation	☐ Eng. Control
Details N/A	*Details* N/A	*Details* Only those fully trained and wearing personal	*Details (E.g. glovebox)*	*Details (E.g. LEV, fume hood)* Chemicals that generate aerosols will be handled in an

		protective equipment are granted access to the laboratory.		operator protection safety cabinet.

Further Details (if required)

- Good personal hygiene practice, hand wash facilities available
- All researchers fully aware of health and safety policies.
- Researcher competent in handling of chemicals.
- Researcher to wear full PPE when handling all chemicals.
- Nitrile gloves to be worn always when handling material. After use dispose as biohazard incinerable waste.
- All surfaces to be regularly swabbed down with Virkon solution 1% at a rate of 300ml/m^2 from a spray container.
- See code of practice for safe use of centrifugation machine.
- Operator competent in use of centrifugation machine.
- See code of practice for safe us of quantitative real-time PCR machine.
- Operator competent in use of quantitative real-time PCR machine.
- All samples will be stored appropriately using suitable containers inside fridges and freezers and clearly labelled in compliance with the Human Tissue Act 2004.
- Hepatitis B vaccine administered prior to any contact with blood samples.

Personal Protective Equipment

☒ Gloves	☒ Eye protection	☒ Coverall/lab coat	☐ Foot protection	☐ Respiratory protection
Details	*Details*	*Details*	*Details*	*Details:*
Nitrile single use gloves.	Safety glasses.	Howie style lab coat to be worn always.	Appropriate footwear. Flat shoes	N/A

☒ Health Surveillance required	☒ Exposure monitoring required

Emergency Arrangements	
First Aid: Consult a physician. Show this safety data sheet to the Doctor in attendance.	
Eyes	Wash thoroughly with plenty of water for at least 15minutes and if necessary consult a physician.
Skin	Wash off with soap and plenty of water. Remove any contaminated clothing. If required take person immediately to hospital and consult a physician.
Ingestion	Do not induce vomiting. Never give anything by mouth to an unconscious person. Rinse mouth with water. Consult a physician.
Inhalation	If breathed in, move person into fresh air. If not breathing, give artificial respiration. Consult a physician.

Fire: Extinguisher Type			
☐ Water	☐ Foam	☐ Powder	☐ CO_2

Spillage/release:

- Treat all spills as hazardous waste. Clean up using the spills kit stored in DB034.
- Blood or faecal sample spills should be wiped up immediately using virkon solution 1% at a rate of 300ml/m^2 . Dispose as biohazard incinerable waste.

Waste Disposal Procedure

All samples to disposed of following the clinical waste procedure – autoclaving and clinical waste disposal route.

All solutions and nitrile gloves used when handling material are to be disposed of as biohazard incinerable waste.

Persons likely to be exposed			
☒ Staff	☒ Student	☐ Visitor	☐ Contractor
☐ Public	☐ Other (specify)		

Additional risks: for example, circumstances where work will involve exposure to more than one substance hazardous to health, consider the risk presented by exposure to such substances in combination. Also, non-routine maintenance may present additional risk of exposure.

- Non-routine events, such as power cut or machine breaking will not affect the safety of any person. This will only compromise the samples.

Signed by Head of School/ Service or nominee:		Review date due:	Click or tap to enter a date.
Date:	Click or tap to enter a date.		

Notes:

Hierarchy of control

Change the task or process so that the hazardous substance is not required or generated.

Replace the substances with a safer alternative.
Totally isolate or enclose the process.
Partially enclose the process and use local exhaust ventilation.
Ensure good general ventilation.
Use a system of work that minimises the chance and degree of exposure.
Provide personal protective equipment (PPE).
Train and inform staff in the safe system of work and risks.
Additional supervision.
Examination, testing and maintenance of engineering controls and/or PPE.
Monitoring of exposure.
Health Surveillance.
Other (specify).

Appendix 5.4. Risk assessment form for the biomarkers assessment.

Thomas Hothersall - Risk Assessment Form	University of Central Lancashire UCLan

Risk Assessment For	Assessment Undertaken By	Assessment Reviewed By
Service / School / Faculty: School of Sport and Health Sciences	**Name: Thomas Hothersall**	**Name: Gareth Shadwell**
Location of Activity: UCLan Nutrition Suite	**Date: 18/02/2023**	**Date: 17/08/2023**
Activity: Project: Palmitic acid (16:0) consumption in plant-based diets and its effect on metabolic health. Blood sampling. Handling of pre-provided sealed faecal samples.	**Signed by Dean of Faculty / Head of School / Director of Service or their nominee:**	

New Risk Assessment: ☒	Amended Risk Assessment: ☐	If amended, please state why?

Significant change to workplace, processes or design ☐	Training ☐	Consultation ☐	Audit ☐	New machinery, substances or procedures introduced ☐	Following, accident, injury or near-miss ☐	Regulatory requirement ☒	Best practice ☐	Other please state:

What are the actual or potential hazards? (List in order of importance)	**List groups of people who might be harmed by the hazards and state how they may be harmed?**	**What are the existing controls in place?** (Operational, procedural, policy, instructions, training, competency, PPE, consultation, etc.)	**Do you need to do anything else to control this risk?**	**Action by who?**	**Action by when?**	**Status? Complete / outstanding**	**Remaining level of risk (low, medium or high)**
Setup of laboratory equipment	Participant, investigator, students and staff	All unnecessary equipment, chairs, stools, clothing, bags are cleared and put away. The surrounding equipment must be kept to a minimum, must be checked in advance to greatly minimise chance of malfunction(s), and must be checked for electrical safety. There must be sufficient space for the participant to sit or lie down at any time during or after the procedure.	N/A	Investigator	Before/during use	N/A	Low
Poorly maintained facilities (slips, trips and falls)	Participant, investigator, students and staff	Area should be well maintained and checked prior to the arrival of the participant. The entire laboratory floor will be kept clear as much as possible, not just the area surrounding the test. This includes items such as bags and water bottles being kept out of the way of the test, as well as other potential staff, participant and student footfall. Chairs, stools and other equipment will be pushed in/away and in as safe as a place as possible. Wires will be out of the way or taped down to avoid trip hazards. It is also important to ensure that the floor is kept dry, if the floor has been dried recently, a wet floor sign must be used.	N/A	Investigator	Before/during use	N/A	Low
Transportation of blood & faecal samples	Staff	Samples and cooler will be labelled appropriately for both biological specimen and biohazard(s). All samples will be triple packaged; a primary receptacle e.g., blood tube. Secondary leak-proof packaging (sealable plastic bag) and an outer package (cool box). To ensure no contact between primary receptacles, each will be stored individually in a sealable plastic bag. Secondary packaging will be secured in the cool box using bubble wrap. The cooler containing the samples will then be secured and stored in the back seat on the floor.	N/A	Investigator	Before/during use	N/A	Low

What are the actual or potential hazards? (List in order of importance)	**List groups of people who might be harmed by the hazards and state how they may be harmed?**	**What are the existing controls in place?** (Operational, procedural, policy, instructions, training, competency, PPE, consultation, etc.)	**Do you need to do anything else to control this risk?**	**Action by who?**	**Action by when?**	**Status? Complete / outstanding**	**Remaining level of risk (low, medium or high)**
Health & safety / hygiene during blood collection	Staff, participants	Hygiene practice will be adhered to in a high-standard - always (washing of hands, PPE, wearing a lab coat and safety goggles). Researchers will be fully aware of all relevant health and safety policies. Nitrile gloves to be worn always when handling material. After use, gloves will be disposed of as biohazard incinerable waste. All collection apparatus will be sterile. Alcohol swabs will be used to clean skin surface prior to blood retrieval. Hygiene instructions will be visible on the lab wall (as shown in the PIS) • Safe blood sampling equipment. • Safe disposal of sharps container. • Immediate disposal of any leftover sample and spillage following standard procedure. • Skin antisepsis available. Sterile, single-use blood-sampling devices.	N/A	Investigator	Before/during use	N/A	Low
Unscreened blood samples	Staff, participants	The samples/waste will be handled appropriately as they are unscreened. Contaminated waste/sharps will be disposed of appropriately (incinerated). PPE will be used at all times. Alcohol swabs and disinfectant will be used to clean areas.	N/A	Investigator	Before/during use	N/A	Low
Blood sample collection	Staff	Minimal bruising or swelling is possible at site of injection. Participant(s) will be briefed fully as to potential effects and side-effects of having a blood test, as well as recommendations/strategies to prevent bruising or swelling. In the event of any excess pain or discomfort being experienced - testing may be terminated. Participant(s) will be advised to visit their GP.	N/A	Investigator	Before/during use	N/A	Low

What are the actual or potential hazards? (List in order of importance)	**List groups of people who might be harmed by the hazards and state how they may be harmed?**	**What are the existing controls in place?** (Operational, procedural, policy, instructions, training, competency, PPE, consultation, etc.)	**Do you need to do anything else to control this risk?**	**Action by who?**	**Action by when?**	**Status? Complete / outstanding**	**Remaining level of risk (low, medium or high)**
Blood & faecal assessment protocols	Staff, participants	Sample analysis will be conducted following code of practice for safe use of centrifugation machine. Operator competent in use of centrifugation machine. Sample analysis will be conducted following code of practice for safe us of quantitative real-time PCR machine. Operator competent in use of quantitative real-time PCR machine.	N/A	Investigator	Before/during use	N/A	Low
Risk of infection (when blood is taken and stool sample is processed)	Staff, Participants	Blood sample area (arm) will be cleansed thoroughly before blood is drawn to remove bacteria/risk of infection. The staff and participants must wear PPE. Protocol/instructions will be explained should an infection occur: Wash immediately with clean water for several minutes, then leave the skin to dry. Apply antiseptic. Protect with a plaster/suitable dressing.	N/A	Technician	Before	N/A	Low
First aid & emergency procedures	Chemical Hazards	Fully qualified first aid staff will be present at all times - **Thomas Hothersall**. An emergency telephone is located in the laboratory. Qualified first aid staff will be on hand at all times. A defibrillator is also available in DB023.	N/A	Investigator	Before/during use	N/A	Low
Electrical hazards	Investigator, technician	All electrical equipment is annually PAT tested and are regularly inspected for faults, faulty equipment to be removed from use immediately. Extension leads not to be linked together and adaptor blocks not used.	N/A	Technician	Annually	N/A	Low
Chemical Hazards	Investigator, technician	See COSHH Form.	N/A	Investigator	Before/during use	N/A	Low
Termination of testing	Investigator	Participation is voluntary and participants can withdraw at any time for any reason. The investigator must end testing if the participant becomes unwilling or unable to continue.	N/A	Investigator	During session	N/A	Low

What are the actual or potential hazards? (List in order of importance)	**List groups of people who might be harmed by the hazards and state how they may be harmed?**	**What are the existing controls in place?** (Operational, procedural, policy, instructions, training, competency, PPE, consultation, etc.)	**Do you need to do anything else to control this risk?**	**Action by who?**	**Action by when?**	**Status? Complete / outstanding**	**Remaining level of risk (low, medium or high)**
Lone working	Investigator	There is to be absolutely no lone working, especially with venous blood sampling. There must be a qualified member of staff present in case of emergencies.	N/A	Investigator	N/A	N/A	Low
Fire	Investigator, Staff, Participants	Nearest fire extinguishers are outside DB017b, nearest fire exit is under the staircase next to the main entrance and the fire assembly point for Darwin Building is the car park between Leighton & JB Firth Buildings.	N/A	Investigator	During session	N/A	Low
Bioelectrical Impedance Analysis (Machine)	Investigator, Staff, Participants	One participant (plus investigator) in the room at any one time; to avoid both participant discomfort as well as lessen the risk of accident, trip or fall. Personal belongings and items of clothing and/or accessories (i.e., bags, coats) will be stored at a reasonable distance from the machine as to prevent falling, tripping or entanglement with the equipment. All surfaces of the machine will be disinfected before and after each use to avoid cross-contamination of any kind between participants (users of the machine).	N/A	Investigator	During session	N/A	Low

Appendix 5.5. Data protection checklist for the biomarkers assessment.

Legal and Governance: Data Protection Checklist
Version: FINAL v5.4 November 2021

University of Central Lancashire
UCLan

Data protection checklist: Teaching, research, knowledge transfer, consultancy and related activities

All activities which involve personal data of any kind, in any way, must comply with data protection legislation (the UK General Data Protection Regulation (UK GDPR) and Data Protection Act 2018 (DPA)). This checklist outlines the requirements of the data protection legislation and the measures you must take when processing personal data; it also provides a mechanism for recording the steps you will take to ensure the personal data you are using are safeguarded and the reputation of the University is upheld.

Ensuring personal data are processed transparently, fairly and lawfully with due regard for individuals' privacy and ensuring that personal data remain secure are paramount. Demonstrating that we have considered the requirements of data protection legislation when conducting our activities will provide assurances to students, employees, research participants and business partners that their personal data is protected at UCLan. Organisations can be fined up to £17.5 million or 4% of global annual turnover for breaches of the UK GDPR; therefore it is important that we get it right from the outset. If it is possible to use anonymised data so that individuals cannot be identified from it and still achieve your aims, this is always the preferred method of operating. Truly anonymised data (which cannot be reconstructed or linked to any other data you hold or may hold in the future to enable you to identify individuals from it) does not constitute personal data because it cannot be used to identify individuals.

Section 1: Does my activity involve personal data?

What is *personal data*?

Personal data is data relating to a living individual who can be identified from those data (or from those data and other information in our possession or likely to come into our possession).

Personal data can be factual (such as name, address, date of birth) or can be an opinion (such as a professional opinion as to the causes of an individual's behavioural problems or a response to a survey question). Voice recordings are personal data because the individual can be identified from his or her voice. Information can be personal data even if it does not include a person's name or

other obvious identifiers. ***Example***: *a paragraph describing a specific event involving an individual or a set of characteristics relating to a particular individual may not include their name but would clearly identify them from the set of circumstances or characteristics being described or represented.*

If you are unsure whether or not your activity involves personal data, please contact the Information Governance team to discuss on DPFOIA@uclan.ac.uk.

More information about personal data is available in the Data Protection Policy.

What is *processing*?

The UK GDPR is concerned with the processing of personal data. 'Processing' means any operation or set of operations which is performed on personal data or on sets of personal data, whether or not by automated means, such as collection, recording, organisation, structuring, storage, adaptation or alteration, retrieval, consultation, use, disclosure by transmission, dissemination or otherwise making available, alignment or combination, restriction, erasure or destruction.

Does your project involve personal data?	Yes	*Please provide details:* *Some partial identifying information will be collected regarding individuals' gender, age, ethnicity, lifestyle and anthropometry. This information will be anonymised, and no name and content of the participants will be recorded. The data collected on the dietary habits will have the personal identifiers. This data will be anonymised at a later stage.*
If yes, are you processing that data? If you have any kind of access to that data, including just holding it, you are likely to be processing it.	Yes	*Please provide details:* *Data will be stored on a password-encrypted device. All potential identifying personal information be numbered and randomise and handled as anonymous.*

If your proposed activity involves processing personal data, you must complete the remainder of this checklist, providing answers to each relevant question.

Section 2: Have I addressed all the key areas of data protection compliance?

This checklist will ensure that you have properly considered how you are processing the personal data you are collecting. You should be able to answer

'yes' to all questions or explain why the question is not applicable. You must provide details to support your answers so that your proposal can be approved. If you are a researcher completing this form as part of your ethics application, you can complete parts of this checklist by referencing your answers on the relevant parts of your Ethics Application Form, if they cover the same detail.

Type of activity:	**Blood, Faecal Sample/Biomarkers Study**
Activity name/title:	**Palmitic acid (16:0) consumption in plant-based diets and its effect on metabolic health.**

Processing personal data transparently, fairly and lawfully
Data protection legislation requires us to process personal data transparently, fairly and lawfully. In practice, it means that you must: • have lawful grounds for collecting and using the personal data; • not use the data in ways that have unjustified adverse effects on the individuals concerned; • be transparent about how you intend to use the data, and give individuals appropriate *privacy notices* when collecting their personal data; • handle people's personal data only in ways they would reasonably expect; and make sure you do not do anything unlawful with the data.

28. Have you checked and confirmed that the intended uses of personal data (including special category data or criminal convictions data) have a lawful basis?	Yes	
29. What is the lawful basis for processing the personal data? *In almost all cases at UCLan, the lawful basis for processing personal data for research purposes will be that the processing is necessary for a task carried out in the public interest (Article 6(1)(e) UK GDPR). If you think there is an exceptional reason why this does not apply*		*A task carried out in the public interest*

to your research, there are alternative lawful bases from the UK GDPR which can be considered. You must contact the Information Governance team for advice, particularly if you are considering relying on consent as a lawful basis. Further information about lawful bases can be found here.		
30. Are you processing special category data or criminal convictions data?	No	*N/A*
31. If the answer to question 3 is 'yes', what is the lawful basis for processing this type of personal data? *If you are processing this type of data for research purposes and the processing is in the public interest, the lawful basis is likely to be Article 9(2)(j) UK GDPR by virtue of section 10 and Schedule 1(4) DPA.*		*N/A*
32. If the intended use of the personal data would or would be likely to have an adverse effect on one or more individuals, have you considered and documented why that adverse effect is justified?	Not applicable	*N/A*
33. Have you documented why you are collecting the specific items of information to demonstrate they are necessary for your stated purposes *e.g. if you are carrying out research into how students' music preferences affect their degree classification and also collecting participants' shoe sizes, can you show you have a legitimate need for this information, when they are not obviously linked?*	Yes	*Relevant information is included in the Ethics application.*

34. Have you written an appropriate privacy notice to provide to individuals at the point you collect their personal data? *A privacy notice is a legal requirement and tells individuals how you will process their data if they give it to you, among other things. Clear and detailed guidance about privacy notices and what they must contain is available here. If you are carrying out research, you should refer participants to the University's standard privacy notice for research participants, which must be supplemented by your participant information sheet.*	Yes	*Yes, all related and relevant information is provided on both the Participant Information Sheet(s) and Participant Consent Form(s).*

Consent
Consent to participate in a research study is not the same as relying on consent as a lawful basis on which to process personal data. Researchers are strongly discouraged from relying on consent as a lawful basis for processing personal data for research purposes. If you are relying on consent as the lawful basis for processing personal data, the UK GDPR requires that a certain standard of consent is obtained. Consent as a lawful basis to process personal data is only valid if it is freely given, fully informed, unambiguous and **capable of being withdrawn at any time**. If consent is withdrawn, you must cease all processing of personal data which is being carried out on the basis of that consent. For research purposes, this means that if a participant withdraws consent to process their personal data, all processing must cease and all personal data already collected about that participant must be deleted or destroyed. This may have significant implications for your research. The UK GDPR is clear that an indication of consent must be unambiguous and involve a clear affirmative action (an opt-in). A failure to respond to a notification that an activity will take place unless an individual opts out is not valid consent. The UK GDPR also requires granular consent options for distinct processing operations e.g. participants may consent to you processing their survey responses but they may choose not to consent to you taking photographs of them during focus groups. Consent should be separate from other terms and conditions. You must keep clear records to demonstrate consent has been obtained. You need to tell people about their right to withdraw and offer them easy ways to withdraw consent at any time.

Consent should only be used as a lawful basis for processing personal data if valid consent can be obtained; the consent can be withdrawn at any time; and withdrawal of consent will not impact your research. Note that where group recordings are made e.g. focus groups, it will be very difficult to remove one participant's personal data (their voice and opinions) from the recording if that participant withdraws consent to process their personal data. This is one example of why consent as a lawful basis for processing personal data will not be appropriate for many types of research.

You may still obtain participants' consent to participate in various aspects of your research, even if you do not rely on consent as a lawful basis for processing the personal data you subsequently collect. Participants may withdraw their consent to participate in any future aspects of the research but you may continue to process any personal data already collected **if** you have relied on a lawful basis for processing other than consent.

Further detailed information about obtaining valid consent under the UK GDPR is available here.

35. Have you checked and confirmed that consent is necessary and is the most appropriate basis for your processing? *Note that this question relates to consent as a lawful basis for processing personal data and **not** consent to participate in your research.*	N/A	
36. If you are processing special category data or criminal convictions data on the basis of consent, have you planned to obtain individuals' explicit consent? *As the standard for consent is so high under the UK GDPR, there is not likely to be a significant difference between consent and explicit consent.*	N/A	
37. If you are relying on individuals' consent as the lawful basis for using their personal data, have you developed a process for managing the withdrawal of consent at any point during or after the research?	N/A	

38. If you are relying on consent as a lawful basis to process personal data, have you ensured that the individual understands their rights and is capable of giving consent? This should be assessed on a case-by-case basis. *If you are processing personal data about children or those with reduced capacity, consider whether or not you need to obtain consent from parents, guardians or legal representatives. You must consider how to provide clear consent statements to those whose first language is not English.*	N/A	

Security
The UK GDPR requires you to process personal data securely. This means that you must have appropriate security measures in place to protect the personal data you hold from being lost, damaged, destroyed or disclosed inappropriately. Security measures include technical security measures to keep electronic information secure; physical security measures to keep hard-copy information secure; and processes and procedures around the management of, and access to, the information. Organisations can be fined up to £17.5 million for breaches of security involving personal data. The UK GDPR requires us to implement appropriate technical and organisational measures to ensure a level of security appropriate to the risk. It is important that any personal data you collect or use during your activities remains secure until it is securely destroyed, which includes ensuring that only those who are authorised to access and use the data can do so. **For further guidance on information security, please see the Information Governance pages of the UCLan intranet and the LIS IT Security Policy. You may also find the information on the ICO website useful.**

39. If you are intending to publish information which could identify individuals, have you made those individuals aware that this will happen via your privacy notice and obtained their consent, if appropriate?	Not applicable	N/A

40. Will papers, files, audio visual recordings, CDs, USB (memory) sticks, microfiche or other media which contain personal data be kept in locked cabinets, cupboards, drawers etc. when the offices are vacated or if they are stored off UCLan premises?	Yes	N/A
41. Do all individuals who will have access to or be using the personal data understand that it must not be provided to any unauthorised person (which includes disclosing information to family members or other representatives of data subjects, unless the data subject has given consent for us to do this)?	Yes	*All members of the supervisory team and researcher are aware of this.*
42. Do all individuals who will have access to or be using the personal data understand their responsibilities under data protection legislation and have they received data protection training? *A UK GDPR e-learning course is available to staff and students via Blackboard.*	Yes	*All members of the supervisory team have undertaken GDPR training as appropriate.*
43. Do you have appropriate procedures in place to ensure the security of the personal data if it is removed from or collected and stored away from UCLan offices for any reason? *You must provide details of how you will ensure personal data remains secure if you remove it from UCLan premises or collect and store it away from UCLan premises. Please note that electronic data must only be removed if it is stored on encrypted devices or media e.g.*	Yes	*All digital data collected will be saved and stored on a password protected computer in Preston Campus of the University of Central Lancashire that has encryption and two-factor authentication facilities. The digital data will only be accessible by the lead researcher and supervisory team. All personal contact details will be retained for 28 days following results. All data collected via email will be saved and stored, as well as password protected with two-factor authentication, via Microsoft OneDrive.*

an encrypted disc or USB stick, an encrypted laptop or Dictaphone etc. Alternatively it can be accessed remotely via a secure connection. If an unencrypted device containing personal data is lost or stolen, it is likely to lead to a substantial fine for a breach of data protection legislation. Non-electronic records must be rigorously safeguarded at all times and not left unattended or in view of unauthorised people. Laptops, USB sticks and other devices, papers or any other form of personal data must not be left in cars.		
44. Will the personal data be stored on the UCLan network, or in a Microsoft 365 account provided by UCLan, in a secure location with restricted access, to prevent unauthorised parties who have no right or need accessing the data?	Yes	Stated in the Ethics form
45. Are all individuals who will have access to or use the personal data aware that personal information should only be stored on the UCLan network or in cloud storage provided by UCLan and should only be stored on equipment owned or leased by UCLan, unless exceptional circumstances apply, after taking advice from LIS? *Storage under such exceptional circumstances must include the use of appropriate security measures. No personal information should be stored on any removable media e.g. USB sticks, CDs or devices e.g. laptops, smartphones, Dictaphones unless they are encrypted. BitLocker is the*	Yes	*All members of the supervisory team understand fully.*

recommended tool for encrypting external devices; guidance can be obtained from LIS Customer Support. UCLan makes available Microsoft 365 cloud storage for staff and students, including OneDrive for Business. This is the only approved cloud storage facility.		
46. Are all individuals who will have access to or use the personal data aware that any information accessed via remote working methods such as Outlook Web App, UCLan Global or similar must be treated securely in line with relevant legislation and all University guidelines? *UCLan business information, including personal data, should not be stored on personal, non-UCLan equipment or devices unless exceptional circumstances apply, after taking guidance from LIS, even where personal equipment and devices are used to access information remotely.*	Yes	*All members of the supervisory team understand fully.*
47. Are all individuals who will have access to or use the personal data aware that email is not a secure method of communication and can easily be sent to the wrong recipient and do they know how to encrypt documents so that they can be attached to an email and sent securely? *N.B. Encryption passwords must be provided separately and never included in the same email as the encrypted attachment. Guidance is available here. Users should consider whether or not email is the most appropriate method. Consider using*	Yes	*All members of the supervisory team understand fully.*

UCLan's OneDrive for Business instead.		
48. Are all individuals who will have access to or use the personal data aware that all non-electronic material which contains personal data and has been authorised for disposal must be disposed of via the University's confidential waste service (including handwritten notes, computer print-outs etc.)?	Yes	*All members of the supervisory team understand fully.*
49. Are all individuals who will have access to or use the personal data aware that any paper documents, electronic media or hardware which has been designated for disposal must be kept in a secure location until it has been appropriately destroyed and any information it contains is no longer accessible or recoverable? *Electronic media and hardware must be disposed of in line with LIS guidelines and procedures.*	Yes	*All members of the supervisory team understand fully.*
50. Data protection legislation prohibits the transfer of personal data to countries or territories outside the European Economic Area (EEA) unless certain conditions can be met. Can you confirm that if you intend to transfer personal data outside the EEA, you have taken advice from the Information Governance team to ensure you can do so in compliance with data protection legislation? *Transfers includes via email, websites and by virtue of using*	N/A	N/A

'cloud' providers which store your data on their servers based overseas, as well as physically holding the information overseas. For detailed guidance see Sending personal data outside the UK.		

Third parties acting on behalf of UCLan
Under some circumstances, it will be necessary or desirable to work with organisations external to UCLan, such as charities, research organisations, private companies, other public sector organisations, contractors, service providers or any other types of third parties. If a third party is acting on our behalf e.g. providing a service for us or on our behalf and that activity involves the third party accessing, collecting or otherwise processing personal data, they are a *data processor* under the UK GDPR. Guidance on the use of data processors, including a checklist to ensure their use is appropriate, is available here.

51. Are you using a data processor?	No	*If no, then you do not need to complete the questions regarding data processors below.*
52. If you are using a data processor or you need help deciding if the proposed arrangement does involve a data processor, have you read the guidance and, if necessary, taken advice from the Information Governance team to ensure all data protection requirements around the use of data processors have been satisfied?	N/A	

53. If you are using a data processor, have you taken advice on information security from the Information Governance team and the Information Security team in LIS? *It is a legal requirement to ensure a data processor is capable of putting in place appropriate technical and organisational security measures to protect personal data. If your data processor is based outside the EEA, additional measures must be in place.*	N/A	
54. If you are using a data processor, have you taken advice from the contracts team in Legal and Governance or from Procurement (as appropriate) to ensure you have appropriate contractual arrangements in place to cover the use of a data processor? *It is a legal requirement to have UK GDPR-compliant contracts in place with data processors.*	N/A	

Once this form has been completed, it should be attached to your Ethics Application Form (if applicable) and submitted as directed. If your activity does not require further or any ethical approval, this form should be retained with your project documentation as a record of your considerations and data protection compliance. If you require any further advice or guidance to help you complete this checklist, please contact the Information Governance team: DPFOIA@uclan.ac.uk. Members of staff can also find a variety of guidance documents and FAQs on the Information Governance intranet pages.

Appendix 5.6. Recruitment poster for the biomarkers assessment.