THE UNIVERSITY OF HULL

The effect of acute virgin coconut oil supplementation and aerobic exercise on triglyceride levels following postprandial hypertriglyceridemia in healthy males.

being a Thesis submitted for the Degree of MSc Sport, Health and Exercise Science in the University of Hull

by

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ii. Abstract

BACKGROUND: A substantial body of literature postulates that high levels of blood triglycerides (TG) cause oxidative stress which impairs vascular function and may instigate a pathway to cardiovascular disease (CVD) (Padilla *et al.,* 2006). Exercise has been shown to promote the removal of TG and reduce postprandial hypertriglyceridemia (PHTG) following a high-fat meal (McClean *et al.*, 2007). Research (Bueno *et al.,* 2015) also suggests that virgin coconut oil (VCO) is a rich source of medium-chain triglycerides (MCT) which may provide a mechanism against CVD.

AIM: Investigate the relationship between VCO and exercise following PHTG on TG levels.

METHODS: Nine healthy subjects (age 26.0 years ± 4.66) participated in a randomised crossover design: (1) High-fat meal alone (2) high-fat meal followed 1 hour after with moderate (60% max watt) exercise (3) high-fat meal with VCO followed 1 hour after with exercise. TG was measured in venous blood at baseline, 2 and 4 hours postprandial. HDL, cholesterol and other biochemical markers were measured.

RESULTS: Compared with control, TG levels significantly decreased with exercise 4 hours postprandial (P =0.044), and exercise with VCO group decreased at 2 hours (P =0.010) and 4 hours (P =0.012) postprandial, respectively. However, exercise alone did not significantly reduce TG levels 2 hours (P =0.274) postprandial. There was no significant difference in cholesterol and HDL levels between groups 2 hours and 4 hours postprandial, respectively.

CONCLUSION: Results show that a single session of moderate aerobic exercise can decrease TG levels following a high-fat meal. Furthermore, VCO further enhances the effects of moderate exercise in reducing TG levels following PHTG.

iii. Acknowledgements

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vii. Abbreviations

- Cardiovascular disease (CVD)
- Body mass index (BMI)
- Confidence interval (CI)
- Postprandial hypertriglyceridemia (PHTG)
- Triglycerides (TG)
- Medium-chain triglycerides (MCT)
- Virgin coconut oil (VCO)
- Saturated fat (SFA)
- Coronary heart disease (CHD)
- Low-density lipoprotein (LDL)
- Average American diet (AAD)
- High-density lipoprotein (HDL)
- Essential fatty acids (EFA)
- Free fatty acids (FFA)
- Long-chain triglycerides (LCT)
- Heart rate (HR)
- Rates of perceived exertion (RPE)

1.0. Introduction

1.1. The Context in Which the Research Took Place

Recent progress in nutritional research has found that irregularities in the postprandial state are significant factors in the development of atherosclerosis (Gill *et al.,* 2004). Hypertriglyceridemia is an abnormality in which elevated triglyceride (TG) levels are found in the blood and is now known to be a central risk factor in the development of cardiovascular disease (CVD) (Boren *et al.*, 2014). Postprandial hypertriglyceridemia (PHTG) usually occurs after a high-fat meal and has been shown to cause endothelial dysfunction, increase blood TG, increase oxidative stress, and that repeated bouts of PHTG can promote the development of atherosclerosis (Chan *et al.*, 2013). It has been postulated that PHTG-induced impairment in vascular function can be decreased by performing moderate intensity exercise 1 and 2 hour postprandial (McClean *et al.*, 2007). A reduction in circulating blood TG mediated by exercise, which imposes a positive change in oxidative stress markers is the likely cause of these vascular improvements (Padilla *et al.,* 2006).

Medium-chain triglycerides (MCTs) are stored less in the adipose tissue and are rapidly metabolised, making them a potential tool for weight control (Costa *et al.*, 2010). In addition, MCTs have been shown to increase energy expenditure (Bueno *et al.,* 2015). Thus, these TGs may provide health benefits in reducing CVD risk factors. However, additional research is required to provide conclusive evidence of the effectiveness of MCTs and future MCT research should focus on the health benefits, applications and effects on exercise performance (Clegg, 2010).

Virgin coconut oil (VCO) has been growing in popularity because of the potential cardiovascular benefits; VCO is a rich source of MCTs (59.02% to 62.27%) (Gopala Krishna *et al.,* 2010). However, research of VCO is currently lacking to conclusively recommend dietary alterations to improve CVD risk factors (Babu *et al.*, 2014). Literature of VCO and its cardiovascular benefits are at an early stage, limited to very few human trials. The effects of increased VCO consumption on CVD risk factors needs to be evaluated in randomised control trials. As well as these RCTs, the combination of VCO with exercise among various populations is critical to better establish ways to reduce CVD risk (Yeap *et al.*, 2015). There is

currently a lack of research investigating PHTG and subsequent blood lipid profiles, oxidative stress and the potential health benefits of exercise coupled with VCO.

1.2. Experimental Aims and Objectives

The primary aim of this investigation is to study the acute effects of moderate intensity exercise and VCO supplementation on postprandial TG levels following a high-fat meal in an attempt to reduce TG concentrations during the postprandial state. A natural food supplement will be utilised to examine its efficacy in attenuating TG levels following a high-fat meal. The effects of exercise and exercise coupled with VCO supplementation on TG levels will also be investigated as this might provide the most significant means in reducing TG levels following a high-fat meal. Additional important blood metabolites will be analysed throughout the investigation: cholesterol and high-density lipoprotein.

Aim: Investigate the effect of exercise and VCO supplementation on postprandial TG concentrations following a high-fat meal.

2.0. Literature Review

2.1. Dietary Fat, Carbohydrates and Human Health

2.1.1. Dietary Fat and Current Guidelines

Current government guidelines promote a high-carbohydrate (>50%) and low-fat diet (<35%) (Food Standards Agency, 2013). Furthermore, the Food Standards Agency nutrient and food based guidelines for UK institutions states that of the 35% of fat consumed no more than 11% should constitute saturated fat (SFA). Fat comprises of 9 calories per gram in comparison to carbohydrates and protein which contains 4 calories per gram respectively (Gordon & Hayes, 2012). This makes fat an abundant and rich source of energy and this promotes a potential to create a calorie surplus whilst eating high-fat foods; instead carbohydrates are often preferred and they are advertised as a low-calorie alternative, for example fat-free Greek yoghurt (Flores-Martin et al., 2013). Recent research suggests despite fat containing more than double the calories to carbohydrates, increased fat intake can lead to a reduced daily calorific intake (Lennerz *et al*., 2013). Lennerz *et al*., (2013) study found brain activity after a typical high-carbohydrate meal in the late postprandial period stimulated brain regions associated with increased hunger and cravings which caused subjects to consume more calories throughout the day. Further research by Ludwig *et al.* (2013) found a high-carbohydrate meal can make you feel hungrier hours later as opposed to consuming a low-carbohydrate meal. Both investigations attributed this to the high levels of blood sugar postprandialy following the high-carbohydrate meal. The increased blood sugar levels will come plummeting down leaving you with a sudden crash in energy, known as hypoglycemia (low blood sugar) (Lennerz *et al*., 2013). The research (Lennerz *et al*., 2013; Ludwig *et al.,* 2013) suggests that high-fat diets might play a role in reducing hunger cravings and in decreasing obesity by lowering daily calorie intake.

The Masai (a Nilotic ethnic group living in Africa) have very low rates of chronic heart disease (CHD), despite a diet high in fat and SFA (Mbalilaki *et al.*, 2010). In a cross-sectional investigation of 130 Masai subjects, 82% reported a high-fat low-carbohydrate diet. Compared with their urban Bantu and rural counterparts who consumed more carbohydrate and less fat, they had a lower BMI, systolic blood pressure and a more favourable blood lipid profile (Mbalilaki *et al.*, 2010).

Nevertheless, the observations might be due to genetic properties specific to the Masai so further research is needed in different populations. In medicine, the lipid profile is a panel of blood tests used to screen for abnormalities in lipids, for example TG and cholesterol. The evident improvement in lipid profiles in the Masai positively correlates to a reduced risk of CHD and CVD (Mbalilaki *et al.*, 2010). The European Prospective Investigation into Cancer (EPIC) Greek cohort study (Turati *et al.,* 2014) analysed data which included 20,275 healthy subjects. The study evaluated the association between a diet high in glycemic load and people following a more traditional Mediterranean diet. Turati *et al.* (2014) concluded that compared to the high dietary glycemic load diet, the Mediterranean diet could result in a 40% reduced risk for CHD, and over a 50% reduced fatality risk from CHD.

A systematic review by Mente *et al.* (2009) concluded that evidence supported the following: foods with a high-glycemic load or index have harmful implications on CHD risk factors and a protective link exists with monounsaturated fatty acids, nuts and vegetables with preventing CHD. This research (Mente *et al.,* 2009; Mbalilaki *et al.*, 2010; Turati *et al.,* 2014) provides strong evidence associating health benefits with SFA consumption and that current literature should alter the recommended government guidelines concerning SFA consumption.

2.1.2. Obesity and Disease

Obesity is defined as a person having too much body fat and is commonly measured using BMI (Xue *et al.,* 2011). Obesity is linked with complex characteristic metabolic disturbances and regularly occurs in Western World culture with typical lifestyle disorders which are the foundations for future metabolic syndrome development (Pastucha *et al.,* 2013). These changes are highlighted in several journals (Brown & Allison, 2013; Matheus *et al.*, 2013; Tuominen *et al.*, 2013) as being central to future CVD and metabolic disease. BMI, which is ominously correlated with increased risk of: CVD, hypertension, type-2 diabetes, dyslipidemia, and several cancers (Tuominen et al., 2013). BMI has repeatedly been associated with colon cancer; An investigation (Ning *et al.,* 2010) found the risk of colon cancer increases by 18% with each stepwise BMI increase of 5 kg/m^2. Of note, meta-analysis reported this association was stronger in men than women. This research gives more understanding of the

relationship increased BMI and sex can have on colorectal cancer risks. Rapid nutrition and lifestyle changes over the past two decades have led obesity to become a worldwide public health issue (Nestle, 2012; Behzad *et al.*, 2012; Chen *et al.*, 2013). The National Health and Nutrition Examination survey published data that stated prevalence of obesity has intensified from 22.9% - 30.5% between 1988 and 1994 to 55.9% - 64.5%, between 1999 and 2000 (Matheus *et al.*, 2013). Furthermore, The Health and Social Care Information Centre (2014) stated in their report that between 1993 and 2012 obesity rose from 16.4% to 25.1% among women and 13.2% to 24.4% in men.

Pastucha *et al.* (2013) examined the prevalence of metabolic syndrome and insulin resistance in 274 obese children (128 boys, 146 girls) aged 9-17 years. The aims of the study were to assess the prevalence of insulin resistance according to Quantitative Insulin Sensitivity Check Index (QUICKI) and Homeostasis Model Assessment for Insulin Resistance (HOMA-IR) homeostatic indexes, and to evaluate the diagnostic value of these respective indexes concerning the early detection of metabolic syndrome. The investigation used anthropometric and laboratory examinations to determine the prevalence of insulin resistance and metabolic syndrome. In the 274 obese subjects, 37% had metabolic syndrome was detected and most notably in 86% of participant's insulin resistance was found. The investigation highlighted a high occurrence of insulin resistance in obese children without metabolic syndrome. The research raises concerns regarding the current diagnostics criteria as the current criterion fails to highlight insulin resistance in the inaugural metabolic disturbances detection (Pastucha *et al.,* 2013). The study employed a large sample size and conformed to the International Diabetes Federation for children and adolescent's diagnostic criteria making the results highly reliable. On this basis, attention should preventative measures in insulin resistant obese children are required regardless of not meeting the metabolic syndrome diagnosis criteria.

Brown *et al.* (2000) investigated the relationship between BMI and cholesterol, hypertension, dyslipidaemia and blood pressure. Data from the United States among adults (1988-1994, National Health and Nutritional Examination Survery III) that included measurements of weight, height, lipids and blood pressure was investigated to quantify the relationship between the factors. Brown *et al*. (2000) found the prevalence of high blood pressure (both mean levels of diastolic and

systolic) increased as BMI increased. Furthermore, cholesterol increased when BMI was above 25 (overweight 25-29.9, obese > 30kg/m^2). Of note, cholesterol did not increase consistently with increasing BMI over 25. BMI was associated with significant increases in abnormal lipid profiles and high blood pressure. Brown's *et al.* (2010) research quantifies a strong relationship between BMI with abnormal lipids and hypertension and the results of this investigation highlight the importance of the prevention and control of obesity and overweight populations. Nevertheless, a substantial body of literature (Matsuzawa *et al.,* 1995; Bouchard, 2008; Dietz *et al.,* 2009; Kasner *et al.,* 2012) questions the reliability of BMI as a measure of obesity because BMI also takes into account lean body mass, thus, providing an inaccurate diagnosis. Matsuzawa *et al.* (1995) and Kasner *et al.* (2012) advocated visceral fat as the biggest contributor to hypertension, significantly more than BMI. Matsuzawa *et al.* (1995) and Hiuge-Shimizu *et al.* (2012) research highlighted visceral fat as the key foundation of CVD and hypertension.

2.2. Lipids in the Body

2.2.1. The molecular Structure of Fat

Triglycerides (TG) are the unique fatty-acids that comprise of a fat molecule that will determine its characteristics and properties (Abujazia *et al*., 2012). The number of carbon atoms in the fatty-acid will determine the molecules chain length (Flores-Martin *et al*., 2013). Every fatty-acid is comprised of a specific number of carbon and hydrogen atoms, the longer the chain, the more energy that TG molecule exerts when it is metabolised (Whitney *et al.*, 2010; Insel *et al.*, 2012).

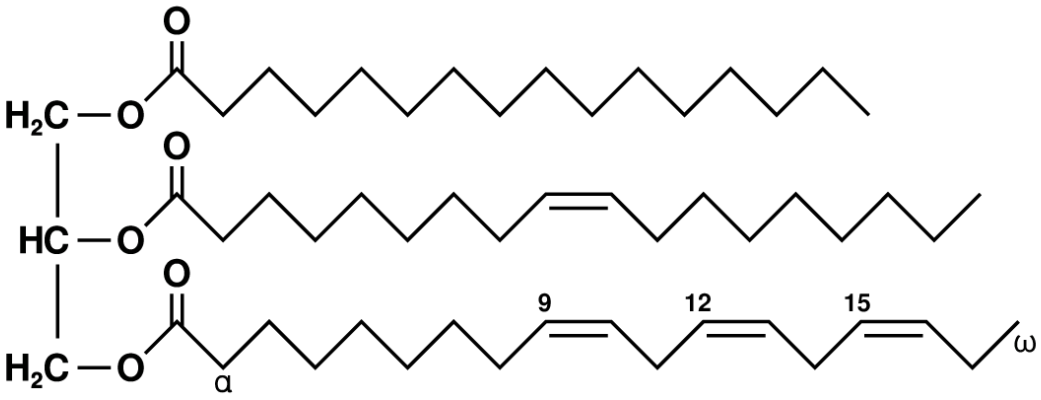

Figure 1.0 (Scienceinthebox, 2012) is an unsaturated TG molecule which consists of one glycerol molecule (left part) and three fatty-acids (right part).

TG is made up of glycerol and fatty-acids and the different forms of fat are diverged from different chemical structures (Insel *et al.*, 2013). TG are trimesters of glycerol, with the molecule being created as a result of the reaction between carboxylic acid and an organic alcohol (Patty *et al*., 2010). A TG molecule is similar to the shape of a capital E as is illustrated in *Figure 1.0* (Kabara, 2008). The connection between fatty-acids (horizontal) and glycerol (vertical) cause fats to have ester bonds (Dalgarno & Kingston, 2002).

Fat-soluble vitamins A, D, E and K throughout the body are transported by dietary fats (Insel *et al.,* 2013) and vital essential fatty acids (EFA) are distributed by fat (Whitney *et al.,* 2010). Fat plays a key role in the cushioning and the protection

of the internal organs and maintaining membrane structure (Webb *et al.,* 2008). Of note, EFA cannot be produced endogenously, they have to be attained through exogenous means (diet) (Gropper *et al.,* 2009). These EFA are proven to have a healthy benefit on heart health and immune function (Kabara, 2008). Scientific investigations have highlighted the benefits of omega-3 polyunsaturated (a vital EFA) on improving both heart and immune function (Ruxton *et al,* 2004). Furthermore, EFA found in coconut oil have also been attributed to these health benefits, especially the medium-chain triglycerides (MCTs) (Babu *et al.*, 2014).

2.2.2. Lipid Metabolism

The process that involves the degradation and intercourse of lipids is known as lipid metabolism (Glass & Olefsky, 2012). It is critical that to maintain systemic homeostasis the body controls lipid metabolism in a strictly regulated metabolic pathways (Saltiel & Kahn, 2001). The key facets of lipid metabolism are involved in the oxidation of fatty acids which synthesise lipids (Lipogenesis) to produce energy (Saltiel & Kahn, 2001). The metabolism of carbohydrates which might be converted to fats is closely connected with the metabolism of lipids (Zhang et al., 2013). Energy substance disorders are strongly associated with many complex diseases and scientific investigations have highlighted abnormalities in lipid metabolism can cause complex metabolic diseases, for example: obesity, cancer and diabetes (Xue *et al.,* 2011; Zhang *et al.,* 2013). Irregularities in lipid metabolism are vitally important in the prevention, understanding and early detection in several CVD and metabolic diseases (Weiss, 2007).

Laboratory studies (Glass & Olefsky, 2012) investigating lipid metabolism in individuals with type-2 diabetes have found numerous abnormalities with increased TG accretion in the: pancreas, muscles and liver which interferes with insulin signalling and secretion, affecting the body's ability to maintain homeostasis (Raz *et al.,* 2005). A constant overload in TG levels following high-fat meals is strongly correlated with damaging metabolic pathways leading to disease progression (Bae *et al.,* 2001). Individuals suffering with obesity often overload the central (abdominal) adipocytes with high volumes of TG and the central adipocytes are exceptionally sensitive to lipid mobilising hormones (Zechner *et al.,* 2012). Following a high-fat meal the portal circulation (circulates

blood from the small intestine to the liver) is overflowed with high-levels of TG at a metabolically unstable time; the portal circulation should be oxidised and not overflown with FFA (Zechner *et al.,* 2012). With increased TG levels in the portal circulation, a defective reaction is observed in the body's central adipocytes to lipolysis as insulin secretion becomes defective due to the enlarged visceral adipocytes (Spann *et al.,* 2012). Consequently, non-adipose tissues are exposed to increased levels of fat which causes numerous metabolic disturbances (So *et al.,* 2012). Over a chronic period of time, insulin resistance and beta-cell dysfunction occurs because of the ectopic accumulation of TG in the muscles, liver and pancreatic beta-cells (Zheng *et al.,* 2013).

Experimental studies in animals and humans have discovered that peripheral adipose tissue is precisely regulated (Glass & Olefsky, 2012). Peripheral adipose tissue performs a fundamental role in the protection of fluctuating TG in the body's circulation (Zechner *et al.,* 2012). The central adipose tissues can disturb the equilibrium by emitting a substantial amount of TG (Walther *et al.,* 2012). To decrease the volume of central adipocyte fat stores it is important to increase peripheral fat stores, this averts surplus fat deposition in the abdominal area. Lipoprotein is often overexpressed in obese patients in the abdominal adipocytes in comparison to subcutaneous fat stores, consequently, decreasing and regulating the activity of lipoprotein lipase (enzyme that hydrolyses TG) is needed for optimal homeostasis (Raz *et al.,* 2005). Healthy lifestyle adjustments incorporating diet choices and exercise can decrease mesenteric fat and activity, thus, reducing lipoprotein lipase activity.

Gradual and damaging metabolic disturbances ensue when multiplication in the adipose tissue occurs (Brindal *et al.,* 2012; Pastucha *et al.,* 2013). When insulin levels are high for an extensive period of time this can cause insulin secretion and signalling problems leading to insulin resistance (Reaven, 1988). Insulin resistance causes a decrease in the body's sensitivity to the hormone which is fundamentally considered a stage for prediabetes (Zhang *et al.,* 2013). The body needs to sustain a chronic state of hyperinsulinemia by the beta-cells continuously secreting insulin to prevent the deterioration of glucose tolerance (Zhang *et al.,* 2013). If this process fails then a gross decomposition of glucose homeostasis transpires often eventually leading to type-2 diabetes (Alibegovic *et al.,* 2009). When hyperinsulinemia is not sustained then plasma TG concentration

will not be regulated efficiently and normally resulting in the loss of homeostasis (Pastucha *et al.,* 2013). Due to the upsurge in plasma TG concentrations increased levels of hepatic glucose production will transpire and the significant increases in TG levels are directly linked with glucose intolerance and insulin resistance (Ebbert & Jensen, 2013; Kou et al., 2013).

2.2.3. Cholesterols Role in the Body

Cholesterol is an organic lipid molecule and is biosynthesised by all human and animal cells (Olson, 1998). It is required by animal cells to maintain membrane fluidity and cell structural integrity (Hanukoglu, 1992). Cholesterol enables the animal cell membrane to maintain cell-viability and integrity without the need of a cell wall unlike a plant and bacteria cell would. Thus, unlike plant and bacteria cells which are restricted by their cell walls, the cell can move about and change shapes (Olson, 1998). Furthermore, cholesterol acts as a precursor in the biosynthesis of bile acids, vitamin D and steroid hormones (Lithander *et al.,* 2013). Each cell synthesises cholesterol by a complex 37-step process started by HMG-CoA reductase, an intercellular protein enzyme (Eisa-Beygi *et al.,* 2013). However, high levels of lipids in the blood stream (including cholesterol) are strongly associated with the development of atherosclerosis (Dubois *et al.,* 1994; Lithander *et al.,* 2013).

A lipoprotein which is often associated with cholesterol is a biochemical assembly made of lipids and proteins which enable lipids to emulsify into the protein molecule to move through water inside and outside of cells (Kumar *et al.,* 2011). Lipids are able to be transported in the blood stream via the plasma lipoprotein particles which are classified under low-density lipoproteins (LDL) or high-density lipoproteins (HDL) (Lithander *et al.,* 2013). The lipoprotein particle metabolism refers to the handling of lipoprotein molecules in the body (El-Eter *et al.,* 2015). Two pathways divide the metabolism process, endogenous and exogenous and this is conditional on whether the lipoprotein molecules originated in the liver through de novo synthesis of triacylglycerol's (endogenous) or are composed mainly of dietary lipids (exogenous).

2.2.4. Postprandial Hypertriglyceridemia

Postprandial hypertriglyceridemia (PHTG) is a metabolic abnormality following the ingestion of a meal (Chan *et al.,* 2013). PHTG can encourage the development of atherosclerosis (Gaenzer *et al.,* 2001) and is now considered a risk factor in CVD development (Bae *et al.,* 2001). Chan *et al.* (2013) suggests PHTG is principally caused by an overproduction and/ or reduced catabolism of TG-rich lipoproteins and that PHTG is a consequence of pathological genetic conditions and additional coexistent medical conditions, predominantly insulin resistance and obesity. The retention of remnant particles in the artery wall is caused by an accumulation of TG-rich lipoproteins in the postprandial state (Boren *et al.,* 2014). Most remnant particles because of their size are not able to cross the endothelium efficiently as they cannot be emulsified by the lipoproteins, thus, they remain in the blood stream (Ooi *et al.,* 2001). Elevated levels of these particles lead to accelerated CVD and atherosclerosis because these remnant particles contain roughly 40 times more cholesterol than LDL (Boren *et al.,* 2014). The cardiovascular advantages of weight loss and exercise could inhibit advantageous effects of TG-rich lipoprotein metabolism and therefore lowering the amount of TG after a meal will reduce the levels of remnant particles.

An overload in TG during the postprandial state initiates endothelial dysfunction, inflammation, oxidative stress and the formation of small dense LDL particles (Chanu, 1999). Roberts *et al.* (2000) and Ceriello *et al.* (2002) postulate that PHTG leads to endothelial dysfunction through oxidative stress mechanisms though a rapid inactivation of nitric oxide via reactive oxygen species precipitating in endothelial dysfunction. The over-production of superoxide anion, which can decrease nitric oxide production, enables the potent long-lived oxidant peroxynitrite to rise in high concentrations (Cai & Harrison, 2000). The decreased bioavailability of nitric oxide might have damaging consequences in the regulation of vascular tone (McClean *et al.,* 2007). Research by Bae *et al.* in 2001 investigated the relationship between PHTG and the risk factors on atherosclerosis. The study analysed the acute effects of PHTG on oxidative stress and endothelial function which are critical pathways in the development of atherosclerosis. Bae *et al.* (2001) reported that TG levels were positively associated with the production of superoxide anion. In principle, PHTG stimulates the production of superoxide anions from the leukocytes, potentially via the up-

regulation of inflammatory markers. These superoxide radicals can then react with nitric oxide derived from the endothelium, resulting in a reduction in nitric oxide bioavailability Fukai *et al.*, 2002). The subsequent decrease in nitric oxide bioavailability may be responsible in the attenuation in endothelial function (Wang *et al.*, 2006). Bae et al. (2001) found that in a high-fat meal group serum TG levels and PMA-activated leukocyte superoxide anion production was significantly higher ($P < 0.05$) at 2 hours postprandial compared to the control. The analysis of data found an increase in TG levels was positively correlated with an increase in leukocyte superoxide anion production ($P = 0.001$). This study suggests acute PHTG increases oxidative stress and thus, endothelial dysfunction which might pave the way in the development of atherosclerosis over a chronic period of time.

In the clinical environment the acknowledgement of PHTG has been severely impeded by the deficiency in established clinical for investigating postprandial lipemia and by technical difficulties (Boren *et al.,* 2014). Moreover, for the type of dyslipidaemia there are no internationally established management guidelines. Additional work to crease a clinical protocol concerning the investigation of postprandial lipemia is required, along with internationally agreed management guidelines for PHTG (Chan *et al.,* 2013). This research will help manage PHTG and reduce risk factors of CVD and CHD.

2.2.5. Medium-Chain Triglycerides

Fats in food and in the body vary in length and they are chemically classified in accordance to the number of carbon atoms they possess (St-Onge *et al.*, 2008). MCT are TG which have an aliphatic tail between 6-12 carbon atoms and long-chain TG are 16 or more carbon atoms in length (St-Onge *et al.*, 2008). Like all TG, MCT constitute of three fatty-acids (2 or 3 of medium-chain in length) and a glycerol molecule. However, these fatty acids are metabolised very differently; MCT passively diffuse in to the portal system (long fatty-acids diffuse into the lymphatic system) from the GI tract without the constraint to modify like long-chain fatty-acids (Wanten & Naber, 2004). This requirement not to modify means they are metabolised faster and are a good source of immediate energy (St-Onge *et al.*, 2008). Interestingly, bile salts are not required for the digestion of MCT and as a result MCT are used in the treatment process in patients with malabsorption

and malnutrition syndromes as they do not require energy for utilisation, absorption or storage (Nosaka *et al.*, 2009). The human body can easily metabolise MCT so they are largely deemed a good biological inert source of energy (Nosaka *et al.*, 2009). MCT have potential advantageous attributes in protein metabolism, however, can be contraindicated in certain conditions as they tend to induce metabolic acidosis and ketogenesis (Wanten & Naber, 2004). MCT have been used successfully in the treatment of numerous malabsorption ailments because of their capacity for the body to absorb them rapidly (Takeuchi *et al.*, 2008). A low-fat diet coupled with MCT supplementation has been depicted as the foundation in the treatment of primary intestinal lymphangiectasia (Waldmann's disease) (Vignes & Bellanger, 2008). Numerous studies (Liu, 2008; Neal & Cross, 2010; Stafstorm & Rho, 2012) have publicised encouraging results for epilepsy and neurodegenerative disorders (e.g. Parkinson's disease and Alzheimer's) through the use of ketogenic dieting triggered by MCT supplementation.

Several studies have demonstrated MCT assist in the process of excess calorie burning causing weight loss (St-Onge & Jones, 2003; St-Onge *et al.,* 2008; Takeuchi *et al.*, 2008). St-Onge & Jones (2003) research aimed to examine the relationship between thermogenic responsiveness of MCT consumption and body composition. A randomised crossover controlled feeding trial in healthy overweight males (n=19) with a diet enriched in either long-chain triglycerides (LCT) or MCT for a period of 4 weeks each. Body weight decreased on day 28 ($P < 0.05$) with MCT intake compared with LCT consumption and fat oxidation was also increased in the MCT group ($P = 0.01$). However, on day 2 body weight ($P = 0.12$) and fat oxidation ($P = 0.08$) resulted in no significant change between MCT and LCT groups. Of note, men with a lower initial body weight had the greatest rise in energy expenditure on day 28 with MCT consumption which suggests MCT supplementation might enhance health benefits when elicited over a long time period. This data also suggests that MCT consumption might stimulate fat oxidation and energy expenditure as reflected by the loss of subcutaneous adipose tissue and body weight. These findings concur with St-Onge *et al.* (2008) who found MCT consumption over a 16-week supplementation period significantly reduced body weight in subjects. Furthermore, MCT significantly reduced diastolic blood pressure, fasting serum glucose and total

cholesterol. These results suggest MCT supplementation can be integrated into a weight-loss programme without the fear of adverse effects on CVD and metabolic risk factors. Despite a lot of research promoting the health benefits of MCT, some are met with methodological flaws. Kaunitz *et al.* (1958) reported weight loss in 20 obese patients of up to 13kg after a 2-month MCT regimen, however, this was during a low-energy diet (1200kcal). Consequently, the investigation's (Kaunitz *et al.,* 1958) data set is not reliable as any diet with a drastically reduced calorific intake will promote weight loss. This highlights the importance of studies with good methodological rigour needed in MCT research. It is worth noting however, the diet composed of 50g of MCT promoted increased weight loss and satiety compared with the LCT diet.

Hill *et al.* (1982) investigated non-obese volunteers (n=10) who were given a dietary intervention for 6 days which provided 150% of the recommended daily allowance of fat. A randomised crossover design was implemented with lipids ingested as 40% LCT or 40% MCT. At the end of each respective diet protocol, no significant changed in body weight was recorded which highlights that the overconsumption over dietary fat doesn't necessarily incur the weight gains government food guidelines would suggest. Furthermore, *in vivo* research by Gelibter *et al.* (1983) investigated if overfeeding rats with a diet enriched in MCT (45% of calories) would impede weight gains in comparison to rats overfed with high amounts of LCT. For 6 weeks, rats either consumed the MCT or LCT diet twice daily. Rats fed MCT gained 20% less weight ($P = 0.001$) than rats fed LCT. Furthermore, mean adipocyte size was smaller ($P < 0.05$) in the MCT-fed rats. The reduction in fat deposition in the MCT-fed rats might have occurred from obligatory oxidation of MCT fatty acids in the liver enabling almost no derivatives of MCT to be incorporated into body fat. The data suggests MCT has a potential mechanism for the prevention of obesity via nutritional strategies. However, research appears conflicted as a review by Costa *et al.* (2010) demonstrated. Costa *et al.* (2010) performed a literature review by selecting 14 long and short-term interventions of controlled clinical trials; among these only four showed an increase in energy expenditure, six showed a reduction in body weight amongst subjects and only one showed a beneficial effect on satiation. Thus, these inconclusive results require additional controlled studies with a standardised

amount of MCT to be investigated so MCT can be used in obesity nutritional treatment effectively.

Juxtaposed to Costa *et al.* (2010), a systematic review by Bueno *et al.* (2015) of randomised controlled trials concluded that individuals who replaced LCT with MCT showed a significant decrease in body weight (P = 0.001), body fat (P = 0.001) and waist circumference (P = 0.001). Furthermore, Mumme and Stonehouse's (2015) investigation concurred with Bueno *et al.* (2015) that MCT in comparison with LCT decreased body weight (P = 0.001), body fat (P = 0.001) and waist circumference (P = 0.001). Both investigations concluded that despite the significant results, the replacement of LCT with MCT must be cautiously taken.

Hauenschild *et al.* (2010) investigated the effect of a 7-day diet plan rich in MCT in a cohort of 32 patients with severe hypertriglyceridemia. After the diet period plasma TG levels decreased significantly (P < 0.05) and total cholesterol significantly decreased (P < 0.01). Of note, the diet was accepted and well tolerated by patients. These results suggest a diet rich in MCT can acutely be used in the treatment of hypertriglyceridemia by safely and rapidly lowering plasma TG levels. In addition, research by Yusoff *et al.* (2015) designed a similar study to Hauenschild *et al.* (2010) and investigated MCT as a potential treatment approach for managing hypertriglyceridemia. Yusoff *et al.* (2015) found the MCT intervention group displayed significantly decreased levels of plasma TG concentrations (P < 0.05) within a 28day period. Furthermore, research by Furman *et al.* (1965) and a review by Marten *et al.* (2006) concurred with Hauenschild *et al.* (2010) and Yusoff *et al.* (2015) that MCT can reduce TG levels due to the way it is metabolised differently to LCT.

Despite some studies finding inconclusive results, there is overwhelming evidence that MCT have health benefits which might aid in weight loss and reducing circulating blood TG levels. However, it is clear additional research is needed to determine the quantity required for the management of healthy body composition and body weight to confirm the efficacy of MCT supplementation.

2.3 Saturated Fat, Cholesterol and Heart Disease

2.3.1 The Lipid Hypothesis

In 1958 the lipid hypothesis was reported which is a theory that low-density lipoprotein (LDL) cholesterol in the blood is raised by ingesting saturated fat (Keys *et al.*, 1958; Keys *et al.*, 1986). This can lead to: atherosclerosis, CVD, CHD and early mortality (Babu *et al.*, 2014). Early research (Keys *et al.*, 1958) concluded that CHD was the result of poor nutrition and not the result of aging. Keys *et al.* (1986) sample size consisted of 12,763 males, 40-59 years of age, from four regions of the world (Northern Europe, Southern Europe, United Sates, and Japan) whom enrolled on the study as 16 cohorts. The research showed that total serum cholesterol was directly and independently linked at the population and individual level to an increased cardiovascular risk and heart disease (Keys *et al.*, 1986). The investigation further demonstrated that average SFA intake was strongly correlated with increased serum cholesterol level and 10-year CHD mortality rates. However, these strong correlations were mainly due to the large variations among the 16 cohorts in SFA intake, average serum cholesterol levels and heart disease mortality rates and the results of this study came under strong criticism. Yerushalmy and Hilleboe (1958) illustrated Keys *et al.* (1958) had only selected to analyse six countries from a possible 21 for which data was available from the original study. This facilitated the study to make a strong correlation between SFA and CHD. However, a full analysis of the complete dataset would have made the link between CHD and SFA intake far less apparent (Yerushalmy & Hilleboe, 1958). Yerushalmy and Hilleboe (1958) also stated that Keys *et al.* (1958) was investigating a tenuous association instead of any probable evidence of causation. Additionally, numerous other principal factors could have been considered but were overlooked. For instance, the consumption of sugar was not studied, yet might have been a more significant dietary intervention instead of fat and could have displayed a stronger correlation. Furthermore, in 1960 in Zutphen (Netherlands), a random sample of 878 males aged 40-59 were examined for the Seven Countries Study (Keys *et al.*, 1958). In Zutphen the average SFA intake in middle-aged men was high. SFA intake amounted to 18% of total energy intake with a small standard deviation of 3% of energy. The Zutphen study concluded that dietary SFA was not associated with 10-year CHD mortality and serum cholesterol. This further impairs the credibility of Keys *et al.* (1958) study, and

highlights the controversy surrounding the amendment to nutrition guidelines in 1961 concerning SFA.

Controversy still surrounds previous research concerning the causality and strength of the relationship between CHD mortality and dietary fat, especially as research of cholesterol has become more complex due to the different sub-particles of LDL and HDL (see page 28) (Uffe, 2002; Taubes, 2008). A recent meta-analysis in 2014 concluded that current evidence obviously does not support CVD guidelines that promote the low consumption of SFA (Chowdhury *et al.,* 2014). Nevertheless, Walter Willet of Harvard has confronted this study with dispute, continuing to support the older negative understanding of SFA (Kupferschmidt, 2014).

2.3.2. Saturated Fat and Research

Published review articles (Esrey *et al.*, 1996; Ravnskov, 1998; de Souza *et al.*, 2015) concluded that no association was found between SFA consumption and heart disease in observational studies. A review article (de Souza *et al.*, 2015) investigated the relationship between SFA and trans fat intake with risk of all-cause mortality CVD and type-2 diabetes. The investigation concluded that SFA consumption is not linked with CVD, CHD or type-2 diabetes (de Souza *et al.*, 2015). However, trans fat was associated with all-cause mortality.

A meta-analysis by Siri-Tarino *et al.* (2010) evaluated the association of SFA with CVD in prospective cohort studies, the objective was to summarise the evidence related to the association of the risk of CVD, CHD and stroke with dietary SFA in prospective epidemiologic studies. Throughout a 5 to 23 year follow up of 347,747 subjects, 11,006 developed a stroke or coronary heart disease and the consumption of SFA was not linked significantly with a greater risk of CVD, stroke or CHD. Consideration of study quality, sex and age did not change the results. Siri-Tarino *et al.* (2010) determined that there is no significant evidence for deducing that dietary SFA is linked with an increased likelihood in CVD or CHD. Additional meta-reviews by Ravnskov (1998) and Mente *et al.* (2009) also concurred with the research by Siri-Tarino *et al.* (2010) that SFA intake is not strongly associated with CVD. The conclusions drawn upon in these investigation's hold strong significance in the literature due to several strengths the research methods employed which included: the use of the random-effects

model which permitted for the heterogeneity of variance amid studies in their analyses and the inclusion of bigger studies with a substantial amount of incident cases which comprised in the selection of prospective epidemiologic studies that statistically adjusted for relevant covariates.

A caveat of Siri-Tarino *et al.* (2010) research was the dependence on the accuracy of the dietary assessments of the component studies. Depending on the method used, accuracy can vary particularly in overweight subjects. The underreporting of calories in dietary assessments has frequently contributed to error when research studies report statistics and findings (Willett, 1998). Commonly the most accurate method of dietary assessment is a 4-7 day food record but such means are usually not practical in large cohort studies. A solitary 24-hour recall is somewhat easier to gather, however, the data does not display chronic dietary patterns. In large epidemiologic studies Food Frequency Questionnaires have become the method of choice as they can assess long-term diets and are low-cost; nonetheless, they are subject to systematic and random errors.

The Women's Health Imitative study (Barbara *et al.*, 2006) is a long-term health study specifically focused on reducing colorectal cancer, breast cancer and CHD in postmenopausal women. A cohort of 48,835 postmenopausal women aged 50-79 years enrolled on The Women's Health Imitative study. The study's objective was to test the hypothesis that a diet low in SFA would lower the risk of CVD. Women were assigned to a comparison group (29,294 [60%]) or an intervention group (19,541 [40%]) in a free-living setting. The intervention group was designed to increase daily intake of vegetables, grains to at least 6 servings, and fruit to 5 servings, and reduce fat intake by 20% of calories. Mean fat intake decreased by 8.2% of total energy intake in the intervention group vs the comparison group by year 6. Furthermore, small decreases in SFA (2.9%), polyunsaturated (1.5%) and monounsaturated (3.3%) were observed. Diastolic blood pressure, LDL and cholesterol levels were significantly reduced by 0.31 mmHg, 3.55 mg/dL and 4.29% respectively in the intervention group. However, levels of TG, insulin, glucose and HDL cholesterol did not significantly change between groups. The reduced dietary fat and SFA diet had no significant effects on incidence of stroke or CHD. A reduced total fat intake over a mean of 8.1 years did not significantly reduce the risk of stroke, CVD or CHD in postmenopausal women. Furthermore,

it only achieved modest effects of CVD risk factors, suggesting a reduced fat dietary intervention does not reduce the risk of CVD disease. This randomised control trial by Barbara *et al.* (2006) concluded similar findings to previous research by Gorder *et al.* (1998). The Multiple Risk Intervention Trial involved 12,866 middle age men whom were at a high risk of CHD. Similar to the Women's health Imitative study, subjects were randomised into a low-fat diet group or a control group. After 6 years, no significant difference was detected between the groups.

Additional research (Yamagishi *et al.*, 2010) in a Japanese cohort (average SFA intake is low) focused on testing the hypothesis that SFA intake is linked with the risk of CVD mortality. A study cohort of 58,453 Japanese adults, aged 40-79 years participated in the study for a mean of 14.1 years. The research found SFA had inverse effects; a 31% reduction in mortality from stroke, and 18% reduction in mortality from CVD was revealed. These conclusions by Yamagishi *et al.* (2010) concurred with research by Takeya *et al.* (1984), Gillman *et al.* (1997), and Yamagishi *et al.* (2009), whom all concluded SFA intake decreased the risk of stroke mortality and CVD. However, research by Ginsberg *et al.* in (1998) differed in conclusions with this research. Ginsberg *et al.* (1998) studied the effects of reducing dietary SFA on plasma lipids and lipoproteins in healthy participants. The investigation conducted a randomised crossover-design trial with a cohort of 103 subjects (57 women and 46 men) aged between 22 to 67 years. Three diets were utilised in the study: an average American diet (AAD), a low-SFA diet and a Step 1 diet (a low-fat, low-protein, high-carbohydrate). Each diet was consumed for a total of 8 weeks with blood samples through weeks 5 to 8. The three respective compositions of each diet are as follows: AAD, 34.3% kcal fat and 15.0% kcal SFA; low-SFA, 25.3% kcal fat and 6.1% kcal SFA; and Step 1, 28.6% kcal fat and 9.0% SFA and all three diets provided ≈275 mg cholesterol/d. In comparison with the AAD, plasma total cholesterol fell 9% on low-SFA and 5% on Step 1. LDL cholesterol was reduced by 11% in the low-SFA group and 7% on Step 1 compared with the AAD (both $P < 0.01$). High-density lipoprotein (HDL) fell 11% on low-SFA and 7% on Step 1 (both $P < 0.01$) and plasma TG levels were elevated by 9% between AAD and Step 1, however, from Step 1 to low-SFA did not increase further. Amongst most subgroups, changes in plasma TG were not significant. Interestingly, plasma lipoprotein(a)

concentrations rose in a stepwise manner as SFA was decreased. Ginsberg *et al.* (1998) concluded that in a well-controlled feeding study, reductions in plasma total and LDL cholesterol levels were parallel with stepwise reductions in SFA. Previous research by Reiser (1973) and more recently by Muller *et al.* (2003) also concurred with Ginsberg *et al.* (1998); all three papers concluded that a reduction in SFA was correlated with beneficial effects on plasma cholesterol.

Nevertheless, some of the methods conveyed in Ginsberg's *et al.* (1998) research question the reliability of the results. The study's main focus was to determine the effects of reducing dietary SFA on plasma lipids, lipoproteins and thrombogenic factors, however, when SFA was reduced across groups carbohydrates correspondingly increased from 48% (AAD), 55% (Step 1) to 59% (low-SFA) respectively. As a result, the researchers cannot be sure that the reduction is LDL and total cholesterol was caused by a reduction in SFA or by an increase in carbohydrates. This parallels to the weaknesses of Keys *et al.* (1986) Seven Countries research. Both Ginsberg *et al.* (1998) and Keys *et al.* (1986) failed to keep carbohydrate consumption consistent. Consequently, how can they be sure an increase risk in heart disease is caused by SFA or carbohydrate ingestion? This is a methodological flaw and the research would have been able to draw stronger conclusions if carbohydrate levels were kept constant throughout. Numerous studies (Austin *et al.,* 2011; Wang *et al.,* 2012; Maki & Phillips, 2015) have highlighted the damaging effects a high-carbohydrate diet can have on metabolic pathways.

Despite several papers concluding the damaging effects SFA has on CVD and CHD risk factors, the majority of these papers have methodological flaws. The overwhelming amount of high-quality current literature which found no link between SFA consumption with CVD and CHD is titanic. Results of high-quality meta-reviews suggest publication bias, as studies tend to be accepted more favourably for publication with significant associations (Siri-Tarino *et al.,* 2010). If unpublished research with null associations were encompassed in Siri-Tarino's *et al.* (2010) analysis, the collective RR estimate for SFA consumption linked with CVD might be even nearer to null. It was this systematic review of the literature that found "there is no significant evidence for concluding that dietary saturated fat is associated with an increased risk of CHD or CVD" (Siri-Tarino *et al.,* 2010,

p.545). In a few years the reviews will probably conclude: there is significant evidence that SFA is protective against CVD and CHD.

2.4. Exercise and Medium Chain Triglycerides

2.4.1. Exercise and Medium Chain Triglyceride Supplementation

In recent years there has been a large interest in the bodybuilding community and with endurance athletes concerning MCT supplementation (Talbott *et al.,* 2006). This interest is owed to the different ways MCT is metabolised and used for energy compared with other fats and carbohydrates (Nevin & Rajamohan, 2008). However, Goedecke *et al.* (2005) found that MCT ingested prior to exercise negatively compromised sprint performance during prolonged ultra-endurance cycling exercise. Eight endurance-trained cyclists participated in the single-blind randomised crossover study and 1 hour before exercise subjects either ingested 32g of MCT or 75g of carbohydrates. Following this, subjects then drank 4.3% MCT + 10% carbohydrate (wt/vol) or 200 mL of a 10% carbohydrate (wt/vol) solution every 20 minutes throughout the respective trials. Between the MCT and carbohydrate group there was no difference in $\dot{V}O_2$ max and respiratory exchange ratio (P = 0.40). However, time-trial times between MCT (14:30 +/- 0.58) and carbohydrate (12:36 +/- 1:6) were significantly (P = 0.001) slower in the MCT time-trial. Furthermore, Clegg (2010) found no benefit in exercise performance when athletes supplemented MCT. It has been proposed that MCT have the ability to maximise an athlete's ability to maintain their glycogen stores. Nevertheless, only two studies to date (November, 2010) have found an improvement in exercise performance. Clegg (2010) concluded that MCT can beneficially alter body composition by increasing energy expenditure and fat oxidation and Clegg (2010) further added that future work on MCT research should focus on the applications and health benefits. However, further studies are required to test the effects of MCT on exercise performance in various levels of endurance trained cohorts.

2.4.2. Exercise and Lipid Metabolism

A growing body of literature postulates that exercise encourages the metabolism of TG levels following a high-fat load and this is strengthened by evidence (Gill *et al.*, 2004) that endurance-trained males display greater rates of TG clearance and reduced rates of PHTG. Further research (Padilla *et al.,* 2006) published moderate-intensity exercise (60% $\dot{V}O_2$max) ameliorates endothelial dysfunction stimulated by the consumption of a high-fat meal. Furthermore, Padilla *et al.* (2006) findings suggest not only can exercise counteract postprandial endothelial dysfunction following the ingestion of a high-fat meal, but also increase brachial artery flow-mediated dilation. Padilla *et al.* (2006) found when a high-fat meal was followed by a bout of moderate intensity exercise, an increase in brachial artery flow-mediated dilation (P = 0.001) transpired. Nevertheless, the study conveyed a small sample size (n=8) which hinders the strength of their findings.

A randomised control trial by McClean *et al.* (2007) investigated the effect of markers of postprandial oxidative stress and pulse wave velocity following moderate aerobic exercise. Trained male subjects (n=10, age 21.5 ± 2.5 years, $\dot{V}O_2$ max 58.5 ± 7.1 ml kg -1 min $^{-1}$) participated in a crossover design study. Subjects either ingested a high-fat meal alone (control), or a high-fat meal followed 2 hours after by 1 hour of moderate intensity exercise (60% max HR) and participants then switched to the other trial 7 days later. Pulse wave velocity, superoxide dismutase, blood lipid hydroperoxides, TG and other biochemical markers were measured at baseline, 1 hour, 2 hours, 3 hours and 4 hours postprandial. Pulse wave velocity increased in the control trial (P < 0.05) at 1 hour, 2 hours, 3 hours and 4 hours, respectively. Pulse wave velocity did not change in the exercise trial (P < 0.05) at 3 hours and 4 hours post ingestion, respectively. Lipid hydroxide levels in the exercise trial decreased at 3 hours postprandial compared to the control trial and superoxide dismutase levels at 3 hours postprandial were lower in the control trial compared to the exercise trial. Serum TG levels in both trials significantly increased at 2 hours, 3 hours and 4 hours postprandial when compared to the respective baseline measurements. However, serum TG's concentration significantly decreased in the exercise trial at 3 hours (immediately post-exercise) and 4 hours (1-hour post exercise) when compared against 2 hours post ingestion. These results postulate a single

session of moderate intensity aerobic exercise can reduce postprandial oxidative stress coupled with circulating TG levels and ameliorate impairments in vascular function and this concurs with Padilla *et al.* (2006) research findings.

2.5. Virgin Coconut Oil

2.5.1. Virgin Coconut Oil Profile

Virgin coconut oil (VCO) is acquired from a mature, fresh coconut kernel and is created without the use of heat or any refining process (Villarino *et al*., 2007). This process enables the phenolic compounds and antioxidant vitamins to be retained in the oil (Elamin *et al.,* 2012). VCO is a rich source of medium-chain triglycerides (MCT) (59.02% to 62.27%) and also contains a high amount of SFA (87%) (see appendix A for the fatty-acid profile of VCO used in this investigaiton) (Liau *et al*., 2011). These MCT can be metabolised quickly and are readily available for energy (Nevin & Rajamohan, 2008). The technical detail concerning the production of VCO are past the scope of this paper, but, are summarised in great detail by Krishna *et al.* (2010).

2.5.2. Virgin Coconut Oil Research

In recent years VCO has been expanding in popularity because of its potential cardiovascular benefits (Babu *et al.*, 2014). A contemporary prospective randomised trial (Liau *et al.*, 2011) found waist circumference in subjects was significantly reduced (2.87 ± 4.95 cm; $P = 0.02$) after a 4-week supplementation period of VCO. Another study in pre-menopausal women in the Philippines found VCO consumption (9.54 ± 8.92 grams) positively increased HDL concentration ($P < 0.05$) thus, advocating VCO is related with beneficial blood lipid profiles (Feranil *et al.*, 2011). In another clinical study (Law *et al.*, 2014) VCO was supplemented in patients with stage III or stage IV breast cancer; the cohort was either randomised to the intervention group (VCO) (n=30) or the control group (n=30). Quality of life was the main outcome measure and it was evaluated from the first to the sixth cycle of chemotherapy. The intervention group achieved significantly better quality of life questionnaire scores ($P < 0.01$) compared to the control group. Furthermore, the intervention group had better scores for

symptoms including, loss of appetite, sleep difficulties, fatigue, and dyspnoea compared to the control group. Law *et al.* (2014) concluded that VCO supplementation improved global quality of life and functional status in breast cancer patients and that VCO reduced the symptoms which are coupled with the side effects of chemotherapy.

It has been postulated that MCT have been found to be a possible antidepressant functional food; but, this had not been reviewed in VCO, which is rich in MCTs and polyphenols (Yeap *et al.*, 2015). Therefore, the aim of Yeap *et al.* (2015) study was to evaluate the antioxidant and anti-stress effects of VCO *in vivo* utilising mice with stress-induced injury. VCO was able to restore oxidative stress and reduce immobility time in mice. Furthermore, mice treated with VCO exhibited lower levels of brain 5-hydroxytryptamine, reduced weight of the adrenal glands and higher levels of brain antioxidants. Subsequently, glucose, corticosterone, serum cholesterol and TG levels were lower in VCO-treated mice. VCO might have potential value as anti-stress functional oil according to these results and in addition several further investigations *in vivo* (Abujazia *et al.*, 2012; Hayatullina *et al.*, 2012; Kamisah *et al.*, 2015) have found health benefits concerning VCO consumption. Nevertheless, these results were concluded in mice and rats respectively, it is not yet conclusive whether these benefits are observed in humans so additional clinical trials are required.

A strong body of literature (Abujazia *et al.*, 2012; Hayatullina *et al.*, 2012; Kamisah *et al.*, 2015; Yeap *et al.*, 2015) postulates that VCO has a number of health benefits. However, evidence concerning VCO supplementation is presently low in methodological rigor and is insufficiently lacking to conclusively recommend dietary alterations to improve CVD risk factors (Babu *et al.*, 2014). Research regarding cardiovascular benefits and VCO are at an early stage and are presently restricted to very few human trials and animal studies. However, initial compelling evidence suggests VCO may be advantageous in promoting cardiovascular health benefits (Kamisah *et al.*, 2015). Future research studies by preventive cardiology experts and nutritionists should give emphasis on the following areas of research to better address this area. Animal studies researching the biochemical effects of VCO consumption on lipid metabolism and the pathways altered in lipid metabolism through VCO consumption should be

performed. This would enable the literature to elucidate VCOs biochemical basis and its cardio-protective effects (Kamisah *et al.*, 2015). Translational research is also important along with laboratory studies and is an essential area of future research in assessing the potentially pivotal role of MCT in reducing CVD risk factors (Yeap *et al.*, 2015).

Long-term longitudinal prospective cohort studies amid various economies and populations to study their CVD risk factors and food intake are also strongly needed (Babu *et al.*, 2014). The effects of increased VCO consumption on primary and secondary prevention of CHD need to be assessed in clinically designed studies. As well as these trials, the combination of MCT/ VCO with exercise among various populations with and without CVD are critical clinical studies to better establish synergistic and independent roles in decreasing CVD risk. There appears to be a distinct lack of research investigating PHTG and subsequent oxidative stress, blood lipid profiles and the potential health benefits of VCO coupled with exercise. Reducing circulating TG levels after a meal will reduce risk factors associated with CVD. Thus, the aim of this study was to measure markers of postprandial oxidative stress and TG levels following an acute bout of exercise coupled with VCO consumption.

3.0. Hypotheses

3.1. Alternate Hypothesis

Moderate intensity exercise 1 hour postprandial of a high-fat milkshake will reduce the TG response significantly ($P < 0.05$) over a 4-hour period. VCO coupled with exercise will further reduce the TG response postprandial.

3.2. Null Hypothesis

Exercise will result in reducing the TG response after a high-fat milkshake. VCO coupled with exercise also did not result in reducing the TG response postprandial.

3.3 Dependant and Independent Variables

In a study quantifying the effect of exercise and VCO on TG response, the independent variable is the dosage of exercise and VCO. The dependant variable is the TG response of the subjects during the three trials. The controlled variables are: VCO brand, macronutrient ratio of the milkshakes, calorific value of the milkshake, the timing of the milkshake and exercise, subjects in a state of fast upon waking, when to take TG readings and the ability of the researcher team to accurately use laboratory equipment.

4.0. Methods

4.1. Subject Characteristics

Succeeding ethical approval by the University of Hull's Ethical committee and in accordance with the Declaration of Helsinki (2004), a sample of recreationally trained (participated in at least 2 hours of team or individual sport a week) males (*n=9*) were recruited for the present investigation using social media, posters and e-mail from the University of Hull's student population. All recruited participant's ethnicity was White British; females were not recruited due to their estrous cycle potentially being a methodological complication. Recruitment took place from April 1 2014 until October 12 2014. All participants completed a Medical Health Questionnaire (see appendix B) before involvement to safeguard from any health/ medical ailments that would compromise their participation. Subjects aged between 20 and 32 participated in the study and were excluded if they met any of the following; lipid-lowering medication, diabetes mellitus, chronic medical disease, current use of drugs which may influence lipid metabolism, psychological unsuitability and cardiovascular problems within the past 52 weeks. Detailed study information including requirements and potential risks were thoroughly explained before informed consent (see appendix C for consent form) was acquired from subjects.

4.2. Anthropometric Measures

Measurements for height and body mass were attained from all subjects upon arrival at the laboratory. Body mass was obtained using scales (Seca 761, England) and stature was recorded by means of a freestanding stadiometer (Seca 213, England). Body fat % was measured using a hand-held bioelectrical impedance machine (Omron BF306, England). Anthropometric measures of age, height, weight, and body fat % are provided in *Table 1.0*.

Table 1.0 Anthropometric measures (n=9). Note all values are means ± standard deviation.

Characteristic	Mean ± SD
Age (yrs)	26.0 ± 4.6
Body mass (kg)	81.4 ± 8.5
Height (cm)	177.1 ± 4.8
Body Fat (%)	15.5 ± 4.1
BMI (kg/m^2)	25.9 ± 3.2

4.3. Max Watt-Test

Before the trials, subjects completed a max watt -test on an isokinetic exercise bike (Daum Electronic, Germany) 2- 7 days prior to the experimental intervention. The results were used to determine the experimental exercise protocol intensity. The max watt-test protocol utilised a ramp protocol with an initial starting power of 100 watts and increased by 25 watts every 30 seconds and subjects cycled until failure. Rates of perceived exertion (RPE) (Borg, 1973) and heart rate was measured via a portable short-angle telemetry device (Polar Electro, Finland) every 30 seconds. Subjects max-HR was taken at the end of the test.

4.4. Experimental Design

In order to test the efficacy of VCO and exercise on circulating TG, a randomised, double blinded, cross over design was implemented. Participants were assigned using a lottery system randomised into one of the following groups: Group 1 (control) ingested a high-fat meal and did not participate in an exercise intervention. Group 2 consumed the identical experimental meal and completed the exercise protocol 1 hour postprandial. Group 3, ingested an alternative high-fat experimental meal which included VCO and completed the exercise protocol 1 hour postprandial. A fourth group of VCO and no exercise was not employed because the aim is to investigate the relationship between VCO and exercise. Baseline measurements were taken before the experimental meal was

consumed. The experimental meal was then consumed and further blood analysis was obtained 2 hours and 4 hours postprandial. Following a one week wash out period, the subject would return to the laboratory and would be allocated into a different group. Participants completed each group once. A schematic overview of the experiment is outlined in *Figure 2.0.*

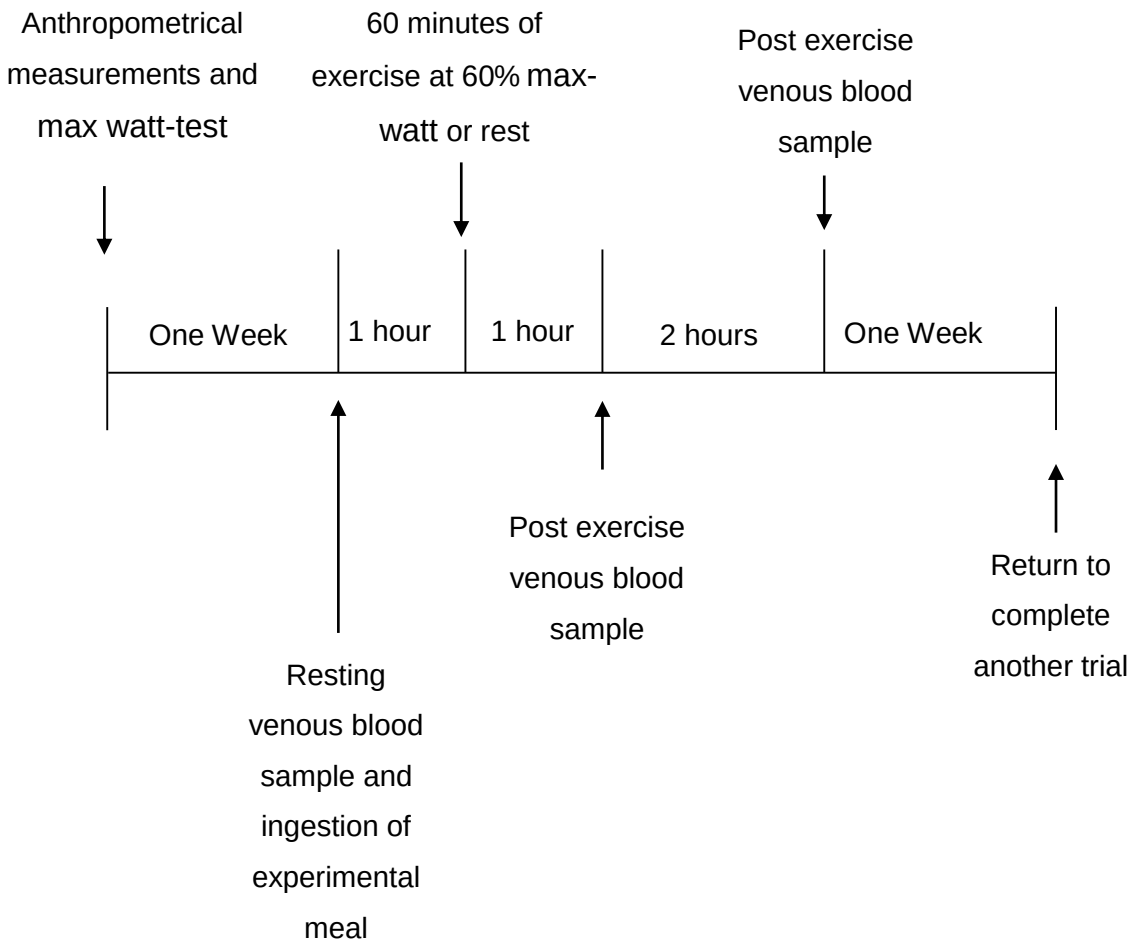

Figure 2.0 Schematic overview of the experimental protocol.

4.5. Experimental Meal

After baseline measures were obtained, subjects consumed the experimental high-fat meal which consisted of whole milk, premium dairy vanilla ice-cream and double cream (groups 1 and 2), or whole milk, premium dairy vanilla ice-cream and VCO (group 3) (see appendix D and E). The content of the meal was calculated in accordance with body mass, with each participant receiving 0.6g of

fat (76% energy intake), 0.2g of carbohydrates (15% energy intake) and 0.1g of protein (9% energy intake) per kg of body mass. For an 80kg male, this represents an intake of 515kcal. This energy intake is similar to a previous study were subjects achieved PHTG (McClean *et al.,* 2007). The investigation utilised this VCO dosage to ensure both experimental meals were very similar in calorific and macronutrient profiles. After baseline venous blood samples, subjects were required to consume the meal within a 15-minute period. Fluid consumption was restricted to water (500ml with meal), which subjects drank *ad libitum*.

4.6. Exercise Protocol

Subjects exercised 60 minutes after the consumption of the test meal for 60 minutes on an isokinetic bike (Daum Electronic, Germany). Subjects cycled at 60% of their max-watt and the watts were adjusted accordingly to ensure HR values and RPE were kept constant at 60%. This meant lowering or increasing the resistance (watts) on the isokinetic bike using the bikes programming system. Heart rate was measured via a portable short-angle telemetry device (Polar Electro, Finland). RPE was measured every 10 minutes (Borg, 1973).

4.7. Blood Biochemistry

All subjects were required to complete a 12 hour overnight fast and avoid high-fat foods for 24 hours which helps limit the dietary effects on plasma lipids (McClean *et al.*, 2007). In addition, caffeine was not allowed 24 hours prior. While subjects lay in the supine position a tourniquet was fitted to the bicep's distal region. Using a sterilised alcohol wipe (70%) a prominent forearm vein was selected and cleaned. Venous blood samples were collected using The Vacutainer™ (Becton-Dickinson, Oxford, UK) method. Blood was collected using Lithium heparin vacutainers (Becton-Dickinson, Oxford, UK). Following blood collection lithium heparin vacutainers were placed on ice for 10 minutes. Afterwards TG, HDL and total cholesterol levels were quantified in duplicate by pipetting 30 µl of whole blood out of the vacutainer using a 100 µl Eppendorf and onto a Reflotron test strip (Roche, Germany). The Reflotron test strips were analysed using the Roche Reflotron Plus (Roche, Germany). Results were displayed on the screen and then printed off.

Blood lactate, glucose, pH, pCO2 and pO2 were analysed using the ABL800 FLEX Analyser (Radiometer, England). A safePICO syringe (Radiometer, England) was inserted into the ABL800 FLEX Analyser inlet for automatic identification and mixing and aspiration of the blood sample. Results were printed off and recorded.

4.8. Statistical Analysis

Statistical analysis was carried out using SPSS statistic package-version 11 (Surrey, England). Unless otherwise stated, data was expressed as mean ± SD. Using a repeated measure two-way analysis of variance (ANOVA), data was analysed, with one within (time) and one between (trial) subject's factors. For a significant interaction effect, a Bonferroni-corrected paired samples t-test was used for analysis within subject factors. A one-way ANOVA with a posteriori Turkey Honestly Significant difference test were used to analyse between subject differences. $P < 0.05$ was established as the alpha level. The distribution of data was checked before performing parametric tests.

5.0. Results

The following section presents the main experimental findings across the control, exercise and VCO groups. Raw data sets can be found in appendix F (TG), G (Cholesterol), H (HDL); mean intervals (±SD) of biochemical markers are displayed in *Table 2.0*.

5.1. Compliance and Adverse Outcomes

All participants (n=9) completed this experiment (100%). Out of a possible 27 experimental high-fat meals, all doses were consumed giving a compliance rate of 100%. All bouts of exercise were successfully completed by the participants. As a consequence of VCO ingestion or exercise, no adverse physiological effects were reported during the trial period.

5.2. Triglyceride Response

Acute exercise and VCO supplementation caused a postprandial reduction in TG concentration ($P < 0.05$) in comparison to the control group. The investigation demonstrated a fall in TG levels at 2 hours (18.0%) and 4 hours (31.3%) postprandial between groups 1 and 2. Increased reductions in TG levels were demonstrated between groups 1 and 3 at 2 hours (36.8%) and 4 hours (38.6%) postprandial.

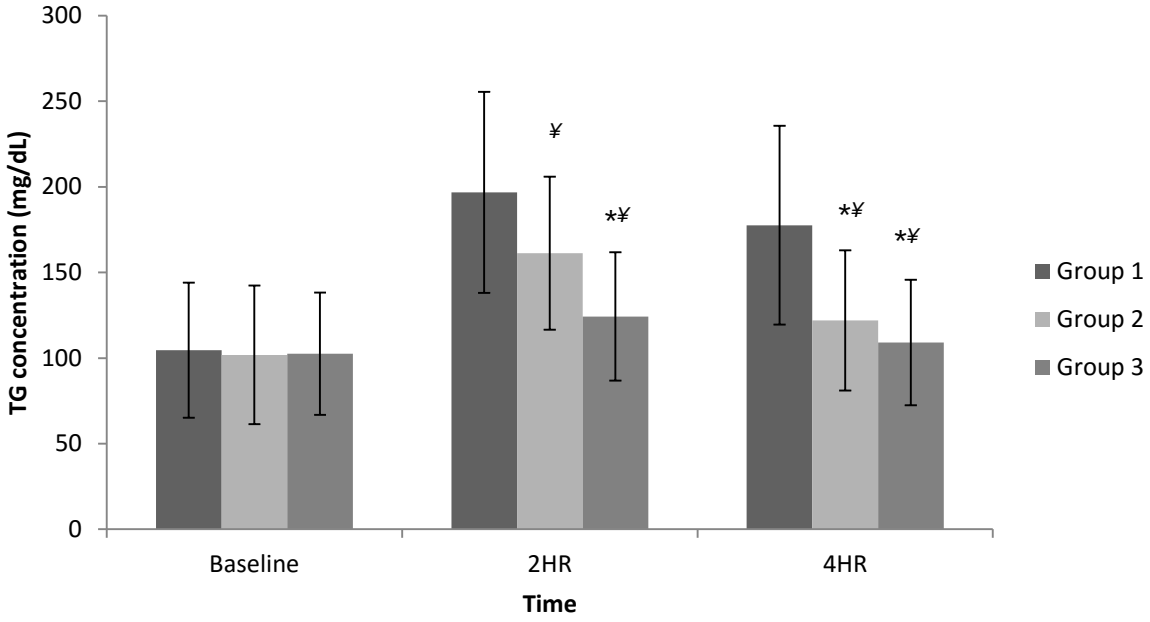

*Figure 3.0 Triglyceride concentration over time across all 3 trials (n=9). *denotes between group difference compared to control (P < 0.05) ¥denotes a within group difference compared to baseline (P < 0.05).*

5.3. Investigation Data Set

The data presented in *Table 2.0* shows the acute effects of VCO and exercise on blood lipids following a high-fat meal. The effects of VCO consumption and exercise on circulating TG levels are presented in *Figure 3.0;* no between group differences were found at baseline, but a significant difference was found at two hours ($P = 0.010$) and four hours ($P = 0.012$) postprandial between group 1 and group 3. Furthermore, a significant difference was found at four hours ($P = 0.044$) postprandial between group 1 and group 2. Cholesterol, HDL and glucose found no between group differences were found at baseline, two hours or four hours postprandial, respectively. Blood lactate and pCO_2 significantly increased in response to exercise ($P < 0.05$).

*Table 2.0 Blood lipids at baseline, 2 hours and 4 hours postprandial for groups 1, 2 and 3 (n=9). Note all values are means ± standard deviation. *denotes between group difference compared to control (P < 0.05) ¥denotes a within group difference compared to baseline (P < 0.05).*

	Baseline	2hr	4hr
Cholesterol (mmol/L)			
Group 1	8.42 ± 1.93	8.65 ± 1.86	8.72 ± 1.81
Group 2	8.09 ± 2.01	8.25 ± 2.01	8.29 ± 2.12
Group 3	8.58 ± 2.03	9.10 ± 2.13	9.02 ± 2.21
HDL (mmol/L)			
Group 1	1.23 ± 0.20	1.27 ± 0.19	1.25 ± 0.19
Group 2	1.19 ± 0.20	1.28 ± 0.19	1.15 ± 0.15
Group 3	1.22 ± 0.15	1.43 ± 0.19	1.29 ± 0.16
Glucose (mmol/L)			
Group 1	5.6 ± 0.7	5.0 ± 0.4	5.3 ± 0.2
Group 2	5.8 ± 1.0	4.8 ± 0.5	4.8 ± 0.5
Group 3	5.8 ± 0.6	5.0 ± 0.4	5.3 ± 0.5
Lactate (mmol/L)			
Group 1	1.1 ± 0.3	1.0 ± 0.4	0.8 ± 0.2
Group 2	1.0 ± 0.4	2.8 ± 1.3 *¥	0.9 ± 0.3
Group 3	1.0 ± 0.3	2.5 ± 1.2 *¥	1.1 ± 0.6
pCO_2 (mmHg)			
Group 1	55.3± 5.5	56.0 ± 5.2	51.9 ± 6.5
Group 2	48.6 ± 11.1	45.1 ± 5.8 *¥	49.6 ± 3.7
Group 3	49.6 ± 9.8	42.9 ± 7.3 *¥	50.0 ± 6.7
pO_2 (mmHg)			
Group 1	5.6 ± 0.7	5.0 ± 0.4	5.3 ± 0.2
Group 2	5.8 ± 1.0	4.8 ± 0.5	5.3 ± 0.4
Group 3	5.8 ± 0.6	5.1 ± 05	5.3 ± 0.5

6.0. Discussion

6.1. Interpretation of Data

The aim of this investigation was to measure biochemical markers of postprandial oxidative stress and TG levels following an acute bout of exercise and VCO supplementation. In the present study, experimental evidence highlights a decreased amount of venous blood TG concentration following exercise and VCO supplementation. Exercise and VCO was found to exhibit a prophylactic effect against biomarkers of oxidative stress compared to control trials. The increased reduction in TG concentrations when exercise was coupled with VCO may be due in part to the rapid metabolism of the MCT found in the oil.

This investigation is one of the first to research the effects of VCO consumption and moderate intensity exercise following the ingestion of a high-fat meal on TG levels. Mean TG in the control group significantly increased up to 4 hours following the consumption of the high-fat meal. The control trial (group 1) observed increases in TG concentrations at 2 hours and 4 hours postprandial. Significantly decreased TG levels were found in the exercise group (group 2) 4 hours postprandial and in the VCO group (group 3) at 2 hours and 4 hours postprandial, respectively. No significant change in cholesterol or HDL levels existed between groups.

A main finding of this study is that moderate intensity exercise and exercise coupled with VCO consumption attenuated the increase in TG levels following the consumption of a high-fat meal, as observed in the control group (see *Figure 3.0*). In all groups TG increased at 2 hours and 4 hours postprandial. However, in comparison with the control group, group 2 displayed significantly lower 4 hours post ingestion ($P = 0.044$) TG levels, and group 3 was lower at both 2 hours ($P = 0.010$) and 4 hours ($P = 0.012$) postprandial, respectively. Despite group 2 not showing a significant reduction in TG levels 2 hours postprandial, it is evident that exercise, and exercise with VCO blunted the postprandial increases in TG levels demonstrated in the control group.

The increase in TG levels observed in the present study's control trial concurs with previous research (Gaenzer et al., 2001; Gill *et al.*, 2004; Chan *et al.*, 2013; Boren *et al.*, 2014); the research demonstrated the ingestion of a high-fat meal

alone can significantly impair vascular function. Furthermore, the data in the control group suggests an augmented oxidative stress manifested by an increase in TG levels postprandial. Increased TG levels have been positively correlated with an increase in superoxide's (Padilla *et al.,* 2006). McClean *et al.* (2007) found following a high-fat meal, oxidative stress is elicited by an increase in lipid hydroxides and elevated TG levels. McClean *et al.* (2007) research concurred with Padilla *et al.* (2006) that TG levels are positively correlated with an increase in superoxide levels. McClean *et al.* (2007) concluded that their results suggest postprandial impairment is mediated though an oxidative stress mechanism. It is plausible that the increased TG levels observed during the current investigation's control trial mediated oxidative stress causing postprandial impairment by increasing superoxide levels. These findings support existing research (Bae *et al.*, 2001; Tsai *et al.*, 2004) that a high-fat meal causes oxidative stress. Therefore, a reduction in TG levels in the exercise and VCO trials following a high-fat meal in the present investigation highlights a potential mechanism to reduce postprandial oxidative stress.

In the present study when exercise was coupled with VCO supplementation it had positive effects on nitric oxide bioavailability due to the observed reduction in postprandial TG levels. The premise being, increased levels of TG are positively correlated with an increased production of superoxide anion (Wang *et al.*, 2006). The present investigation found VCO supplementation coupled with exercise blunted the TG response significantly which according to research would see a reduction in superoxide anion (Bae *et al.*, 2001; Fukai *et al.*, 2002; McClean *et al.*, 2007). The resulting reduction in superoxide anion will have positive effects on nitric oxide bioavailability which in turn will reduce CVD risk factors by not damaging endothelial cells through oxidative stress mechanisms (Wang *et al.*, 2006). However, the present study was not able to test for superoxide anions nor nitric oxide, so such conclusions cannot be drawn upon definitively. Nevertheless, TG data presented in the current investigation concurs with high-quality research (Bae *et al.*, 2001; Fukai *et al.*, 2002; McClean *et al.*, 2007) whom all significantly associated a reduction in TG levels with a positive effect on endothelial function and a reduction in postprandial oxidative stress.

Postprandial hypertriglyceridemia (PHTG) is the result of high blood TG levels and elevated levels of TG are strongly associated with the development of

atherosclerosis (Chan *et al.*, 2013). PHTG was observed in the present investigation during the control trial. PHTG follows the ingestion of a high-fat meal and is now considered a risk factor in CVD (Gaenzer et al., 2001). An over accumulation of blood TG levels in the postprandial state enables a retention of remnant particles in the artery wall (Boren *et al.*, 2014). These particles lead to the accelerated development of atherosclerosis. Research (Chanu, 1999; Ooi *et al.*, 2001; Ceriello *et al.,* 2002) would suggest the remnant particles would have occurred during the control trial in the present investigation because of the elevated TG levels observed in the subjects (*see Figure 3.0)*. The present study measured cholesterol levels, however, did not measure sub-fractions of LDL; such conclusions of remnant particles of LDL can only be hypothesised based on previous research.

Research (Gill *et al.*, 2004; McClean *et al.*, 2007) concurs with the present investigation's findings regarding TG levels and exercise. The present study found a bout of moderate intensity exercise (60%) following a high-fat meal attenuated to a reduction in circulating TG levels. It has become common knowledge of the health benefits of exercise in reducing the risk of CVD (Kristo *et al.*, 2013); the optimal intensity to achieve such benefits is still debated. A large body of literature postulates that single bouts of moderate intensity exercise may reduce PHTG by 20% (Gill & Hardman, 2003; Padilla *et al.,* 2006; McClean *et al.*, 2007). The current investigation displayed a reduction in TG levels of 18.0% (2 hours) and 31.3% (4 hours) postprandial between group 1 and group 2. Further reductions in TG levels were observed between group 1 and group 3 at 2 hours (36.8%) and 4 hours (38.6%) postprandial. The observed reduction in TG levels in the present investigation highlights improved vascular function following a single session of moderate intensity exercise by significantly reducing postprandial TG levels. This finding is consistent with research by Gill *et al.* (2004), McClean *et al.* (2007) and Kristo *et al.* (2013) and further supports the theory that exercise reduces postprandial TG levels.

The mechanisms in which exercise counteracts the deleterious effects of a high-fat load appear multifunctional. First, exercise can potentially act through a direct flow mechanism (Padilla *et al.*, 2006). Padilla *et al.* (2006) investigated the effects of endothelial function with acute moderate intensity exercise. The research found a bout of moderate intensity exercise (60% VO_2 max) following a high-fat

meal increased brachial artery flow-mediated dilation and caused a reduction in TG levels. As the present investigation demonstrated a reduction in TG levels, it is possible exercise in current study caused an increase in brachial artery flow-mediated dilation within subjects. This would in-turn reduce the risks factors associated with CVD following a high-fat meal. Additional research by McClean *et al.* (2007) looked at the effect of postprandial oxidative stress markers and pulse wave velocity following moderate aerobic exercise (60%). McClean *et al.* (2007) highlighted a significant reduction in pulse wave velocity, TG levels, lipid hydroxides and an increase in superoxide dismutase in the exercise group. Interestingly, in McClean *et al.* (2007), Gill *et al.* (2004), and Padilla *et al.* (2006) results, reduced TG levels were strongly correlated with positive changes in pulse wave velocity. Therefore, in the present investigation positive effects on pulse wave velocity following exercise could have been mediated via a decrease in TG levels. Thus, findings in the present study postulate a reduction in postprandial TG levels in groups 2 and 3 might attenuate a reduction in oxidative stress and increase endothelial function in subjects is plausible.

In the current study, a session of moderate intensity exercise with VCO consumption resulted in further decreased TG levels compared with the exercise group as highlighted in *Figure 3*. It is worth noting that in the exercise group, TG levels were not significantly reduced 2 hours postprandial but they were 4 hours post ingestion. Of note, VCO coupled with exercise resulted in significantly reduced TG levels at both 2 hours and 4 hours postprandial, respectively. This may in part be due to the MCT in the VCO which are rapidly absorbed (Nosaka *et al.*, 2009). Wanten and Naber (2004) attenuate the rapid absorption to the fact MCT passively diffuse into the portal system from the GI tract and are rapidly oxidised in the liver without the requirement to modify like LCT. It has been hypothesised that this rapid absorption of MCT leads to an increase in energy expenditure, subsequently decreasing the deposition into the adipose tissue (Marten *et al.,* 2006). This is a possible explanation why numerous studies (St-Onge & Jones, 2003; Takeuchi *et al.*, 2008; Bueno *et al.,* 2015) have shown a reduction in body weight following MCT consumption. Cohorts in MCT research tend to be in a clinical or obese population; therefore, it was not known whether the current study's lean cohort display beneficial effects on atherosclerosis risk factors following MCT supplementation. The current study suggests that in a lean

population (body fat 14.8% ± 4.4), VCO consumption had a positive effect on atherosclerosis risk factors by reducing postprandial TG levels.

Plasma TG levels increase following the ingestion of a fat-containing meal and return to baseline 6-12 hours later (Costa *et al.*, 2010). The amount of TG levels postprandial is confidently correlated with CVD risk factors (Marten *et al.,* 2006). In the current study postprandial TG levels were significantly lower with the intake of MCT rather than the control or exercise group which had increased LCT intake. The current investigation's findings parallel research by St-Onge *et al.* (2008) and Hauenschild *et al.* (2010) who's research documented that the acute supplementation of MCT significantly reduced TG levels in subjects. Of note, St-Onge *et al.* (2008) and Hauenschild *et al.* (2010) experiments were carried out with very high amounts of chronic MCT dosage in the diet and they did not utilise the use of exercise in the study. Therefore, it remained uncertain whether the present study's relatively low and acute MCT dosage coupled an exercise intervention would have similar effects on TG levels which was demonstrated in St-Onge *et al.* (2008) and Hauenschild *et al.* (2010) research. Despite this, the current investigation found that exercise enhances the positive effects of MCT consumption on postprandial TG levels and consequently reduces CVD risk factors by increasing TG clearance (Padilla *et al.,* 2006). Furthermore, the present investigation postulates that even in small doses, MCT are rapidly absorbed and utilised for energy immediately and therefore a reduction in postprandial TG production was observed (Costa *et al.*, 2010).

Despite the present study finding that small doses of MCT reduces TG levels, MCT consumption might increase TG production, especially when consumed in excess of calorific needs (Costa *et al.*, 2010). Consequently, this would increase TG levels and might account for elevated fasting serum TG levels over time (Marten *et al.,* 2006). As TG and cholesterol secretion are regulated in a synchronised way, an overproduction in TG may lead to an increase in cholesterol levels and might accelerate atherosclerosis development by increasing plasma cholesterol (Eisa-Beygi *et al.*, 2013). However, a study by Gelibter *et al.* (1983) explored the effect of ingesting excess calories with a diet enriched in MCT (45% of calories). Rats fed MCT gained 20% less weight (P = 0.001) than rats fed LCT. This suggests that even when MCT are consumed in excessive amounts they have protective mechanisms against obesity and the

development of atherosclerosis. The results from Gelibter *et al.* (1983) research might be attributed to a reduction in TG levels in the MCT fed rats. Despite Gelibter *et al.* (1983) not measuring postprandial TG levels, similar methods were conveyed in the current research study concerning the comparison of LCT *vs.* MCT. The present investigation found a significant reduction in TG levels between subjects ingesting MCT opposed to LCT. It appears dietetic supplementation of MCT does not attenuate to an abnormal amount of TG levels in the blood postprandial and promotes a reduction in abdominal obesity (Costa *et al.*, 2010). The present study highlights the potential importance in supplementing MCT through food sources like VCO to reduce growing obesity and CVD rates. Further research is needed to quantify the optimum dose of MCT.

A large body of literature suggests the advantages of MCT on blood lipid profiles and CVD health benefits (St-Onge & Jones, 2003; St-Onge *et al.*, 2008; Takeuchi *et al.*, 2008; Neal & Cross, 2010; Stafstorm & Rho, 2012; Kamisah *et al.*, 2015). In recent years, a focus of literature has investigated the potential effects of VCO due to its rich MCT profile on CVD. Yeap *et al.* (2015) found that VCO reduced TG levels along with serum cholesterol levels. The present study's results concurred with Yeap *et al.* (2015) findings that VCO lowers TG levels. However, the present study saw no change in cholesterol levels across the 3 trials. As a consequence, the current study suggests that exercise and exercise coupled with VCO supplementation following a high-fat meal does not have a positive effect on cholesterol levels 2 hours and 4 hours postprandial. It is worth noting that the majority of studies found VCO had a positive impact on cholesterol levels over a longer time period (6+ days). Of note, the current study did not account for remnant particles of LDL. It is plausible that while cholesterol levels remained the same in this study amongst subjects, a reduction in remnant particles existed in the VCO and exercise groups, as previous acute research might suggest (McClean *et al.*, 2007; Kamisah *et al.*, 2015). The present study only explored the effects of an acute bout of exercise and VCO consumption on blood lipids and consequently there is need for further research investigating the effects of acute and chronic exercise and VCO intervention on blood lipids.

The current study provides evidence that VCO supplementation prior to exercise had no positive effect on exercise performance. To evaluate this, no significant change in heart rates, RPE or blood lactate existed between both the exercise

groups. It is postulated that VCO has the ability to maximise an athlete's ability to maintain their glycogen stores (Talbott *et al.,* 2006). Nevertheless, very few studies have found an improvement in exercise performance with MCT supplementation. The results in the present investigation concur with findings by Clegg (2010) that MCT consumption did not result in a significant improvement in exercise performance. Clegg (2010), Goedecke *et al.* (2005) and the current study suggest that future MCT research should focus on the applications and health benefits and not exercise performance. However, the current study was not designed to thoroughly investigate the effects of MCT *vs.* LCT in relation to exercise performance, and in consequence cannot provide a substantial claim on the effect of MCT on exercise performance. Additional studies are required to test the effects of MCT on exercise performance which are specifically designed to test performance.

In the current study VCO supplementation caused between group differences in HDL levels at 2 hours and 4 hours postprandial compared with the control trial. Despite the data not being significant, a rise in HDL occurred in all but one subject postprandial suggesting that VCO might increase HDL. This postulation is supported by significant findings in MCT research by Mensink *et al.* (2003) that VCO consumption positively increases HDL concentration when supplemented. The MCT found in the VCO in the present investigation may have caused the increase in HDL. These findings suggest that VCO and exercise may have advantageous effects in response to HDL metabolism which may decrease the atherosclerosis risk factors associated with a high-fat meal. The one particular subject whom had a higher HDL reading in the control group at 2hour post ingestion had a baseline HDL reading of 26.3 mg/dL in the control group and in comparison had a baseline reading of 21.3 mg/dL and 21.4 mg/dL in the other two trials, respectively. It is plausible that the subject was not 12 hour fasted which caused this increase in baseline venous HDL. However, as no direct evidence of this is presented it is not possible to make any definitive conclusions. Nevertheless, research by Hill *et al.* (1990) and St-Onge *et al.* (2008) found that MCT consumption does not lead to an increase in HDL. Future research concerning VCO supplementation and HDL is required to draw stronger conclusions of the effects it has.

The current study is important in further understanding the benefits of MCT consumption and exercise on reducing TG levels following a high-fat meal. VCO and exercise has a role to play in the prevention of chronic disease and obesity. However, several limitations need to be highlighted in the current investigation which should be addressed in future research.

Firstly, the investigation studied the effects of VCO and exercise on postprandial TG levels and was not able to detect differences in superoxide aninions, endothelial function and superoxide dismutase levels. Therefore, the ability for the current study to draw conclusions of postprandial oxidative stress is limited to blood lipid profiles. This is an area for consideration for the future if further research is to be conducted. Nonetheless, the investigation consisted of a good scope of TG data which has been proven to be strongly correlated with postprandial oxidative stress (McClean *et al.*, 2007). Future research by cardiology experts and nutritionists should give emphasis on investigating the acute and chronic effects of VCO and exercise on preventing atherosclerosis risk factors. In addition, these studies should use different doses of exercise and VCO to determine the optimal levels in reducing atherosclerosis risk factors. Similar dietary investigations usually employ a longer time period (minimum of 4 weeks). A longer time frame may allow for stronger conclusions to be elicited on the effects of VCO and exercise following a high-fat meal which will enable further scope for analysis. The resultant lengthier time period of VCO supplementation might have exhibited a significant difference in cholesterol and HDL between control and VCO group in the current study. The investigation could then have studied subjects fasting blood lipid levels and if they were affected following chronic VCO supplementation. However, this study still highlights the acute benefits of VCO consumption and exercise on atherosclerosis risk factors by the attenuation of TG levels.

As MCT are absorbed and metabolised faster than LCT, the addition of another time point for taking venous blood (1 hour postprandial) would have enabled stronger analyses. The premise being that TG levels in the VCO group might have peaked at this time point due to the faster rates of metabolism. Furthermore, the addition of further time points at 5 hours and 6 hours postprandial would have facilitated the investigation to see how long it took for TG levels to return to baseline. However, the current method provided enough scope to strongly

recommend exercise and VCO as good facilitators in reducing circulating blood TG levels. In addition, another VCO group which did not exercise would have permitted stronger analysis of the effects of MCT on postprandial TG levels as the current study only observed the health benefits of VCO intake when coupled with exercise. Furthermore, the cohort came from the same ethnic background and location, thus, cohort studies amongst numerous economies and populations to study their CVD risk factors and food intake are also strongly needed. Additional research is required to understand if similar effects occur in women as the cohort was all males, the future inclusion of both sexes will address this limitation.

7.0. Conclusion

Findings from the current investigation indicate that moderate intensity exercise performed 1 hour postprandial can control TG levels following PHTG in male participants. Furthermore, when exercise is coupled with VCO these effects are further enhanced. Supplementary evidence suggests the rapid metabolism of the MCT in VCO enabled greater TG clearance following moderate intensity exercise. These findings might suggest that VCO and exercise may have advantageous effects on lipid metabolism following PTHG and could reduce the risk factors associated with atherosclerosis risk factors stimulated by a high-fat meal. It is likely that the reduction in blood TG levels mediated by exercise and VCO imposed a positive change in oxidative stress markers. This suggests that even when MCT are consumed in relatively low amounts, they provide protective mechanisms against obesity and the development of atherosclerosis. Further research is needed to quantify the optimum dose of MCT.

Despite the prevalence of atherosclerosis risk factors being low in a cohort of young males (age 26 years ± 4.66), nutrition transition is rapid and the problem of PHTG might increase atherosclerosis risk factors. Henceforth, early measures to prevent elevated TG levels through exercise and VCO are highly recommended following a high-fat meal. However, it needs to be emphasised that the current study only investigated the acute effects of VCO when coupled with exercise and future research should be conducted for a longer period of time and investigate the effects of VCO without exercise on TG levels. This allows further investigation of the acute and chronic benefits of VCO and exercise on blood lipid profiles and atherosclerosis risk factors. This study highlights the importance of VCO and exercise as a potential prophylactic and therapeutic intervention to CVD by reducing postprandial TG levels.

8.0. References

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9.0. Appendices

Appendix A – Fatty acid profile of VCO

Table 3.0 The Coconoil fatty acid profile of the VCO used throughout the study.

Certificate Number: TCHT692981-1 Final

ECLIPSE SCIENTIFIC GROUP

Lab Ref.	Sample Details	Method Number	Test	Result	Units	Flag
CHT2307386	Desc: Ref: Sample. OVCO. Order No: BWA-12/SL/OVCO Date Received: 30/05/2012	AM/C/603	Salt (Determined Via Chloride)	<0.05	g / 100g	
		AM/C/107	FAME C6.0 Caproic Acid	0.56	g / 100g	
		AM/C/107	FAME C8.0 Caprylic Acid	7.13	g / 100g	
		AM/C/107	FAME C10.0 Capric Acid	5.59	g / 100g	
		AM/C/107	FAME C12.0 Lauric Acid	46.63	g / 100g	
		AM/C/107	FAME C14.0 Myristic Acid	18.76	g / 100g	
		AM/C/107	FAME C14.1 Myristoleic Acid	<0.01	g / 100g	
		AM/C/107	FAME C15.0 Pentadecanoic Acid	0.01	g / 100g	
		AM/C/107	FAME C16.0 Palmitic Acid	7.61	g / 100g	
		AM/C/107	FAME C16.1 Palmitoleic Acid	0.01	g / 100g	
		AM/C/107	FAME C17.0 Heptadecanoic Acid	<0.01	g / 100g	
		AM/C/107	FAME C17.1 Heptadecenoic Acid	<0.01	g / 100g	
		AM/C/107	FAME C18.0 Stearic Acid	3.00	g / 100g	
		AM/C/107	FAME C18.1 Oleic Acid	5.23	g / 100g	
		AM/C/107	FAME C18.2 Linoleic Acid	0.90	g / 100g	
		AM/C/107	FAME C18.3 Linolenic Acid (omega 3)	<0.01	g / 100g	
		AM/C/107	FAME C18.3 Linolenic Acid (omega 6)	<0.01	g / 100g	
		AM/C/107	FAME C18.4 Octadecatetraenoic Acid	<0.01	g / 100g	
		AM/C/107	FAME C20.0 Arachidic Acid	0.08	g / 100g	
		AM/C/107	FAME C20.1 Gadoleic Acid	0.03	g / 100g	
		AM/C/107	FAME C20.2 Eicosadienoic Acid	<0.01	g / 100g	
		AM/C/107	FAME C20.3 Eicosatrienoic Acid (omega 3)	<0.01	g / 100g	
		AM/C/107	FAME C20.3 Eicosatrienoic Acid (omega 6)	<0.01	g / 100g	
		AM/C/107	FAME C20.4 Arachidonic Acid (omega 3)	<0.01	g / 100g	
		AM/C/107	FAME C20.4 Arachidonic Acid (omega 6)	<0.01	g / 100g	
		AM/C/107	FAME C20.5 Eicosapentaenoic Acid	<0.01	g / 100g	
		AM/C/107	FAME C22.0 Behenic Acid	0.01	g / 100g	
		AM/C/107	FAME C22.1 Erucic Acid	<0.01	g / 100g	
		AM/C/107	FAME C22.4 Docosatetraenoic Acid	<0.01	g / 100g	
		AM/C/107	FAME C22.5 Clupanodonic Acid	<0.01	g / 100g	
		AM/C/107	FAME C22.6 Docosahexaenoic Acid	<0.01	g / 100g	
		AM/C/107	FAME C24.0 Lignoceric Acid	0.03	g / 100g	
		AM/C/107	Saturated Fatty Acids (in sample)	89.42	g / 100g	
		AM/C/107	Monounsaturated Fatty Acids (in sample)	5.28	g / 100g	
		AM/C/107	Polyunsaturated Fatty Acids (in sample)	0.90	g / 100g	
		AM/C/107	Estimated Total Omega3 (In Fat)	<0.1	g / 100g	
		AM/C/107	Estimated Total Omega 3 (In Sample)	<0.1	mg / 100g	
		AM/C/107	FAME C15.1 Pentadecenoic Acid	<0.01	g / 100g	

The results for saturated, monounsaturated and polyunsaturated fatty acids in the sample use a 0.956 conversion factor for non fatty acid material in the fat.

The values above for the total monounsaturated fatty acids and total polyunsaturated fatty acids are inclusive of both cis and trans components.

Timothy Lumb

Laboratory Manager - Nutritional Chemistry

For and on Behalf of Eclipse Scientific Group

Appendix B – Pre-Medical Health Questionnaire

Pre-Exercise Medical Questionnaire

The information in this document will be treated as strictly confidential

Name:

.....

Date of Birth: ..... Age: ..... Sex: .....

Blood pressure: …… Resting Heart Rate: …….

Height (cm): ……..... Weight (Kg): ……...

Please answer the following questions by putting a circle round the appropriate response or filling in the blank.

1. How would you describe your present level of **exercise** activity?
   Sedentary / Moderately active / Active / Highly active

2. Please outline a typical weeks exercise activity

…….....

…….....

…….....

3. How would you describe your present level of **lifestyle** activity?
   Sedentary / Moderately active / Active / Highly active

4. What is your occupation?

…….....

5. How would you describe your present level of fitness?
   Unfit / Moderately fit / Trained / Highly trained

6. Smoking Habits Are you currently a smoker? Yes / No
   How many do you smoke …….. per day
   Are you a previous smoker? Yes / No
   How long is it since you stopped? ..... years
   How many did you smoke? ..... per day

7. Do you drink alcohol? Yes / No

   If you answered **Yes** and you are male do you drink more than 28 units a week?
   Yes / No

If you answered **Yes** and you are female do you drink more than 21 units a week?

Yes / No

8. Have you had to consult your doctor within the last six months?
Yes / No
If you answered **Yes**, Have you been advised **not** to exercise?

Yes / No

9. Are you presently taking any form of medication?
Yes / No
If you answered **Yes**, Have you been advised **not** to exercise?

Yes / No

10. Do you have a history of fainting during or following exercise?
Yes / No
If **Yes**, please provide details.....
.....
.....

11. To the best of your knowledge do you, or have you ever, suffered from:

a Diabetes? Yes / No **b** Asthma? Yes / No
c Epilepsy? Yes / No **d** Bronchitis? Yes / No
e ✸Any form of heart complaint? Yes / No **f** Raynaud's Disease Yes / No
g ✸Marfan's Syndrome? Yes / No **h** ✸Aneurysm / embolism? Yes / No
I Anaemia Yes / No

12. ✸Are you over 45, and with a history of heart disease in your family?
Yes / No

13. Do you currently have any form of muscle or joint injury?
Yes / No
If you answered **Yes**, please give details…….
……
……..

14. Have you had to suspend your normal training in the last two weeks?
Yes / No
If the answer is **Yes** please give details……
……
……

15. ✸ Please read the following questions:
a) Are you suffering from any known serious infection? Yes / No
b) Have you had jaundice within the previous year? Yes / No
c) Have you ever had any form of hepatitis? Yes / No
d) Are you HIV antibody positive Yes / No
e) Have you had unprotected sexual intercourse with any person from an HIV high-risk population? Yes / No

f) Have you ever been involved in intravenous drug use? Yes / No
g) Are you haemophiliac? Yes / No
16. As far as you are aware, is there anything that might prevent you from successfully completing the tests that have been outlined to you? Yes / No.

IF THE ANSWER TO ANY OF THE ABOVE IS YES:

a) Discuss with the test administrators or another appropriate member of the department.

b) Questions indicated by (✸) answered yes: Please obtain written approval from your doctor before taking part in the test.

PLEASE SIGN AND DATE AS INDICATED ON THE NEXT PAGE

Participant Signature: ……
Date……

Test Administrator:……..
Date……
Supervising staff member.....
Date.....

Parent (if minor)…….. Date: …….

THIS SECTION IS ONLY REQUIRED FOR RETURN VISITS!

For any future testing sessions it is necessary to verify that the responses provided above are still valid, or to detail any new information. This is to ensure that you have had no new illness or injury that could unduly increase any risks from participation in the proposed physical exercise.

ANSWER THE FOLLOWING QUESTION AT EACH REPEAT VISIT.

Is the information you provided above still correct, and can you confirm that you have NOT experienced any new injury or illness which could influence your participation in this exercise session?

Repeat 1	Yes / No*	Signature:	Date:
*Additional info required:			
Repeat 2	Yes / No*	Signature:	Date:
*Additional info required:			
Repeat 3	Yes / No*	Signature:	Date:
*Additional info required:			

Repeat 4	Yes / No*	Signature:	Date:
*Additional info required:			
Repeat 5	Yes / No*	Signature:	Date:
*Additional info required:			

Figure 3.0 The example of health screening questionnaire given to subjects.

Appendix C – Consent Form

Informed Consent Declaration

Project title	The effect of acute virgin coconut oil supplementation and aerobic exercise on triglycerides following postprandial hypertriglyceridemia in healthy males.
Principal investigator	Name: Dr Mark Fogarty Email address: m.fogarty@hull.ac.uk Contact telephone number: 01482 463270
Student investigator (if applicable)	Name: Craig Scott Email address: c.scott@2013.hull.ac.uk Contact telephone number: 07896774398

Please Initial

I confirm that I have read and understood all the information provided in the Informed Consent Form (EC2) relating to the above project and I have had the opportunity to ask questions. ☐

I understand this project is designed to further scientific knowledge and that all procedures have been risk assessed and approved by the Department of Sport, Health and Exercise Science Research Ethics Committee at the University of Hull. Any questions I have about my participation in this project have been answered to my satisfaction. ☐

I fully understand my participation is voluntary and that I am free to withdraw from this project at any time and at any stage, without giving any reason. I have read and fully understand this consent form. ☐

Name of participant Date Signature

Person taking consent Date Signature

Figure 4.0 An example of the consent from given to subjects to sign.

Appendix D – Control and Exercise High-Fat Meal Composition

Nutritics for milk

Male, 24, 80kg, 188cm, 22.6 BMI,

Showing Day - ALL of 1

milk

20th Feb 2014 - 21st Feb 2014

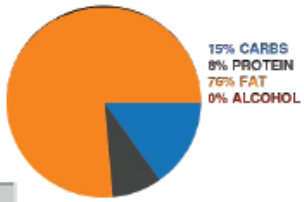
NUTRIENT	INTAKE	RNI	LIMITS
- ENERGY -			
Energy(Kcal)	521	2232	
Energy(Kj)	2153		
- MACRONUTRIENTS -			
! Carbohydrate	21g	268-387g	
! Protein	11g	64-112g	<160g
! Fat	44g	50-87g	>37g
! Water	273ml	1786-2678ml	
Alcohol			<15.9g
- CARBOHYDRATE COMPONENTS -			
Starch			
Oligosaccharide	0.1g		
! NSP		18-24g	
Sugars	20g		<65g
- LIPID COMPONENTS -			
! Saturated fat	28g		<25g
! Monounsaturated fat	11.7g	32-50g	
! Polyunsaturated fat	1.6g		>6.5g <25g
! › omega3(n-3)	0.2g	1.5-3g	>0.5g
! › omega6(n-6)	0.7g		>2.5g
Trans-fatty acids	1.6g		<2.5g
Cholesterol	104mg		<300mg
- MINERALS & TRACE ELEMENTS -			
! Sodium	149mg	1600mg	>500mg <2400mg
! Potassium	507mg	4700mg	>1600mg
Chloride	241mg	2500mg	
! Calcium	370mg	700-1500mg	>400mg <1500mg
Phosphorus	304mg	1000mg	>300mg <5000mg
Magnesium	36mg	350mg	<500mg
! Iron	0.1mg	9mg	>7mg <45mg
! Zinc	1.3mg	14mg	>5mg <25mg
! Copper		1.7mg	>0.4mg <10mg
! Manganese		5.5mg	>1.4mg
! Selenium	5µg	75µg	>20µg <400µg
Iodine	124µg	150µg	>70µg <1100µg
- VITAMINS -			
Vitamin A (ret eq)	614µg	1500µg	>500µg <9000µg

! Vitamin D	0.3µg	10-20µg	>2.5µg <80µg	
! Vitamin E	1.2mg	19mg	>4mg <540mg	
Vitamin K_1	4.7µg	120µg		
! Thiamin (B_1)	0.1mg	0.9mg	>0.6mg <100mg	
Riboflavin (B_2)	0.8mg	1.3mg	>0.8mg <40mg	
! Niacin total (B_3)	2.7mg	14.7mg	>12mg <35mg	
! Pantothenic Acid (B_5)	2.1mg	3-7mg		
! Vitamin B_6	0.2mg	1.7mg	>1mg <100mg	
! Folic Acid (B_9)	27µg	300-600µg	>100µg <1000µg	
Vitamin B_{12}	2.8µg	2.4µg	>1µg <2000µg	
! Biotin (B_7)	7.8µg	25-60µg	<900µg	
! Vitamin C	6mg	220mg	>40mg <2000mg	
- OTHER -				
GL	6.3			N/A
PRAL	0.3			N/A

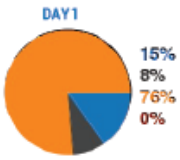
Figures from Nutritics guidelines for male non-athletes 17-55 years old

Generated by Nutritics v3.06 on 20 Feb 2014

Macronutrient Analysis



	CARBOHYDRATE	PROTEIN	FAT	ALCOHOL
Intake	20.5g	11g	44.2g	0g
g/kg body-weight	0.3	0.1	0.6	0
Kilocal	79	44	397	0
Kilocal %	15%	8%	76%	0%



Diet Log

DAY 1

Whole milk, average	250g
Cream, fresh, double, including Jersey cream	50g
Ice cream, dairy, premium	50g

Figure 5.0 A macronutrient and micronutrient composition of the high-fat meal used in the control and the exercise group.

Appendix E –High-Fat Meal Composition for Virgin Coconut Oil Trial

Nutritics for milk

Male, 24, 80kg, 188cm, 22.6 BMI,

Showing Day - ALL of 1

milk

20th Feb 2014 - 21st Feb 2014

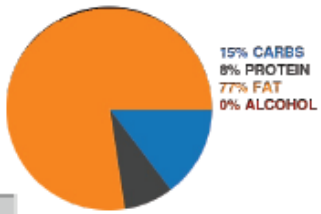
NUTRIENT	INTAKE	RNI	LIMITS	LOWER LIMIT / ACTUAL INTAKE / RECOMMENDED / UPPER LIMIT
- ENERGY -				
Energy(Kcal)	515	2232		
Energy(Kj)	2130			N/A
- MACRONUTRIENTS -				
! Carbohydrate	19.7g	268-387g		
! Protein	10.2g	64-112g	<160g	
! Fat	44g	50-87g	>37g	
! Water	249ml	1786-2678ml		
Alcohol			<15.9g	
- CARBOHYDRATE COMPONENTS -				
Starch				N/A
Oligosaccharide	0.1g			N/A
! NSP		18-24g		
Sugars	19.6g		<65g	
- LIPID COMPONENTS -				
! Saturated fat	34g		<25g	
! Monounsaturated fat	6.4g	32-50g		
! Polyunsaturated fat	1.1g		>6.5g <25g	
! › omega3(n-3)		1.5-3g	>0.5g	
! › omega6(n-6)	0.4g		>2.5g	
Trans-fatty acids	0.7g		<2.5g	
Cholesterol	35mg		<300mg	
- MINERALS & TRACE ELEMENTS -				
! Sodium	138mg	1600mg	>500mg <2400mg	
! Potassium	475mg	4700mg	>1600mg	
Chloride	223mg	2500mg		
! Calcium	345mg	700-1500mg	>400mg <1500mg	
! Phosphorus	278mg	1000mg	>300mg <5000mg	
Magnesium	34mg	350mg	<500mg	
! Iron	0.1mg	9mg	>7mg <45mg	
! Zinc	1.2mg	14mg	>5mg <25mg	
! Copper		1.7mg	>0.4mg <10mg	
! Manganese		5.5mg	>1.4mg	
! Selenium	3.5µg	75µg	>20µg <400µg	
Iodine	106µg	150µg	>70µg <1100µg	
- VITAMINS -				
! Vitamin A (ret eq)	184µg	1500µg	>500µg <9000µg	

! Vitamin D	0.2µg	10-20µg	>2.5µg <80µg	
! Vitamin E	0.5mg	19mg	>4mg <540mg	
Vitamin K_1	1.8µg	120µg		
! Thiamin (B_1)	0.1mg	0.9mg	>0.6mg <100mg	
! Riboflavin (B_2)	0.7mg	1.3mg	>0.8mg <40mg	
! Niacin total (B_3)	2.6mg	14.7mg	>12mg <35mg	
! Pantothenic Acid (B_5)	2mg	3-7mg		
! Vitamin B_6	0.2mg	1.7mg	>1mg <100mg	
! Folic Acid (B_9)	23µg	300-600µg	>100µg <1000µg	
Vitamin B_{12}	2.5µg	2.4µg	>1µg <2000µg	
! Biotin (B_7)	7.4µg	25-60µg	<900µg	
! Vitamin C	5.5mg	220mg	>40mg <2000mg	
- OTHER -				
GL	6.1			N/A
PRAL	-0			N/A

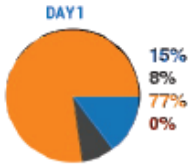
Figures from Nutritics guidelines for male non-athletes 17-55 years old

Generated by Nutritics v3.06 on 20 Feb 2014

Macronutrient Analysis



	CARBOHYDRATE	PROTEIN	FAT	ALCOHOL
Intake	19.7g	10.2g	44.3g	0g
g/kg body-weight	0.2	0.1	0.6	0
Kilocal	76	41	398	0
Kilocal %	15%	8%	77%	0%



Diet Log

DAY1

Whole milk, average	250g
Ice cream, dairy, premium	50g
Coconut oil	27g

Figure 6.0 A macronutrient and micronutrient composition of the high-fat meal used in the VCO trial.

Appendix F – Triglyceride Raw Data

Table 4.0 The raw data for all subjects and time points for TG levels (mg/dL).

Subject	*Group1 Baseline*	*Group 1 2hr*	*Group1 4hr*	*Group 2 Baseline*	*Group 2 2hr*	*Group 2 4hr*	*Group 3 Baseline*	*Group 3 2hr*	*Group 3 4hr*
1	70	180	160	70	113	91	70	97.9	70
2	78.5	182	146	70	95	90	70	90.2	80.9
3	184	332	266	122	191	161	146	161	152
4	147	215	276	183	225	162	150	162	133
5	70	136	112	70	115	70	70	70	70
6	94.9	213	190	146	204	180	147	161	164
7	75.2	131	122	78.4	165	77.9	96.1	90.8	78
8	101	186	173	78	179	130	73	127	104
9	121	196	153	99.1	164	136	101	159	130
Mean	104.62	196.78	177.56	101.83	161.22	121.99	102.57	124.32	109.10
Standard Deviation	39.40	58.65	58.07	40.48	44.68	40.89	35.73	37.51	36.59

Appendix G – Cholesterol Raw Data

Table 5.0 The raw data for all subjects and time points for cholesterol levels (mg/dL).

Subject	*Group1 Baseline*	*Group 1 2hr*	*Group1 4hr*	*Group 2 Baseline*	*Group 2 2hr*	*Group 2 4hr*	*Group 3 Baseline*	*Group 3 2hr*	*Group 3 4hr*
1	185	192	185	159	159	158	156	176	161
2	136	138	136	129	134	129	158	164	165
3	111	120	117	101	103	100	104	105	103
4	168	170	180	153	162	164	168	190	193
5	110	115	120	100	100	102	115	121	121
6	141	153	152	151	164	167	154	160	161
7	124	122	132	123	130	128	124	136	132
8	192	195	194	194	170	180	197	199	199
9	198	197	199	201	215	217	215	224	228
Mean	151.67	155.78	157.22	145.67	148.56	149.44	154.56	163.89	162.56
Standard Deviation	34.76	33.84	32.61	36.28	36.04	38.18	36.58	38.36	39.93

Appendix H –High-Density Lipoprotein Raw Data

Table 6.0 The raw data for all subjects and time points for HDL levels (mg/dL).

Subject	*Group1 Baseline*	*Group 1 2hr*	*Group1 4hr*	*Group 2 Baseline*	*Group 2 2hr*	*Group 2 4hr*	*Group 3 Baseline*	*Group 3 2hr*	*Group 3 4hr*
1	20.2	21.9	20.9	17.9	17.6	17.3	21.2	26.5	21.8
2	24.8	21.3	21.1	23.8	26.3	19.5	24.2	28.1	25.6
3	22.7	22.4	22.5	21.9	19	23.1	22.4	25.1	24.5
4	26.3	28	26.8	21.3	22.5	18.2	21.2	24.5	22.6
5	21.2	23.4	23.1	21.4	24.4	22.9	22.5	28.6	22.9
6	15.5	17.1	16.9	15.4	21.5	19	15.8	17.9	18
7	19.3	20.1	19.8	19.1	21.2	19.5	18.9	24.6	20.7
8	26.7	27.1	27.5	26.9	26.7	23	27.7	29.8	26.4
9	23.1	25	24.1	25.3	27.5	24.9	24.2	27.2	26.5
Mean	22.20	22.92	22.52	21.44	22.97	20.82	22.01	25.81	23.22
Standard Deviation	3.59	3.42	3.35	3.62	3.49	2.67	3.39	3.49	2.83