The effect of active video gaming on blood lipids as a measure of cardiovascular disease risk

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Abstract

The purpose of the study was to investigate the effects of a four week Nintendo Wii aerobics intervention on blood lipids. Specifically, HDL, LDL, TC, TG and the LDL: HDL ratio. A sample of fifteen (4 males, 11 females) individuals from the University of Central Lancashire took part in the study. Participants were divided into two groups based on their cardiorespiratory fitness levels (low and high). Both groups engaged in the four week Wii aerobics programme, a two week washout period and a control condition containing no exercise. The order in which the groups completed the conditions was randomised. Blood samples were taken pre and post both conditions. Blood pressure, body composition and estimated VO_{2max} were assessed as part of a basic health screen. Additionally, a MetaLyser ® 3B was used to measure respiratory gases whilst exercising. Paired samples t-tests were used to establish significant differences in the measured variables pre to post exercise and control conditions. It was established that a four week Nintendo Wii aerobics intervention significantly ($p \leq 0.01$) increased HDL cholesterol and significantly ($p \leq 0.05$) reduced the HDL:LDL ratio in both fitness groups. No significant differences were found for LDL, TC, or TG in either fitness group. Moreover, the intervention resulted in a significant ($p \leq 0.05$) decrease in resting SBP and estimated VO_{2max} in both fitness groups. A significant ($p \leq 0.05$) reduction in exercising EE, relative VO_2 and METs was established in the low fitness group. In conclusion, it was established that Nintendo Wii aerobics may help to improve numerous cardiovascular risk markers, including; lipid profiles, blood pressure and cardiorespiratory fitness. Specifically, individuals with low cardiorespiratory fitness seem to benefit the most from active gaming. It was also demonstrated that participants show a high level of adherence to active gaming sessions.

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Acknowledgements

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1. INTRODUCTION

Cardiovascular disease (CVD) is the most prevalent chronic disease and the leading cause of death worldwide (Petersen & Pedersen, 2005). It is responsible for the deaths of 3.8 million men and 3.4 million women every year (Hausenloy & Yellon, 2008). In the UK, around one quarter of all deaths are attributable to coronary heart disease (CHD) (BHF, 2008). Specifically, it is estimated that around 3.3 million people in the UK are living with CVD (BHF, 2008). In the US, the number of people with one or more types of CVD is roughly 81,100,000 (AHA, 2010).

There are several risk factors for CVD. Non-modifiable risk factors are age, family history and male gender, whilst modifiable risk factors include high cholesterol, hypertension, smoking, diabetes, obesity and physical inactivity (BHF, 2008). CVD risk is increased further by a clustering of such risk factors. For example, having high total cholesterol (TC) and being physically inactive (Dobiášová, 2004). It is thought that the primary risk factors for CVD are an increase in sedentary behaviours and a decrease in physical activity (Marcus *et al*., 2006). Such findings have been established in classic epidemiology studies (Taylor *et al*., 1962; Paffenbarger *et al*., 1986; 1993). In particular, sedentary activities are believed to be the leading preventable cause of death (Marcus *et al*., 2006). Identifying enjoyable, original modes of exercise may be a way in which physical activity levels can be increased, particularly in young adults (Sell *et al*., 2008).

The expanding popularity of the gaming industry in recent years, coupled with the introduction of user friendly games has resulted in videogames becoming a huge part of modern day society (Dixon *et al*., 2010). Specifically, recent advances in technology have resulted in games consoles that allow the user to exercise whilst playing, for instance, the Nintendo Wii (Mintel, 2009). Such games may provide an alternative

mode of physical activity for individuals that do not enjoy conventional exercise (Lanningham-Foster *et al*., 2009). Physical activity, particularly aerobic exercise (Kelley & Kelley, 2006), may provide protection against CVD by improving lipoprotein profiles (Kraus *et al*., 2002), consequently reducing the risk of atherosclerosis (Petersen & Pedersen, 2005; Pahkala *et al*., 2010).

1.1. Lipids

A poor lipoprotein profile is a well established risk factor for CVD (Kelley & Kelley, 2006). Specifically, a low level of high density lipoproteins (HDL) and elevated levels of low density lipoproteins (LDL) are primary risk factors (ACSM, 2009). Furthermore, elevated triglyceride (TG) levels are also a well established risk factor for CVD (ACSM, 2009). Elevated plasma concentrations of lipids, predominantly cholesterol, are related to the pathogenesis of atherosclerosis, which is responsible for the majority of CVD (Hausenloy & Yellon, 2008). Effective management of irregular blood lipid levels reduces the risk of cardiovascular disease mortality (Trejo-Gutierrez & Fletcher, 2007).

1.1.1 High Density Lipoprotein

HDL contains the least cholesterol (20%) and the most protein (50%) of all the lipoproteins (Bouchard *et al*., 2006). HDL cholesterol (HDL-C) is inversely correlated to CVD (ACSM, 2009). The importance of HDL in reducing the risk of CVD is a result of its potential to remove cellular cholesterol. Additionally, the anti-inflammatory, antithrombotic and antioxidant effects of HDL act in unison to enhance endothelial function and reduce atherosclerosis (Hausenloy & Yellon, 2008). Gorden *et al*., (1989) suggested that a 0.026 mmol/L or 1% decrease in HDL cholesterol is associated with that a 2-3% increase in CHD risk. Previous research has also reported that individuals with HDL levels greater than 0.9 mmol/L had roughly 70% less risk of CHD than those

with levels below 0.9 mmol/L (Hausenloy & Yellon, 2008). However, it is not known if raising HDL levels alone is enough to combat CVD, without changes in both LDLs and TG (ACSM, 2009).

1.1.2 Low Density Lipoproteins

A sedentary lifestyle and increase in body fat are two contributing factors for an increase in concentration of serum LDL (McArdle *et al*, 2006). LDL molecules have the highest cholesterol content of all the lipids (Bouchard *et al*., 2006). They carry around 60-80% of total serum cholesterol and have the highest affinity for cells in the arterial wall (McArdle *et al*., 2006). The oxidation of LDL contributes to the proliferation of smooth muscle cells, which damage and narrow the arteries. Consequently, the pathogenesis of atherosclerotic plaques occurs (McArdle *et al*., 2006). A 1% reduction in LDL cholesterol may potentially reduce the frequency of coronary episodes by 2% (Pedersen *et al.,* 1998 cited in Kelly & Kelly, 2006). Moreover, Baigent *et al*., (2005) suggested that there is a reduction in the risk of major cardiovascular events by 21% for every 1 mmol/L decrease in LDL.

1.1.3 Total Cholesterol

Epidemiological research has established strong, independent relationships between plasma TC and the prevalence of CHD, even in those with normal or somewhat elevated TC levels (Downs *et al*., 1999). In the US, 46.8% of the population have high TC (NHANES 2003-2006 cited in AHA, 2010). The average TC level of adults living in England and Scotland is 5.5 mmol/L (BHF, 2009). These levels are high given the desirable range is less than 5.1 mmol/L (ACSM, 2009). Moreover, the Framingham

Heart study (Ho *et al*., 1993) revealed that 40% of individuals with TC levels ranging between 5.17 and 6.47 mmol/L developed a myocardial infarction.

1.1.4. Triglycerides

TG are an important modifiable risk factor for CVD (ACSM, 2009). High levels of TG have been shown to be independently related to an increased risk of CHD (Stampfer *et al*., 1996; Sarwar *et al.,* 2007), however their relevance to disease is unclear (Baigent *et al*., 2005). It is thought that elevated levels of TG may lead to adverse changes in HDL and LDL. The hydrolysis of triglyceride-rich lipoproteins such as chylomicrons by lipoprotein lipase (LPL), leads to the transfer of cholesterol to HDL from these particles (Bouchard *et al*., 2006). As a result, the amount of cholesterol carried by HDL increases (Bouchard *et al*., 2006), leading to the formation of small dense HDL particles (Gibney *et al*., 2002). Small dense HDL particles are rapidly catabolised by the liver, leading to reductions of circulating HDL concentrations. Similar processes are said to effect LDL. However, small dense LDL particles are inadequately recognised by normal LDL receptors and consequently remain in the circulation for longer periods of time. Due to the smaller size and longer half life, small, dense LDL is more efficient in penetrating the endothelium, thus contributing to arthrosclerosis (Gibney *et al*., 2002).

1.1.5 LDL: HDL Ratio

The National Cholesterol Education Programme (NCEP) recommends targeting LDL and HDL as part of lipid lowering interventions (Fernandez & Webb, 2008). However, it is thought that the LDL: HDL ratio has a greater diagnostic value than either LDL or HDL alone (Manninen *et al*., 1992) and consequently is an excellent predictor of CVD risk (Fernandez & Webb, 2008). Specifically, a continuous, graded association between

LDL: HDL ratio and death from CVD has been reported (Fernandez & Webb, 2008). It has also been accounted that a one unit difference in the LDL: HDL ratio is associated with a 17% higher risk of CHD (Panagiotakos & Toutouzas, 2003). Furthermore, Fernandez and Webb (2008) reported findings that suggest a one unit increase in the LDL: HDL ratio can increase the risk of myocardial infarction by 53-75%.

1.2. Exercise and Lipids

It is suggested that regular aerobic exercise can significantly elevate HDL levels, as well as lower TC, LDL and TG (Kelley & Kelley, 2007). However, recommendations of how to obtain such health benefits can be contentious (Balas-Nakash *et al*., 2010). Generally, many physical activity interventions show conflicting results with regards to reducing CVD (Balas-Nakash *et al*., 2010). Specifically, the effects of exercise on lipoproteins are contradictory in previous research (Isler *et al*., 2001). Nonetheless, Kelley and Kelley (2006) reported that commonly, aerobic exercise interventions provide an overall improvement in lipoprotein profiles. In particular, a development of HDL cholesterol and a significant reduction in LDL cholesterol are observed following aerobic exercise in most cases. Conversely, Kodama *et al*., (2007) claimed the results from many aerobic exercise interventions disagree. It is proposed that the effects of exercise are largely dependent on the specifics of the exercise program, including the duration, frequency and intensity (Kodama *et al*., 2007). Similarly, Kraus *et al*., (2002) suggested that there is evidence to imply that the amount of exercise is more important than the intensity for modifying lipoprotein metabolism.

Participant characteristics at baseline influence changes in lipoproteins following exercise (Kodama *et al*., 2007). For example, those with lower HDL levels initially have

the most to benefit from aerobic exercise (Trejo-Gutierrez & Fletcher, 2007). Likewise, the same relationship has been demonstrated for TG, whereby individuals with poorer TG levels at baseline show greater improvements in TG following exercise interventions than those with normal baseline values (Balas-Nakash *et al*., 2010). Furthermore, individuals with poor body composition show greater increases in HDL than those with more favourable body compositions at baseline (Kelley & Kelley 2007). Accordingly, it is thought that the effect of the exercise on lipids is most beneficial when accompanied by weight loss (Hardman & Stensel, 2003), although studies have still shown positive effects on lipoproteins with little weight loss (Kraus *et al*., 2002).

The most reliable findings have been for HDL (Hardman & Stensel, 2003), with even short periods of exercise showing modest improvements in HDL (Isler *et al*., 2001). Evidence suggests that individuals who are more physically active have higher levels of HDL cholesterol (Drygas *et al*., 2000; ACSM, 2009). Therefore, the benefit of physical activity on HDL is widely acknowledged (Kodama *et al*., 2007). Randomised trials have established a consistent increase in HDL subsequent to exercise interventions, yet the results are mainly dependant on the amount of exercise performed (Trejo-Gutierrez & Fletcher, 2007).

For example, Hardman and Hudson (1994) found a significant ($p \leq 0.05$) increase in HDL levels following 12 weeks of brisk walking in middle aged women. Likewise, Whitehurst and Mendenez (1991) established a significant increase in HDL in middle aged women, subsequent to eight weeks of walking. However, no significant decreases in TC were established by Whitehurst and Mendenez (1991). In contrast, Ferrauti *et al* (1997) found significant increases in TC and TG, but no significant changes in LDL or

HDL following six weeks of tennis training in middle aged men. Higuchi *et al*., (1984) engaged participants in treadmill running (estimated at 8 METs), five times per week for four weeks in total. Results indicated a significant (p <0.01) increase in HDL cholesterol following the four week intervention. HDL cholesterol increased by 22% (66 ± 8 mg/dL) following week two and by 35% (73 ± 11 mg/dL) at the end of week four. There were however, no significant changes in TC or plasma TG (Higuchi *et al*., 1984). Interestingly, the increase in HDL occurred without a significant change in mean body weight (Higuchi *et al*., 1984), supporting the proposal of Kraus *et al*., (2002).

Isler *et al* (2001) examined the effect of eight weeks of step aerobics and aerobic dancing on lipoproteins compared to a control condition. Following three, 45 minute sessions per week, it was concluded that HDL was significantly different amongst all three groups ($p \leq 0.05$), as was TC ($p \leq 0.01$) and the TC:HDL ratio ($p \leq 0.01$). There were no significant differences between the groups for either TG or LDL cholesterol. In particular, post hoc tests revealed that TC was significantly lower in the step aerobics group (126.73 ± 25.38 mg/dL) and aerobics dancing group (131.83 ± 22.68 mg/dL) compared to the control group (143.92 ± 34.33 mg/dL) following the exercise intervention. HDL was significantly higher in the step aerobics group (44.93 ± 8.32 mg/dL) compared to the control group (37.54 ± 7.01 mg/dL). Likewise, the TC: HDL ratio was significantly lower in the step aerobics group (2.86 ± 0.46) compared to the control group (3.86 ± 0.83). These findings suggest that step aerobics is an effective mode of exercise for modifying lipoprotein profiles (Isler *et al*., 2001).

Generally, previous research tends to indicate modest, although significant changes in HDL following exercise (Kodama *et al*., 2007). It is believed to be more beneficial to increase the time of an individual exercise session than to have numerous short sessions

in order to see elevations in HDL cholesterol (Kodama *et al*., 2007). Conversely, Lee *et al*., (2000) and the ACSM (2009) proposed that physical activity can be accumulated in smaller bouts lasting a minimum of 10 minutes in order to meet the minimum guidelines for reducing CVD risk; that being, moderate exercise, five times per week for 30 minutes (ACSM, 2009). Thompson and Rader (2001) remain sceptical; proposing that exercise training has little value for individuals with isolated low HDL levels. Additionally, the results of exercise training should be treated with caution as the increases in HDL are merely modest (Thompson & Rader, 2001).

With regards to LDL, Whitehurst and Mendenez, (1991) found no significant decreases in LDL following eight weeks of walking. However, Yoshida *et al*., (2010) established a significant (p <0.05) decrease in LDL following 16 weeks of supervised aerobic exercise, done 2-3 times per week for 60 minutes at 60-80% of maximal heart rate. Specifically, LDL reduced from 145 ± 75 mg/dL at baseline to 131 ± 21 mg/dL at eight weeks and 129 ± 21 mg/dL at 16 weeks. Furthermore, 24 weeks of endurance training has been shown to induce positive changes in lipoprotein profiles independent of changes in body weight or diet in 100 sedentary individuals aged 55-75 years (Halverstadt *et al*., 2007). LDL, TC and TG all decreased significantly (p =.001, p <.0001 and p <.0001 respectively) following endurance training, whereas HDL sub-fractions increased significantly (p =.01). Particle sizes also decreased in the study with significant decreases small and medium LDL particles (p =.01). Average HDL particle size increased (p =.04) following the exercise (Halverstadt *et al*., 2007).

Reductions in TG have been strongly established as a result of physical activity (Balas-Nakash *et al*., 2010). Single bouts of moderate intensity aerobic exercise have been shown to significantly (p =0.02) reduce postprandial TG levels, therefore improving

CVD risk (Ho *et al*., 2010). A recent intervention study by Balas-Nakash *et al*., (2010) investigated the effects of two different exercise groups on CVD markers in 319 Mexican children over the course of 12 weeks. Specifically, lipoproteins were measured pre and post intervention. One routine consisted of 40 minutes of aerobic exercises, whilst the other contained 20 minutes of lower intensity exercise. Hypertriglyceridemia decreased significantly ($p \leq 0.05$) in both groups, in addition to body weight and blood pressure, signifying that aerobic exercise is effective in reducing a number of CVD markers, including TG (Badlas-Nakash *et al*., 2010). Similar results have previously been established in middle aged men after four months of exercising at mild to moderate intensity, specifically a reduction in serum triglyceride (Huttunen *et al*., 1979). Some trials have even shown that as little as 20 minutes of exercise can reduce TG levels (Perichart-Perera 2008 cited in Badlas-Nakash *et al*., 2010).

It is proposed that exercise adjusts lipoprotein levels by influencing enzymes, which are involved in lipoprotein metabolism, including LPL (Hardman & Stensel, 2003). LPL is a hydrolytic enzyme, which limits the rate of the removal of triglycerides from the circulation (Eckel, 1989), resulting in either a reduction in the rate of triglycerides entering the circulation or an increase in triglyceride clearance, with LPL essentially acting as a "gate keeper" (Hardman & Stensel, 2003). This has been demonstrated by Sady *et al*., (1986) who measured fasting TG in 10 male distance runners following a marathon. Lipoprotein levels and lipolytic activity were measured pre and post marathon. LPL activity increased by 46% from 10.4 ± 2.82 mg/dL to 14.3 ± 1.63 mg/dL subsequent to the race, which resulted in TG levels decreasing by 26% from 80 ± 32.8 mg/dL to 56 ± 12.3 mg/dL (Sady *et al*., 1986). Additionally, HDL cholesterol is understood to be linked to an increase in the appearance of LPL, whilst exercise has been acknowledged to increase the activity of LPL (Thompson & Rader, 2001).

Therefore, if exercising increases the activity of LPL, then the number of HDL particles would be expected to alter also as they are positively related.

1.3. Videogames and physical inactivity

Despite overwhelming evidence suggesting that physical activity can improve lipoprotein profiles, physical inactivity is believed to be the greatest public health risk currently (Blair, 2009). Specifically, physical inactivity is believed to be a result of high levels of screen time, such as video gaming and television viewing (Daley, 2009). Such activities are thought to draw individuals away from physical activities that could be beneficial to their health (Daley, 2009). Additionally, increased sedentary activity of this kind may have negative effects on CVD risk factors (Pahkala *et al*., 2010).

Sedentary activities such as video gaming have become ever more popular amongst people of all ages (Siegel *et al*., 2009), with the demographic for "gamers" extending to both males and females in what was once labelled a male orientated hobby (Mintel, 2008). Specifically, the video game industry has witnessed huge developments over the past few years, with video game consoles increasing hugely in popularity (Mintel, 2008). It is reported that 67% of households in the US play video games, with the average gamer being 34 years old (ESA, 2010). Around 21% of these individuals play every day of the week (Lenhart *et al*., 2008 cited in Miyachi *et al*., 2010). Additionally, reports suggest that 26% of individuals 50 years and older play video games (ESA, 2010). British youth spend a considerable amount of time engaging in sedentary behaviours, such as video gaming (Biddle *et al*., 2003 cited in Graves *et al*., 2008). Specifically, it is thought that around three quarters of British youth play video games for two hours per day, three to seven times per week on average (Daley, 2009).

Sedentary activity of this kind is believed to be a contributing factor to obesity and CVD (Wack & Tantleff-Dunn, 2009).

1.4. Active Video Games

The introduction of active video games provides an opportunity to turn a sedentary past time into an active one (White *et al*., 2010). The Nintendo Wii has extended the video games market to individuals usually uninterested in game consoles, with reports suggesting that one in five owners of the Nintendo Wii had never owned a video game console previously (Jamieson, 2009). It has also been reported that the Nintendo Wii has sold over six million units in the UK alone, making it the fastest selling console of all time in the UK (Nintendo, 2009; Jamieson, 2009). Worldwide sales are said to be at 71 million according to the latest Nintendo financial reports (Gameindustry, 2010), highlighting the huge increase in popularity of such consoles (Mintel, 2008).

Sit *et al*., (2010) suggested that interactive games such as the Nintendo Wii may reduce the amount of time children spend in sedentary pursuits. Active videogames have the potential to increase physical activity levels among the sedentary in the comfort of the home (Graves *et al*., 2008; Lanningham-Foster *et al*., 2009), particularly as they are a convenient activity that could utilise existing enthusiasm to play video games (Daley, 2009). Likewise, Haskell *et al*., (2007) suggested that the accrual of moderate intensity physical activity should be done by providing a variety of activities that suit individual interests, timetables and environments. Consequently, active video games may present an opportunity to replace sedentary gaming, whilst still allowing children and adults' the videogame time that they desire (Barkley & Penko, 2009; Daley, 2009). However,

the efficacy of the Wii as a replacement for sedentary games is highly dependent on giving users a desire to play (Penko & Barkley, 2010).

It has been established that many individuals do not enjoy traditional physical activities (Leenders *et al*., 2003 cited in Sell *et al*., 2008). Thus, establishing innovative, enjoyable modes of exercise could provide a way in which young adults and children can overcome their lack of enthusiasm for exercise and achieve the recommended levels of physical activity (Sell *et al*., 2008). Furthermore, Graves *et al*., (2010) believes that inactive individuals with poor adherence to conventional exercise programmes may be more likely to maintain light to moderate active gaming if they can find enjoyment in the exercise. Moreover, Golan *et al*., (2006 cited in Lanningham-Foster *et al*., 2009) suggested that parents can provide positive behaviour modelling for their children by playing active games themselves. Accordingly, it is probable that active video games will be maintained when children play in the presence of others (Lanningham-Foster *et al*., 2009). Therefore, it could be deemed potentially beneficial to use such games, as increasing opportunities for physical activity is a significant solution for dealing with the prevalence of chronic diseases (Daley, 2009). Evidently, as the Nintendo Wii Fit Plus is now the first computer game to be endorsed by the Department of Health (Wallop, 2009).

It has been reported that individuals enjoy active video gaming equally as much as the sedentary options (Mark *et al*., 2009), if not more (Rhodes *et al*., 2008 cited in Mark *et al*., 2009). Specifically, Graves *et al*., (2010) found that enjoyment of Wii balance and aerobics on the Nintendo Wii Fit game was significantly ($p \leq 0.05$) greater than treadmill walking. Furthermore, it has been demonstrated that individuals show a greater

adherence to "active gaming" lab sessions than those using conventional exercise equipment (Warburton *et al*., 2007; Rhodes *et al*., 2008 cited in Mark & Rhodes, 2009).

1.5. Nintendo Wii

Early research on the Nintendo Wii focused primarily on the Wii sports software released with the console. Graves *et al*., (2007) compared energy expenditure (EE) and heart rate (HR) between active videogames on the Nintendo Wii and sedentary games on the Xbox 360. The HR and EE playing active games on the Wii was said to be significantly ($p \leq 0.001$) higher than that of the sedentary games on the Xbox 360. Further research by Graves *et al*., (2008) examined the intensity of Wii boxing with the focus on upper body movement and EE. EE was found to be even greater when upper body movement was measured, with HR and EE again being significantly ($p < 0.001$) greater than sedentary gaming (Graves *et al*., 2008). In particular, Wii boxing provided a higher EE and HR than that of Wii tennis and bowling. This was thought to be a result of the game requiring the use of both arms compared to sole use of the dominant hand in bowling and tennis (Graves *et al*., 2008). However, Wii Boxing only just exceeded (3.2 METS) the moderate intensity exercise category (ACSM, 2009) and as a result did not elicit an energy cost comparable to other active games such as dance simulations (Graves *et al*., 2008). Conversely, Bausch *et al*., (2008) examined the intensity of Wii sports games and found Wii boxing to provide a MET value of 5.2, and Wii tennis a value of 3.2 METs, both of which are considered moderate intensity activity by the ACSM (2009).

A plausible explanation for the low intensity in early Wii studies was thought to be the negligible use of large musculature whilst playing, as the emphasis seemed to be with

small muscle groups during Wii sports gaming (Graves *et al*., 2008). Alternatively, it is suggested that the methodology of the early studies was a weakness, and as a result EE may not have been accurately measured (Miyachi *et al*., 2010).

Since the first wave of Nintendo Wii research, numerous studies have examined the efficacy of the Wii as a mode of exercise, in addition to comparing it to conventional exercise techniques. Penko and Barkley (2010) compared treadmill walking to Wii boxing, establishing that Wii boxing provided a larger HR and VO_{2max} response when compared to treadmill walking in children. Specifically, the MET value of 3.3 obtained whilst playing Wii boxing was adequate enough to be considered moderate to vigorous physical activity for children, adolescents and adults (Penko & Barkley, 2010). Likewise, Willems and Bond (2009) found Wii Boxing to have similar physiological responses to brisk treadmill walking, including similar levels of fat and carbohydrate oxidation. Moreover, Graf *et al*., (2009) proposed that Wii boxing can provide a twofold increase in EE compared to watching television and is comparable to light treadmill walking. White *et al*., (2010) are in agreement, however they suggested that Wii boxing expends significantly ($p<0.001$) less energy than treadmill running.

As a result of these findings, it is suggested that the metabolic and physiological responses of Wii boxing may allow the game to be used as part of a structured exercise regime, which could potentially be used to obtain health benefits (Willems & Bond, 2009). However, only the boxing game provided such intensity and it is unlikely that both the baseball and tennis Wii sports games could provide any health benefits (Willems & Bond, 2009). Lanningham-Foster *et al*., (2009) established that a child playing a sedentary video game for eight hours per week would elicit a caloric burn of 652 calories. In comparison, an active Nintendo Wii game (Wii Boxing) would yield a

caloric burn of 1990 calories for the same duration. Moreover, adults could potentially burn 124 calories a day playing active games, which is comparable to walking 2mph on a treadmill for the same length of time (Lanningham-Foster *et al*., 2009).

Following on from Wii Sports, Nintendo released the Wii Fit game. The Wii Fit is a game specifically designed to engage people in healthy activities, whilst retaining a fun element (Nintendo, 2010). With regards to exercise intensity, the 68 Nintendo Wii Fit games provide an intensity ranging from 1.3 to 5.6 METS (Miyachi *et al*., 2010). Specifically, 46 of the game modes are classified as light intensity (<3METs), whilst 22 activities are moderate in intensity (3-6 METS) (Miyachi *et al*., 2010). Accordingly, it is believed that playing specific activities on the Nintendo Wii Fit could contribute to the daily recommended levels of physical activity (Miyachi *et al*., 2010), as outline by the ACSM (2009).

White *et al*., (2010) established that Wii Step was played at 40% $\dot{V}O_2$ peak and 52% HR peak in 26 male children. However, it is recommended that children should exercise at 80% $\dot{V}O_2$ peak in order to obtain changes in cardiovascular fitness (Baquet *et al*., 2003 cited in White *et al*., 2010). As a result, some Wii Fit games do not appear to elicit an intensity which could provide changes in cardiorespiratory fitness in children (White *et al*., 2010). Specifically, it is reported that Wii fit games do not elicit a HR or EE comparable to conventional exercise modes, such as treadmill walking, jogging (Graves *et al*., 2010) or running (Graf *et al*., 2009; White *et al*., 2010). This is thought to be a consequence of the intermittent nature of Wii games, and thus a reason for the differences between active gaming and regular exercise techniques (Graves *et al*., 2010). Nonetheless, Graves *et al*., (2010) proposed that light to moderate activities on

the Wii Fit may have the potential to reduce the risk of CVD in older adults based on activity recommendations for that age group.

However, it is believed that the intensity of games on the Wii Fit can be increased. More specifically, the Nintendo Wii "Free Step" mode has been shown to provide an intensity of 3.3 (Miyachi *et al.,* 2010) and 4 METS (Quinn, 2010) in two previous studies, which classifies as moderate intensity physical activity (ACSM, 2009). However, the addition of a 3rd party accessory, the "riiser" (ZooZen, 2010) provides the opportunity to augment the intensity further by raising the height of the Wii balance board by 3 inches. Specifically, Quinn (2010) established that raising the board 3 inches to a total height of 4 inches (ZooZen, 2010) elicited an intensity of 5 METS. Moreover, this can be increased further by adding a second "riiser", bringing the total height of the step up to 7 inches. Using the free step mode with this step height was shown to elicit an intensity of 6 METS (Quinn, 2010), which is vigorous physical activity based on the ACSM (2009) guidelines. This suggests that the "riisers" are an effective tool for increasing the intensity of the free step exercise.

Overall, it appears that integrating physical activity into videogames is a promising opportunity for exercise interventions based on the findings of previous research and the prior video game experience of many children, adolescents and adults (Mark *et al*., 2008). Additionally, physically active gaming has demonstrated positive physiological outcomes when compared to sedentary alternatives (Mark *et al*., 2008).

1.6. Rationale

The purpose of the current study was to investigate the effects of a four week Nintendo Wii exercise programme on plasma lipids using the "free step" mode on the Nintendo Wii Fit game. Specifically, to investigate any potential changes in levels of plasma HDL cholesterol, plasma LDL cholesterol, plasma TC and TG. Step aerobics was chosen as the mode of activity as it is one of the more popular forms of exercise due to the routines being easy for most individuals (Isler *et al*., 2001). Moreover, the ACSM (2009) recommends that exercise should be around 30 minutes in duration, using large muscle groups, with the exercise being rhythmic and aerobic in nature (ACSM, 2009).

The current study was undertaken as there are no randomised, controlled trials that have been published at present assessing the long term impact of active video gaming on health (Graves *et al*., 2010; Daley, 2009). Randomised, controlled trials are necessary in order to assess the effectiveness and sustainability of active video gaming, in addition to its clinical relevance (Daley, 2009). It was also of interest to examine the adherence to the exercise programme, as research in this area is limited (Mark & Rhodes, 2009). Currently, the majority of literature surrounding the Nintendo Wii has focussed on short term physiological outcomes associated with Wii games (Mark & Rhodes, 2009). There appears to be a dearth of evidence examining the effects of the Nintendo Wii at a biochemical level. Specifically, there appears to be no research at present examining the effects of active gaming on CVD risk markers as this study intends to. Furthermore, methodologies in current literature have only used a single bout of exercise on the Nintendo Wii (Mark & Rhodes, 2009). In contrast, this study will use a full training regime using the Nintendo Wii Fit, which does not seem to have been examined previously.

Research has shown that the Nintendo Wii is beneficial in terms of promoting physical activity, with positive outcomes such as increased EE (Graves *et al*., 2008; Willems & Bond, 2009; Graf *et al*., 2009; Lanningham-Foster *et al*., 2009). In particular, the Wii Fit has been shown to elicit moderate intensity physical activity (Miyachi *et al*., 2010) with the potential for this to be increased (Quinn, 2010). Additionally, Sugiura *et al*., (2002) suggested that increasing the number of daily steps by around 2000 in the form of moderate intensity exercise can improve or maintain healthy lipoprotein levels. Thus, given that Quinn (2010) found the Nintendo Wii Fit "free step" to provide 100 steps per minute, an average 20 to 30 minute session would yield 2000 to 3000 steps. Based on these findings, it is thought that using the Nintendo Wii Fit could elicit a moderate to vigorous intensity and potentially improve lipoprotein levels. Consequently, active gaming could provide positive health outcomes such as a reduced risk of CVD, as is common with this level of physical activity (ACSM, 2009).

As a result of previous research (Isler *et al*., 2001; Higuchi *et al*., 1984; Kelley & Kelley, 2006) it is hypothesised that the four week Nintendo Wii exercise programme will result in a significant increase in HDL cholesterol. Additionally, it is hypothesised that TC and TG will decrease significantly subsequent to the exercise intervention, based on previous findings (Kelley & Kelley, 2006; Balas-Nakash *et al*., 2010). However, it is expected that LDL levels will not change significantly. Specifically it is thought that the intensity and duration of the exercise in the current study may be too low to induce a reduction in LDL based on the findings of previous research (Isler *et al*., 2001; Higuchi *et al*., 1984, Kelley & Kelley, 2006, Yoshida *et al*., 2010). Due to the anticipated increase in HDL, it expected that the LDL: HDL ratio will also decrease significantly.

2. METHOD

2.1. Participants

Participants were recruited from May to June 2010. The sample size used for the study was 15. The participants were split into two groups determined by their predicted $\dot{V}O_{2max,}$ with a minimum of six in each group (Table 1). This number was deemed appropriate based on a power calculation (Schoenfeld, 2010) (Appendix 1). This was a combination of students and staff members (3 males and 12 females) from the University of Central Lancashire (UCLan). The sample was reduced from an original number of 17 due to incomplete data for two of the participants. Participants were recruited via email, poster (Appendix 2) and word of mouth. All participants provided informed consent (Appendix 3) and read a participant information sheet (Appendix 4). A risk assessment was completed assessing the suitability of the studies activities (Appendix 5). Participant eligibility was determined via a Physical Activity Readiness Questionnaire (CSEP, 2002) (Appendix 6). Participants were excluded from the study if resting systolic blood pressure (SBP) exceeded 200 mmHg or resting diastolic blood pressure (DBP) exceeded 115 mmHg as per ACSM (2009) guidelines. The study was approved by the School of Psychology Ethics Committee at UCLan (Appendix 7).

Table 1. Participant (n=15) characteristics by predicted $\dot{V}O_{2max}$ as per ACSM (2009) guidelines, calculated using Adams (2000) equation.

	≤ Fair $\dot{V}O_{2max}$ (n=7)		≥ Good $\dot{V}O_{2max}$ (n=8)	
	Mean	±SD	Mean	±SD
Age (years)	40	13	34	14
Stature (cm)	166	6	163	5
Mass (kg)	74[a]	8	64[a]	5
Systolic Blood Pressure (mmHg)	125	12	119	11
Diastolic Blood Pressure (mmHg)	84	9	77	10
Body Fat (%)	35	13	28	13
Predicted $\dot{V}O_{2max}$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	31[b]	5	46[b]	6

[a] significantly ($p \leq 0.05$) different (Independent t-test) (Appendix8)
[b] significantly ($p \leq 0.001$) different (Independent t-test) (Appendix 8)

2.2. Research Design

The study used a cross over design. Specifically, each participant took part in both the experimental (Nintendo® Wii™ step aerobics) and control (no exercise) condition, therefore acting as their own control. The order in which the participants completed the two conditions was randomised. Participants were randomised into groups based on their availabilty. A two week washout period was imposed between the two conditions in order for the effects of the previous condition to subside.

2.3. Procedures

2.3.1. Health Screen

During the initial testing session, all participants completed a pre exercise health screen, consisting of blood pressure measurement, body composition analysis and a predictive VO_{2max} test. Blood pressure was measured using an automatic blood pressure monitor (Boso-Medicus, Germany) to determine resting systolic and diastolic blood pressure (mmHg). Participants were seated in a chair for five minutes prior to measurement. Two meausurements were then taken for an average to be obtained, as per ACSM (2009) guidelines.

Body composition was assessed using air displacement plethysmography (BodPod) (Life Measurement, Inc. USA). Participants' were instructed to wear either undergarments or tight fitting clothing. Additionally, a provided swim cap was also worn to cover the hair and ensure test accuracy. Participants were weighed on the BodPod's incorporated scales to determine body mass. Next, they entered the BodPod and sat with their hands on their lap whilst three, 40-second tests were performed.

Measures of body fat (%) and fat free mass (%) were determined via the BodPod's onboard computer.

The Åstrand-Rhyming (1954 cited in ACSM, 2009) cycle ergometer test was used to predict participants' $\dot{V}O_{2max}$. Participants were seated on a Monark 834E ergometer (Monark, Sweden) with the ball of the foot on the pedals. To ensure the participant was in an appropriate position, the seat height was adjusted so that the legs had a bend of no more than 5° at the bottom of the rotation (Adams, 2000). The workload for each participant was based on their habitual physical activity levels, as advised by the ACSM (2009). These were determined by an International Physical Activity Questionnaire (IPAQ, 2002) (Appendix 9).

To start the protocol, participants began cycling and maintained a pedalling cadence of around 50rpm. The desired power level was then set by adding specific weights onto the ergometer force scale. Participants cycled for 6 minutes and two measures of heart rate ($b \cdot min^{-1}$) were taken during the 5th and 6th minute using an Onyx® 9500 pulse-oximeter (Nonin Medical, Inc, Plymouth). The two recorded values were then averaged, and used to calculate the participants' estimated $\dot{V}O_{2max}$ using a modified Åstrand-Rhyming nonogram (Åstrand-Rhyming, 1954 cited in ACSM, 2009).

2.3.2. Blood Sampling

Following the health screen, a 2mL venous blood sample was taken by a trained phlebotomist. Blood was collected into a BD vacutainer® (BD, England) or s-monovette® (Sarstedt, Leicester) containing EDTA anti-coagulant. Whole blood was then pipetted into 2mL safe-seal microtubes (Sarstedt, Leicester), using a Pipetman®P

(Gilson, USA). The samples where then centrifuged in a MSE Micro Centaur (Sanyo, Japan) at 10,000 rpm for 15 minutes, allowing the plasma to be seperated from the whole blood. Subsequently, the plasma was transferred into safe-seal microtubes via the Pipetman®P and stored in a -25°C freezer (Lec, UK) in preparation for lipid analysis.

2.3.3. Blood Plasma Analysis

The plasma samples were measured by spectrophotometric analysis using a Dr Lange MiniPhotometer P20 (Hach Lange, Berlin). The cholesterol and triglyceride assays used had an inter-assay covariance of 4% and 8% respectively. The stored samples were thawed and brought to room temperature. Initially, a blank reagent (Appendix 10) was inserted into the Dr Lange to calibrate the miniphotometer and measure the absorbancy, which should read zero. For TC and TG analysis, 10µl of the plasma sample was pipetted into a cuvette containing the appropriate reagent and buffer solution (Appendix 10). The plasma sample was then mixed into the reagent by inverting the curvettes three times. When all samples had been mixed appropriately, the correct measurement parameter was selected on the Dr Lange using the electronic onscreen menu. The curvettes were then placed into the miniphotometer individually and a measure of TC and TG was given after approximately three minutes.

For HDL analysis, 200µl of plasma sample was pippetted into 2mL safe-seal micro tubes along with 20µl of a HDL precipitant (Appendix 10). The mixture was left to stand at room temperature for 10 minutes before being centrifuged at 3,000rpm for 10 minutes using a MSE Micro Centaur (Sanyo, Japan). Subsequently, 50µl of the clear supernatant that remained following centrifugation, was pippeted into a curvette

containing the appropriate reagent and buffer (Appendix 10). LDL analysis was completed as per the instructions for HDL, instead using a specific LDL precipitant (Appendix 10) and centrifuging at 10,000rpm for two minutes. When all samples had been mixed, the correct measurement parameter was selected, and the curvettes were placed into the miniphotometer individually. A measure of HDL and LDL was obtained after three minutes.

2.3.4. Nintendo Wii Exercise Programme

The Nintendo Wii Console (Nintendo®, Japan) was used for the exercise protocol. The game used was the free step mode on the Nintendo Wii Fit Plus game (Nintendo®, Japan). For the purpose of this study, the Wii Fit balance board was raised six inches (Figure 1) using two Wii riisers (Zoo Zen Ltd, Hong Kong), consequently increasing the exercise intensity (Quinn, 2010). Participants attended the UCLan laboratory in order to undertake the exercise programme. This allowed the participants to be observed whilst exercising and ensured that all participants were given an identical exercise dose. Specifically, participants were asked to perform 30 minutes of moderate to vigourous intensity step aerobics, maintaining the pace set by the game. Using the in-game speed controls, the first and final five minutes of the programme were performed at a slower pace than the central 20 minutes, to act as a warm-up and cool-down respectively. Participants attended three sessions per week for the four week duration of the exercise protocol. Those that did not obtain a minimum attendance record of 80% (Appendix 11) were excluded from data analysis.

Following completion of the exercise programme, participants' attended a post exercise health screen and gave a second blood sample. Subsequently, they completed a two week wash-out period. At this point, repeated meausures of blood, blood pressure, body compostion and predicted $\dot{V}O_{2max}$ were taken. Thereafter, a four week control period with no exercise was undertaken. Final measurements of blood, blood pressure, body compostion and $\dot{V}O_{2max}$ were taken at the end of this phase (post control). For those that were randomly assigned to the control group first, the order of conditions described were reversed.

2.3.5. MetaLyzer® 3B

During the first and last Nintendo Wii exercise sessions, the MetaLyzer® 3B (Cortex, Biophysik, Germany), was used to obtain measures of respiratory gases. This was done to assess any changes in cardiorespiratory fitness following the Nintendo Wii programme. The MetaLyzer was calibrated by inputting the pressure reading from the labratory barometer into the Metasoft® Software. Next, the pneumotach (volume) was calibrated using a Hans Rudolph three-litre calibration syringe (Hans Rudolph, Inc, USA). Lastly, the gas sensors were calibrated using the ambient air, followed by a known concentration (5.09% CO_2 and 14.46% O_2) of gas (Boc Limited, Germany). Upon testing, participants were fitted with a heart rate belt (Polar, Finland). A Hans Rudolph mask (Hans Rudolph, Inc, USA) was held around the face using a mask harness. Attatched to this was a volume sensor. This was connected to the MetaLyzer via a sample line, which allowed respiratory gases to be detected (Figure 1). From this data, EE was calculated using McArdle *et al*., (1996 cited in White *et al*, 2010) equation.

Figure 1. Participant playing Nintendo Wii Fit with raised Wii Balance board. MetaLyzer® 3B in operation.

2.4. Analysis

Data was prestented as means and standard deviations. A Kolmogrov-Smirnov test was completed to ensure the data was normally distributed. Paired samples t-tests were performed (Appendix 12) to compare pre and post values for both conditions. As only two groups were used in the study, t-tests would of been required following an ANOVA. Therefore, it was considered suitable to use a priori t-tests, without the need for an ANOVA. Additionally, further t-tests were used to compare post washout and baseline values (Appendix 13). In the event of significant difference for both fitness groups in blood lipids, an independent samples t-test (Appendix 14) was performed to compare the level of change between the groups. The statistical package used was PASW (Version 18) (SPSS, UK). Effect size was also calculated (Field, 2009). The alpha level was set to $p \leq 0.05$. For convienience purposes, the two fitness groups "Fair and Below" and "Good and Above" will be referred to as low and high fitness respectively.

3. RESULTS

The engagement of the participants throughout the study is shown in Figure 2.

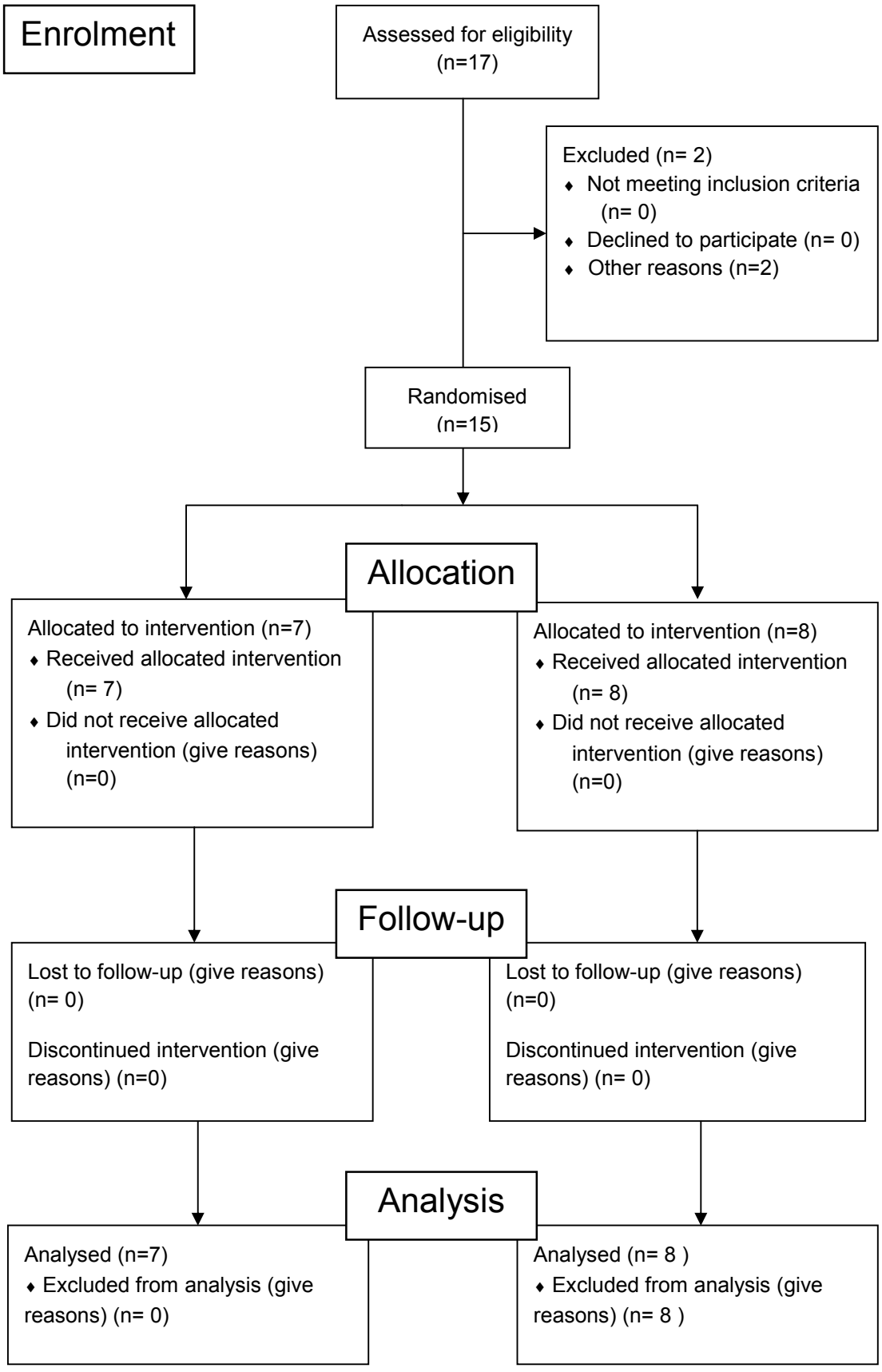
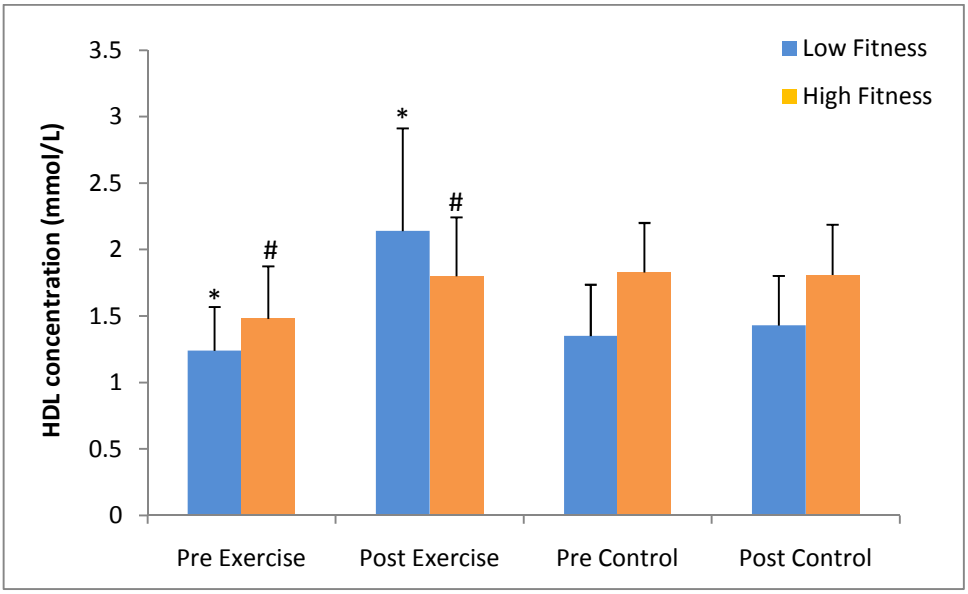


Figure 2. The CONSORT statement flow diagram (Moher *et al.*, 2001)

3.1. High density lipoproteins

HDL concentrations significantly increased in the exercise condition, in both the low fitness ($t_{(6)} = -4.239$ $p \leq 0.01$, d= 1.6) and high fitness ($t_{(7)} = -3.728$ $p \leq 0.01$, d= 1.3) groups (Figure 3). There were no significant differences in HDL in the control condition, in either the low or high fitness group ($t_{(6)} = -1.924$ $p > 0.05$ d= 0.7 and $t_{(7)} = .422$ $p > 0.05$, d= 0.1 respectively). The low fitness group did show a 7% increase. However, the high fitness group displayed a 1% reduction in HDL in the control condition.

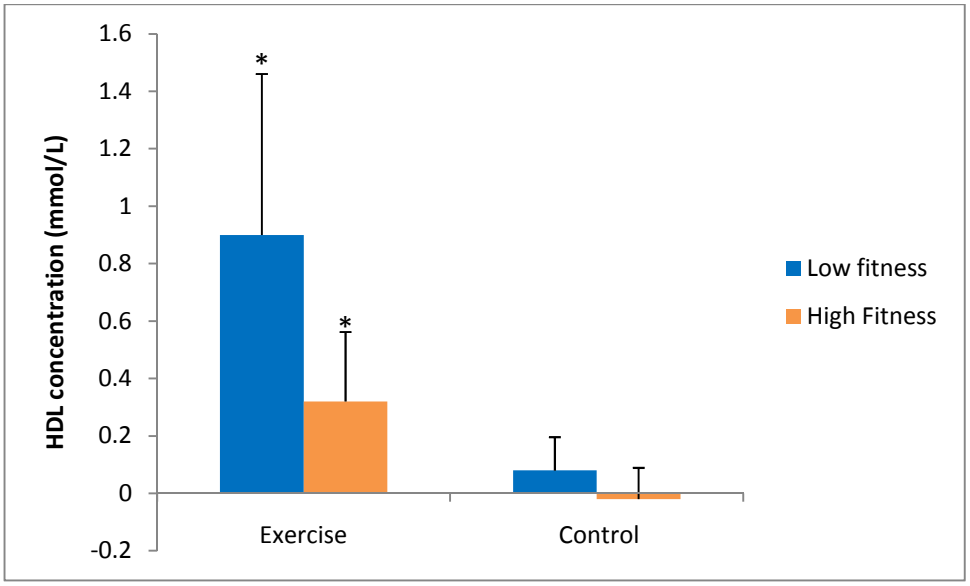


*, # significantly different ($p \leq 0.05$)

Figure 3. HDL concentrations (means ± SDs) for the low (n=7) and high (n=8) fitness groups in the exercise and control conditions.

It was also established that the low fitness group showed a significantly higher ($t_{(13)} = 2.666$ $p \leq 0.05$, d= 1.6) increase in HDL then the high fitness group in the exercise condition (Figure 4). Specifically, it was established that the low fitness group increased

their HDL concentration by 73% (0.9 mmol/L), whilst the high group increased by 22% (0.32 mmol/L).



*Significantly different ($p \leq 0.05$)

Figure 4. Change in HDL concentration between the low (n=7) and high (n=8) fitness groups in the exercise and control conditions.

3.2. Low density lipoproteins

In the exercise condition, both the low ($t_{(6)} = .532$ $p > 0.05$, d= 0.2) and high fitness ($t_{(7)} = -.049$ $p > 0.05$, d= 0.1) groups showed a non-significant difference in LDL concentrations (Figure 5). Although non-significant, there was a reduction in LDL of 3% in the low fitness group, whilst the high fitness group showed no change. In the control condition, the low ($t_{(6)} = -.884$ $p > 0.05$, d= 0.3) and high fitness ($t_{(7)} = -1.425$ $p > 0.05$, d= 0.5) groups showed no significant differences in LDL concentrations. In particular, there was an increase in LDL for both the low (7%) and high fitness group (13%) (Figure 5).

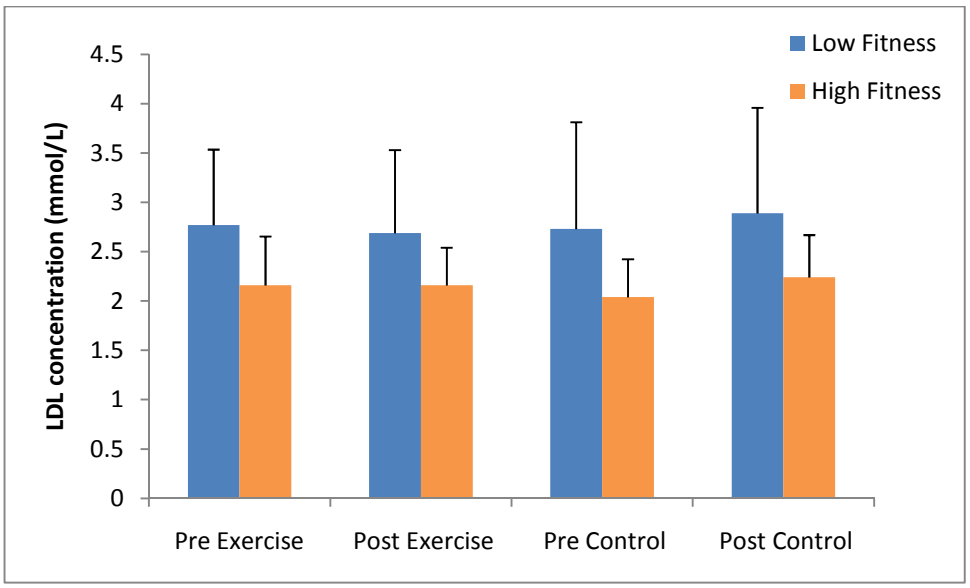


Figure 5. LDL concentrations (means ± SDs) for the low (n=7) and high (n=8) fitness groups in the exercise and control conditions.

3.3. Total cholesterol

For TC, the low fitness group showed no significant difference in the exercise condition ($t_{(6)} = 2.436$ $p = 0.051$, d= 0.9) (Figure 6), although this was approaching significance. Specifically, a 15% reduction in TC was apparent. The high fitness group displayed a non-significant 1% rise in TC in the exercise condition ($t_{(7)} = -.225$ $p > 0.05$, d= 0.1). The low fitness group showed a non-significant increase (5%) in TC in the control condition ($t_{(6)} = -2.284$ $p > 0.05$, d= 0.9). Likewise, the high fitness group demonstrated a non-significant rise (2%) in TC in the control condition ($t_{(7)} = -.233$ $p \geq 0.05$, d= 0.1) (Figure 6).

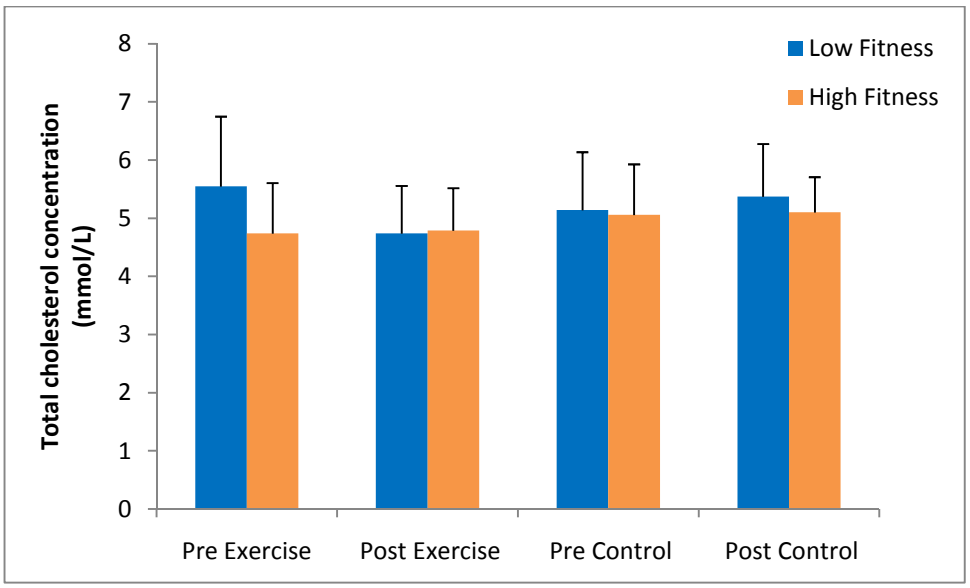


Figure 6. TC concentrations (means ± SDs) for the low (n=7) and high (n=8) fitness groups in the exercise and control conditions.

3.4. Triglycerides

There were no significant differences in TG levels in the low ($t_{(6)} = -.696$ $p > 0.05$, d= 0.3) or high fitness group ($t_{(7)} = -.370$ $p > 0.05$, d= 0.1) for the exercise condition. In particular, the low fitness group showed a non-significant 11% reduction in TG, whilst the high fitness group showed a 5% decrease (Figure 7). In the control condition, no significant differences in TG were seen in the low fitness ($t_{(6)} = -1.683$ $p > 0.05$, d= 0.6) or high fitness group ($t_{(7)} = -.577$ $p > 0.05$, d= 0.2), although both groups showed an increase; 9% and 5% respectively (Figure 7).

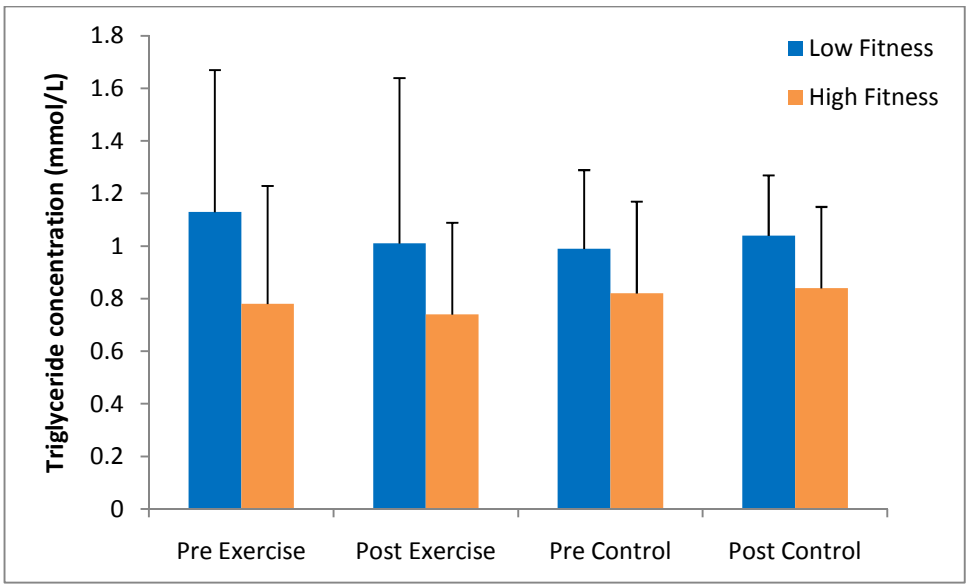


Figure 7. TG concentrations (means ± SDs) for the low (n=7) and high (n=8) fitness groups in the exercise and control conditions.

3.5. LDL: HDL ratio

The LDL: HDL ratio decreased significantly for both the low and high fitness groups following the exercise intervention ($t_{(7)} = 3.489$ $p \leq 0.05$, d= 1.3 and $t_{(7)} = 2.386$ $p \leq 0.05$, d= 0.8 respectively). In the control condition, there were no significant differences in the low fitness ($t_{(7)} = .013$ $p > 0.05$, d= 0.0) or high fitness ($t_{(7)} = 2.124$ $p > 0.05$, d= 0.8) group (Table 2).

Table 2. LDL: HDL ratio means (±SD) for the low (n=7) and high (n=8) fitness groups.

	Low Fitness (n=7)		High Fitness (n=8)	
	Pre	Post	Pre	Post
Exercise	2.2 ± 0.8*	1.3 ± 0.8*	1.5 ± 0.5#	1.2 ± 0.3#
Control	2.0 ± 0.9	2.0 ± 0.8	1.1 ± 0.2	1.2 ± 0.2

*, # significantly different ($p \leq 0.05$)

3.6. Health Screen

For the low fit group, there was a significant decrease in resting SBP following the exercise intervention ($t_{(6)} = -2.659$ $p \leq 0.05$, d= 1.0). Additionally, the low fit group showed a significant increase in estimated $\dot{V}O_{2max}$ following the intervention ($t_{(6)} = -2.448$ $p \leq 0.05$, d= 0.9). All other variables measured as part of the health screen showed no significant differences in either the exercise or control condition (Table 3).

Table 3. Systolic blood pressure (SBP), diastolic blood pressure (DBP), fat percentage, fat free percentage, total mass, estimated $\dot{V}O_{2max}$ and international physical activity questionnaire (IPAQ) data for the low fitness group (n=7) (Means ± SDs).

	Exercise		Control	
	Pre	Post	Pre	Post
SBP (mmHg)	125 ± 12[a]	119 ± 14[a]	125 ± 12	126 ± 11
DBP (mmHg)	85 ± 9	85 ± 11	86 ± 11	87 ± 5
Fat (%)	35 ± 13	33 ± 12	31 ± 13	32 ± 14
Fat Free (%)	65 ± 13	67 ± 12	69 ± 13	63 ± 17
Mass (kg)	74 ± 8	74 ± 9	72 ± 9	73 ± 9
Estimated $\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	29 ± 5[b]	35 ± 9[b]	43 ± 19	36 ± 8
IPAQ	1 ± .49	2 ± .82	2 ± .82	2 ± .69

For IPAQ, 1=Low, 2=Moderate, 3=High level of habitual physical activity
[a,b] significantly different ($p \leq 0.05$)

The high fitness group also displayed a significant decrease in resting SBP following the exercise condition ($t_{(7)} = 2.681$ $p \leq 0.05$, d= 0.9), as well as a decrease in the control condition ($t_{(7)} = 2.521$ $p \leq 0.05$, d= 0.9). They also showed a significant decrease in resting DBP in the control condition ($t_{(7)} = 2.785$ $p \leq 0.05$, 1.0). Moreover, estimated $\dot{V}O_{2max}$ increased significantly following the exercise intervention ($t_{(7)} = -2.549$ $p \leq 0.05$, d= 0.9). All other measures were non-significant in both the exercise and control group (Table 4).

Table 4. Systolic blood pressure (SBP), diastolic blood pressure (DBP), fat percentage, fat free percentage, total mass, estimated $\dot{V}O_{2max}$ and international physical activity questionnaire (IPAQ) data for the high fitness group (n=8) (Means ± SDs).

	Exercise		Control	
	Pre	Post	Pre	Post
SBP (mmHg)	119 ± 11[a]	109 ± 7[a]	119 ± 8[b]	112 ± 8[b]
DBP (mmHg)	77 ± 10	72 ± 5	82 ± 4[c]	74 ± 9[c]
Fat (%)	28 ± 13	28 ± 11	27 ± 12	27 ± 12
Fat Free (%)	72 ± 13	72 ± 11	73 ± 12	73 ± 12
Mass (kg)	65 ± 6	65 ± 7	65 ± 7	65 ± 7
Estimated $\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	43 ± 6[d]	57 ± 15[d]	50 ± 9	56 ± 12
IPAQ	3 ± .53	2 ± .52	2 ± .35	2 ± .89

For IPAQ, 1=Low, 2=Moderate, 3=High level of habitual physical activity
[a,b,c,d] significantly different ($p \leq 0.05$)

3.7. Cardiorespiratory Responses to Exercise

The low fitness group showed a significant decrease in exercising METs ($t_{(6)} = 3.972$ $p \leq 0.05$, d= 1.5), relative $\dot{V}O_{2max}$ ($t_{(6)} = 3.927$ $p \leq 0.05$, d= 1.5) and EE ($t_{(6)} = 4.058$ $p \leq 0.05$, d= 1.5) in the exercise condition (Table 5). HR did not differ significantly in the exercise condition, although it was approaching significance ($t_{(6)} = 2.389$ p =0.054, d= 0.9). These same measures in the rest condition were all non-significant

Table 5. Metabolic equivalents (METs), heart rate (HR), relative $\dot{V}O_2$ and energy expenditure (EE) for the low fitness group (n=7) (Means ± SDs)

	Rest		Exercise	
	Pre	Post	Pre	Post
METs	0.8 ± 0.4	1.0 ± 1.0	4.7 ± 0.6[a]	3.7 ± 0.4[a]
HR ($b \cdot min^{-1}$)	71 ± 5	68 ± 7	125 ± 12	116 ± 17
Relative $\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	3 ± 2	2.2 ± 0.90	16 ± 2[b]	13 ± 1[b]
EE ($J \cdot kg^{-1} \cdot min^{-1}$)	61 ± 32	49 ± 15	334 ± 39[c]	234 ± 19[c]

[a,b,c,] significantly different ($p \leq 0.05$)

In the high fitness group, there were no significant differences in any of the measures for either the rest or exercise condition (Table 6).

Table 6. Metabolic equivalents (METs), heart rate (HR), relative $\dot{V}o_2$ and energy expenditure (EE) for the high fitness group (n=8) (Means ± SDs)

	Rest		Exercise	
	Pre	Post	Pre	Post
METs	1.3 ± 0.7	0.9 ± 0.2	5.0 ± 1.1	4.3 ± 1.0
HR ($b \cdot min^{-1}$)	76 ± 12	71 ± 5	117 ± 16	110 ± 9
Relative $\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	4 ± 3	3 ± 1	18 ± 4	15 ± 4
EE ($J \cdot kg^{-1} \cdot min^{-1}$)	93 ± 54	66 ± 34	360 ± 77	316 ± 75

3.8. Washout period effectiveness

The values for estimated $\dot{V}O_{2max}$ were significantly ($p \leq 0.05$) different at baseline and post washout (pre control condition) in the group of participants that were randomised into the exercise condition first. The other parameters measured were all non significant (Table 7), demonstrating that the washout period was a suitable duration to allow the effects of the exercise intervention to subside before participants started the control condition, excluding $\dot{V}O_{2max}$.

Table 7. Paired samples t-tests comparing baseline and pre control data for participants in randomised groups.

	Started with exercise intervention	Started with control condition
TC (mmol/L)	t (6) = .53; p = .62	t (7) = -.28; p = .79
TG (mmol/L)	t (6) = -.28; p = .79	t (7) = .49; p = .64
HDL (mmol/L)	t (6) = -1.13; p = .30	t (7) = -1.75; p = .12
LDL (mmol/L)	t (6) = - 1.48; p = .19	t (7) = 2.13; p = .07
Estimated $\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	t (5) = -3.38; $p \leq .05$[a]	t (7) = -.95; p = .37
SBP (mmHg)	t (6) = 1.19; p = .28	t (7) = -.49 ; p = .64
DBP (mmHg)	t (6) = .99; p = .36	t (7) = -1.28 ; p = .24
Fat (%)	t (5) = 1.03; p = .35	t (5) = .42; p = .69
Fat Free Mass (%)	t (5) = -1.03; p = .35	t (5) = -.39; p = .71
Mass (kg)	t (5) = .84; p = .43	t (5) = 1.81; p = .13
IPAQ	t (5) = .00 ; p = 1.00	t (7) = 1.00; p = .35

[a]Significantly different ($p \leq 0.05$) between baseline and pre control (post washout) measures signifying ineffective washout period.

4. DISCUSSION

The purpose of the current study was to investigate the effects of a four week Nintendo Wii exercise programme on blood plasma lipids using the "free step" mode on the Nintendo Wii Fit game. Specifically, to investigate any potential changes in levels of plasma HDL cholesterol, plasma LDL cholesterol, total plasma cholesterol and triglycerides. A secondary outcome was to investigate the effects the programme would have on basic health parameters, such as blood pressure, estimated $\dot{V}O_{2max}$ and body composition. Additionally, cardiorespiratory responses to exercise were observed (METs, HR, EE and relative $\dot{V}O_2$). The hypothesis for HDL, LDL and the LDL: HDL ratio were accepted. The hypothesis for TC and TG was rejected.

4.1. High density lipoproteins

Prior research has suggested that small amounts of exercise can induce changes in HDL (Isler *et al*., 2001), whilst step aerobics interventions have previously established a significant increase in HDL (Isler *et al*., 2001). Thus, it was unsurprising that the Nintendo Wii exercise intervention provided a significant increase in HDL in both fitness groups. These results do however, disagree with Trejo-Gutierrez and Fletcher (2007) who proposed that high volume and high intensity exercise is needed to achieve significant increases in HDL. In contrast, the results support Durstine *et al*., (2002) suggestions that low training volumes are beneficial and can positively alter blood lipids. However, certain exercise thresholds must still be met to witness noticeable changes (Durstine *et al*., 2002).

The results also concur with Higuchi *et al* (1984), who also established significant increases in HDL following a four week exercise intervention. Interestingly, the participants in Higuchi *et al* (1984) study worked at 8 METs during their exercise

intervention yet showed a smaller increase in HDL than the low fitness group in the current study, despite them working at a lower average of 4.4 METs. Specifically, an increase of 0.9 mmol/L was established in the present study in the low fitness group, compared to a 0.5 mmol/L increase in Higuchi *et al* (1984) study. However, the participants in Higuchi *et al* (1984) study had higher HDL baseline values than the low fit participants in this study. Furthermore, the low fitness group in the present study showed a significantly higher increase in HDL than the high fitness group. Accordingly, these findings support the research of Kodama *et al*., (2007) and Kelley and Kelley (2006), in that individuals with low baseline HDL levels benefit more from aerobic exercise than those with higher HDL levels at baseline (Kodama *et al*., 2007). Additionally, it is acknowledged that individuals with inferior body composition show greater increases in HDL than those with optimal body composition (Trejo-Gutierrez & Fletcher, 2007). This was witnessed in the current study as the low fitness group had a poorer body composition at baseline than the high fitness group and perhaps saw a greater increase in HDL as a result. It is however, acknowledged that there is much variance in the way specific individuals respond to exercise in terms of altering lipids (Trejo-Gutierrez & Fletcher, 2007).

Specifically, the participants showed a 73% (low fitness) and 22% (high fitness) increase in HDL following the exercise intervention. It has been reported previously that a 3% increase in HDL may relate to a 6-9% decrease in CHD risk (Kelley & Kelley 2006). These results do however, appear somewhat exaggerated based on the increases in HDL found in previous studies (Kelley & Kelley, 2006; 2007; 2008). Nonetheless, it has been established that playing Wii step aerobics has the potential to improve HDL-C levels. Moreover, the control condition in the study established no significant differences in HDL in either fitness group. The good fitness group actually showed a

decrease in HDL of 1%, which is important given that modest decreases of this size can bring about a 2-3% increase in CHD risk (Gorden *et al*., 1989). These findings further reinforce the potential effectiveness of the Wii as a tool for improving specific CVD risk markers. Although it is still uncertain whether an increase in HDL alone can reduce CVD risk (ACSM, 2009).

4.2. Low density lipoproteins

In agreement with Isler *et al*., (2001), there were no significant differences found for LDL following the step aerobics exercise intervention in either fitness group, confirming the original hypothesis. Durstine *et al*., (2002) believes that exercise interventions rarely change LDL levels unless a reduction in dietary fat intake and body weight accompanies the intervention. Given that no dietary intervention was offered in the present study, and no significant reductions in fat percentage or total mass were observed, it is plausible that this may be a reason for the non significant reductions in LDL. Additionally, previous literature (Yoshida *et al*., 2010; Halverstadt *et al*., 2007) that has established a significant decrease in LDL has generally been for longer durations (12 and 24 weeks respectively) than the four weeks provided in this study. Moreover, the exercise in Halverstadt *et al*., (2007) study was endurance training of higher intensity than Wii step aerobics, and the length of each session in Yoshida *et al*., (2010) was 60 minutes compared to 30minutes in this study. The length of each session is thought to be an important factor, as Kraus *et al*., (2002) suggested that the amount of exercise may be more important than the intensity for modifying lipoproteins.

Nonetheless, there was a reduction in LDL of 3% in the low fitness group, which may have some practical significance. Specifically, this reduction would relate to a 6% reduction in coronary episode risk based on the findings of Pedersen *et al.,* (1998 cited

in Kelly & Kelly, 2006) who suggested a reduction in coronary episodes of 2% with every 1% reduction in LDL. Moreover, there were no significant differences in the control condition for both groups, but interestingly an increase in LDL occurred in both groups. Consequently, despite non-significant results in either group, it could be suggested that regular exercise on Wii step aerobics can at least maintain LDL levels, if not lower them slightly.

4.3. Total cholesterol

TC did not differ significantly in either fitness group following the exercise intervention, rejecting the original hypothesis. However, the 15% decrease in TC was approaching significance (p =0.051) in the low fit group following the exercise condition. These results disagree with those found by Isler *et al*., (2001), which is likely due to the shorter training period (Kraus *et al*., 2002) used in the present study compared to that of Isler *et al*., (2001) study.

The results do however, confirm the findings of Durstine *et al*., (2002), who suggested that other interventions (dietary and weight loss) are needed to see significant decreases in TC. Higuchi *et al*., (1984) also found no significant changes in TC following a four week exercise intervention. Much like Higuchi *et al*., (1984), it is believed that a significant reduction in TC would have been established had there been a significant decrease in body fat in the present study. A reduction in body fat and an increase in lean mass have been associated with a reduction in TC (Trejo-Gutierrez & Fletcher, 2007). In the present study, the low fit group saw a decrease in body fat and an increase in lean mass, albeit non-significantly, which is a likely explanation for the almost significant reduction in TC. The mean TC of the low fit group reduced from 5.55 ± 1.20 mmol/L

pre exercise intervention to 4.74 ± 0.82 mmol/L post intervention. This post exercise mean reduced the low fit groups TC below the maximum desirable range of 5.1mmol/l (ACSM, 2009). Additionally, it takes the low fit group out of the range (5.17-6.47 mmol/L) that is linked to a 40% increase in myocardial infarction risk (Ho *et al,* 1993). Consequently, there appears to be a practical significance of exercising using Wii aerobics for individuals in the low fitness group. This is corroborated further by the fact that both fitness groups had a non-significant increase in TC in the control condition (5% and 2% respectively).

4.4. Triglycerides

Neither fitness group showed any significant changes in TG following the Wii aerobics intervention. These results conflict those of Balas-Nakash *et al*., (2010), Huttunen *et al*., (1979) and Perichart-Perera, 2008 cited in Balas-Nakash *et al*., (2010) all of whom found significant decreases in TG following an exercise intervention. The length of each session in the current study seemed appropriate based on Balas-Nakash *et al*., (2010) significant findings. However, the duration of the interventions in the former studies were four times the length of the present study and thus may explain why no significant changes were found. Nevertheless, both groups showed a non-significant decrease in TG. Specifically, the low fit group decreased from 1.13 ± 0.54 mmol/L to 1.01 ± 0.63 mmol/L, while the high fitness group decreased from 0.78 ± 0.45 mmol/L to 0.74 ± 0.35 mmol/L. This represents an 11% and 5% decrease respectively. Although non-significant, a reduction in serum triglycerides represents a reduction in a CVD risk factor (ACSM, 2009). Once more, both groups showed an increase in TG in the control condition. The low fit group showed a 9% increase in TG, whereas the good fitness group showed an increase of 5%. These results suggest that Wii aerobics can maintain TG, when used as part of a structured exercise intervention.

4.5. LDL/HDL Ratio

Unsurprisingly, as a result of the changes in HDL and LDL the LDL: HDL ratio significantly reduced in both fitness groups as a result of the Wii aerobics training programme. In the low fit group the LDL: HDL ratio reduced from 2.2 pre to 1.2 post intervention. The high fit group saw a decrease from 1.5 to 1.2. Although both groups were already within the desirable range for LDL: HDL before the intervention, which is below 3.3, a reduction in the ratio represents a further reduction in the risk of CHD (Fernandez & Webb, 2008).

4.6. Health Screen

4.6.1. Systolic and diastolic blood pressure

SBP reduced significantly following the Wii aerobics programme. These findings concur with Balas-Nakash *et al*., (2010), who also found a significant decrease in blood pressure following a 12 week aerobic exercise intervention. Specifically, the low fit group's SBP reduced from 125 ± 12 mmHg to 119 ± 14 mmHg. These results demonstrate that Wii aerobics can be an effective mode of exercise for reducing resting SBP, much like conventional physical activities (Warburton *et al*., 2006; Lee & Paffenbarger, 2000). Additionally, such findings suggest that exercising using Wii aerobics may potentially reduce hypertension.

The high fitness group saw a significant reduction from 119 ± 11 mmHg to 109 ± 7 mmHg. However, the high fitness group also saw a significant decrease in SBP in the control condition, which suggests that the decrease in SBP was not a result of the Wii aerobics. DBP did not change significantly in either group following the Wii aerobics

intervention. In contrast, Balas-Nakash *et al*., (2010) established a significant ($p \leq 0.001$) decrease in DBP when participants exercised for 20 minutes five times per week, which suggests that a greater frequency of Wii aerobics may be needed to see significant changes in DBP. DBP did however; decrease significantly in the control condition for the high fit group, despite no exercise intervention taking place.

Based on these findings, it is possible that the high fitness group may have under reported their physical activity levels via the IPAQ, and were taking part in more exercise outside of the study that resulted in the decrease in SBP and DBP. This may be explained by the cardiorespiratory fitness levels following the intervention and during the control condition. The high fitness group showed an increase in estimated $V\,O_{2max}$ of 14 $ml \cdot kg^{-1} \cdot min^{-1}$ following the intervention, compared to a 6 $ml \cdot kg^{-1} \cdot min^{-1}$ increase seen by the low fitness group. Moreover, the high fitness group displayed an increase in estimated VO_{2max} in the control condition, whereas the low fitness group showed a decrease. Williams (2001) believes that physical fitness is a more accurate, although indirect measure of physical activity than a self report. Thus, the substantial increase in estimated VO_{2max} seen by the high fitness group in both conditions may suggest increased levels of physical activity outside of the study.

4.6.2. Estimated VO_{2max}

Participants' in both fitness groups showed a significant increase in estimated VO_{2max} following the four week intervention. Such findings show that participating in regular exercise on Wii aerobics improves cardiorespiratory fitness and may provide positive health benefits. In particular, a low level of cardiorespiratory fitness is a primary risk

factor for all cause mortality (Blair, 2009; ACSM, 2009), predominantly from CVD (ACSM, 2009). Furthermore, Warburton *et al*., (2006) proposed that a greater level of cardiorespiratory fitness is associated with a minimum 50% reduction in risk for CVD. However, it must be noted that some of the participants who completed the exercise intervention before the control condition had significantly higher $\dot{V}O_{2max}$ values at the end of the washout period compared with those taken at baseline. This suggests that the washout period may not have been adequate in duration for the effects of the exercise intervention to subside before entering the control condition. Consequently, the results of the control condition should be treated with caution.

4.6.3. Body composition

There were no significant changes in any of the body composition parameters in either fitness group. Caudwell *et al*., (2009) suggested that exercise alone is not enough to induce weight lost in many individuals without a dietary intervention. Furthermore, it has also been reported that 155-180 minutes of moderate intensity exercise per week is needed to see reductions in total mass and percentage body fat (Balas-Nakash *et al*., 2010). Therefore, it is unsurprising that no significant reductions in total mass or percentage fat mass were observed, as only 90 minutes of weekly moderate exercise was provided, and no dietary intervention was offered in the current study.

Nonetheless, the low fit group displayed a reduction in fat mass of 2% and an increase in fat free mass of 2%. Although negligible, the results agree with Miyachi *et al* (2010) in that it is possible for Wii aerobics to contribute to weight management, particularly as the low fit group showed an increase in body fat and a reduction in fat free mass in the control condition. The results support the findings of Slentz *et al*., (2004), who found

that weight gain can be prevented with modest amounts of exercise, irrespective of any dietary changes. Additionally, only the low fit group in the present study showed any changes in body composition, agreeing with Slentz *et al*., (2004) and Trejo-Gutierrez and Fletcher, (2007) that exercise may be more beneficial for weight loss in individuals with a higher body composition at the outset of exercise.

4.7. Cardiorespiratory responses to exercise

4.7.1. Heart rate

Exercising HR in the present study was similar to previous research examining Wii step aerobics (White *et al*., 2010; Graves *et al*., 2010). However, neither the low or high fitness group saw a significant decrease in HR following the Wii aerobics programme. Nonetheless, both groups showed a non-significant decrease in exercising HR following the exercise intervention. The low fit groups HR reduced from 125 $b \cdot min^{-1}$ to 116 $b \cdot min^{-1}$, whilst the high fit groups reduced from 117 $b \cdot min^{-1}$ to 110 $b \cdot min^{-1}$, demonstrating similar responses to regular exercise using conventional exercise techniques (ACSM, 2009).

In terms of exercising intensity, it was established that the low fitness group were exercising at 69% HR peak, and the high fitness group at 64% HR peak, which are comparable to the findings of White *et al*., (2010). Both these intensities are moderate according to ACSM (2009) guidelines, which suggest that exercising on the Wii may contribute to recommended daily physical activity levels. Furthermore, such findings suggest that Wii aerobics provides an intensity great enough to improve cardiorespiratory fitness in adults (Pollock *et al*, 1997 cited in Graves *et al*., 2010), as was evident by the changes in cardiorespiratory fitness established in the present study.

4.7.2. Energy expenditure

Exercising EE decreased significantly in both fitness groups following the four week Wii aerobics intervention. The exercising EE elicited by the participants in the present study was similar to that found by Graves *et al*., (2010). These results were surprising given that the current study used two Wii riisers in an attempt to augment the intensity. It would therefore be expected that the EE ($J \cdot kg^{-1} \cdot min^{-1}$) would be somewhat higher in the present study than in Graves *et al*, (2010), given that the latter study was performed on the Wii balance board alone. Specifically, the young adults (21-38 years) in Graves *et al*., (2010) study elicited an EE of 345.3 ± 59.6 $J \cdot kg^{-1} \cdot min^{-1}$ compared to 334 ± 39 $J \cdot kg^{-1} \cdot min^{-1}$ and 360 ± 37 $J \cdot kg^{-1} \cdot min^{-1}$ expended by the low and high fit group in the present study, both of which had similar average age ranges to those of Graves *et al*, (2010) young adults. It is therefore proposed that the Wii riisers did not effectively increase the intensity of Wii aerobics above using the balance board alone, contradicting the findings of Quinn (2010).

The low and high fitness groups in the current study expended an average of 366 kcal and 324 kcal per week using Wii aerobics. Accordingly, these findings are similar to Lanningham-Foster *et al*., (2009) who proposed a daily caloric burn of 124 playing active video games. In terms of weight loss, the EE provided by Wii aerobics in the present study would not be sufficient to see reductions in body mass, as 250–300 kcal (1050–1260 kJ) per session seems to be the threshold (Warburton *et al*., 2006). Based on the findings of the present study, participants would require 90 minutes of Wii aerobics per session to obtain similar EE. Consequently, Wii aerobics at the frequency/duration used in this study does not appear to be a sufficient stimulus for weight loss. Similarly, Durstine *et al*., (2002) proposed that noticeable differences in lipids generally occur when EE reaches between 1,200 to 2,200 kcals week. Thus, it is

plausible that the EE elicited by the participants during the Wii exercise intervention was not adequate enough to induce significant changes in blood lipids (excluding HDL). However, health benefits can be seen with lower amounts of EE (Warburton *et al*., 2006).

4.7.3 Relative VO_2

Much like the findings for estimated VO_{2max}, relative VO_2 showed an improvement in the low fit group following the Wii aerobics intervention. Specifically, relative VO_2 reduced from 16 $ml \cdot kg^{-1} \cdot min^{-1}$ to 13 $ml \cdot kg^{-1} \cdot min^{-1}$ pre to post intervention, demonstrating a reduced uptake of oxygen for the same exercise workload, which represents an improvement in cardiorespiratory fitness (ACSM, 2009). These findings contradict Graves *et al* (2010), who suggested that Wii aerobics would not be of adequate intensity to develop cardiorespiratory fitness. However, no improvements were observed in the high fit group for relative VO_2, although the results were close to significant (p =0.059). Therefore, it could be suggested that individuals with lower cardiorespiratory fitness benefit more from active gaming than those with high levels of cardiorespiratory fitness. In terms of CVD risk, these findings are important as Myers *et al* (2002) stated that exercise capacity is a greater predictor of mortality than any other CVD risk factor.

4.7.4. Metabolic equivalents (METs)

Similar to Graves *et al*., (2010) and Miyachi *et al.,* (2010), Wii aerobics was shown to be moderate intensity exercise. However, these findings disagree with those found by Quinn (2010), who established a vigorous intensity of 6 METs whilst using Wii aerobics with two Wii riisers. Nonetheless, these findings further support that playing

Wii aerobics may contribute to the daily recommended physical activity levels, based on the ACSM (2009) guidelines.

Over the course of the exercise intervention, METs ranged from 3.7-5.0, which would make Wii aerobics comparable to cricket, golf or gymnastics in terms of intensity (Ainsworth *et al*., 1993). Wii aerobics has been established as a moderate intensity activity. However, in accordance with Miyachi *et al*, (2010), it has once more been established that the exercises do not compared to the authentic versions. Specifically, regular step aerobics using a 6-8 inch step is said to be 8.5 METs (Ainsworth *et al*., 1993), whereas Wii aerobics with a 7 inch step in the present study reached a maximum of 5 METs. Nonetheless, a training effect was established as METs decreased significantly in the low fit group from 4.7 to 3.7 METs following the intervention, whilst the high fit group showed a non-significant decrease from 5.0 to 4.3 METs. This demonstrates a reduction in the energy costs for the same sub maximal workload, and therefore an improvement in respiratory function (ACSM, 2009). However, further improvements in cardiorespiratory fitness would require an increase in METs to elicit an adequate stimulus required for future adaptations (Warburton *et al*., 2006).

4.8. Limitations

As is common with many active gaming studies (Graves *et al.,* 2008; Sell *et al*., 2008; Willems & Bond, 2009), the sample size of the current study was small. Furthermore, the sample consisted only of participants 18 years and older and as a result limits the generalisations to younger populations. The timing of the blood samples may also have been a limitation of the present study. Specifically, blood sampling for lipid assessment should be done following an overnight fast of 12-14 hours (Marshall & Bangert, 2008).

In the current study a fast was not enforced, consequently the blood samples may not have been an entirely accurate representation of the participant's lipid profiles. Much like Graves *et al*., (2007), this decision was made as participant availability was limited to specific times of the day, and in some cases fasting would have been unethical (for example, testing at 4pm so fasting through waking hours). Additionally, alcohol consumption was not controlled for. This is particularly important as alcohol consumed the evening prior to blood sampling can cause hypertriglyceridemia (Marshall & Bangert, 2008). Furthermore, as diet was not monitored, it cannot be ruled out that participants increased or decreased their dietary intake during the study. This could affect measures of body composition and lipids, both of which can be influenced by dietary changes (Caudwell *et al*., 2009; Nestel, 2008).

4.9. Practical implications

Warburton *et al*., (2006) and Rhodes *et al*., (2008 cited in Mark & Rhodes., 2009) both suggested that individuals show a greater adherence to "active gaming" sessions than those using conventional exercise techniques. In accordance, the current study established a 0% dropout rate to the four week exercise intervention using the Nintendo Wii. These findings are particularly significant as dropout rates for structured exercise interventions have ranged from 9% to as much as 87% (Marcus *et al*., 2006). Based on previous research by Graves *et al*., (2010) and Rhodes *et al*., (2008 cited in Mark *et al*., 2009), it is probable that participants enjoyed the active gaming sessions more than if a conventional exercise program was offered and therefore showed a higher rate of adherence as a result. Furthermore, these findings support Graves *et al*., (2010) theory that inactive individuals who do not adhere to regular exercise programs may be able to adhere to light to moderate active gaming. Consequently, people may show a greater

adherence to active gaming on Wii aerobics. Moreover, Wii aerobics could contribute to the daily recommended physical activity levels and potentially provide numerous health benefits which are associated with regular, moderate intensity physical activity, including a reduced risk of CVD (ACSM, 2009).

4.10. Future research

Although significant findings were established in the present study, it is not known whether these would translate to other games on the Wii fit and Wii sports. Future research is needed to establish the effects of different Wii fit games on CVD risk markers. Especially, as the choice of active game is thought to be important for physical activity interventions (White *et al*., 2010). Specifically, it is probable that games like Wii aerobics that require whole body movements using large muscle groups would be more metabolically demanding than games like Wii tennis and bowling, which require only limited upper body movement (White *et al*., 2010).

Further research is also needed to establish the effects of a higher frequency or duration of Wii games. The findings of this study suggest that three sessions per week may not have been enough to see adaptations in some variables. Warburton *et al*., (2006) suggested that a minimum EE of 1000kcal a week is needed for health benefits. In order to obtain this, participants in the current study would have to play Wii aerobics for one hour each day, five days per week. This level of physical activity also meets the requirements of well established physical activity guidelines, such as the ACSM (2009). Thus, interventions using the Wii for this period of time should be explored. Additionally, future research is required in order to assess the effects of similar interventions in younger populations.

4.11. Conclusion

The findings of the current study support the theory of Graves *et al*., (2010), who proposed that light to moderate activities on the Wii Fit may potentially reduce the risk of CVD in older populations. The Wii aerobics intervention had beneficial effects on numerous CVD risk markers including; plasma lipids, cardio-respiratory fitness and blood pressure. Specifically, a significant increase in HDL cholesterol and estimated $\dot{V}O_{2max}$ was established. Moreover, a significant reduction in SBP, and the LDL: HDL ratio was observed. In particular, these findings agree with Warburton *et al*., (2006), in that the most favourable health improvements are seen when individuals with low cardiorespiratory fitness become physically active. However, individuals with high levels of cardiorespiratory fitness also appear to benefit from active gaming too. It has also been demonstrated that participants maintained a high level of adherence to the Wii Fit sessions in the present study, providing encouraging results for maintaining physical activity, thus improving another CVD risk factor, physical inactivity.

Although the physical activity on the Wii provided positive results with regards to HDL, no significant findings were found for LDL, TC or TG. Kelley and Kelley (2006) proposed that aerobic exercise alone may not be sufficient to reduce lipoprotein profiles in individuals with less than optimal levels. Hence, it is thought that additional interventions (dietary and/or pharmacological) may be required to establish significant changes in lipids (Kelley & Kelley 2006). Nonetheless, the reductions in LDL, TC and TG may have some clinical benefits based on the percentage reductions.

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6. SELF REFLECTION

The first thing to highlight is how much more difficult this year has been then I expected. I am likely to be one of the more laid back people you would meet, rarely becoming stressed out, even at things I probably should be stressed out at. This MSc however, seems to have pushed me a little bit further than I have been before. Without over exaggerating, this has easily been the most stressful year of my life. What seemed like a relatively straight forward goal of designing a study, carrying it out and writing about it, seems to have hit every possible hurdle on the way.

Having said that, I am pleased with what I have achieved this year, especially the completion of the testing, given the scope of it all compared to my undergraduate research. At first it seemed a bit too much to handle when we first discussed the project, particularly the idea of trying to find so many people that would give much of their own time to take part in the study for 10 weeks (as well as give me some of their blood). At this point I was really sceptical of achieving this and even thought of quitting altogether as I had convinced myself it was not going to happen. However, the response to the study once we sent out the adverts was fantastic. I guess my inexperience of recruiting for such studies was a reason for my negative attitude.

What this year has confirmed to me, is that I have real difficultly motivating myself to do work when there has not been a deadline set by lecturers or tutors. On several occasions I would ask my supervisor for a deadline for a particular section, just so it would get me moving in terms of doing some work. I am not sure if this is a trait I always display or whether this is a consequence of taking up this MSc straight after my undergraduate degree. I had convinced myself all the way through the 3rd year of my undergraduate degree that this was the last year I would have to study. The thought of

doing an MSc never crossed my mind. However, when the opportunity of a scholarship came up, it was too good of an opportunity to turn down. Coming straight from an undergrad degree onto another, more difficult year has proved very challenging. On an independent project like this, it is very easy to lose focus and not do any work for large periods of time. The testing phase was probably the time I was most focused, largely due to the fact that I was in the labs 9-5pm for 10 weeks. Nonetheless, I have managed to make it to the end, which I guess is testament to my determination to finish this MSc.

In terms of skills learned, I do not think I have gained a lot of new skills, but instead refined the ones I already have. I believe I have become a better writer, mainly due to some excellent classes on the graduate research skills programme, which have helped me to write more succinctly in particular. I feel much more confident in a laboratory setting having spent so much time in the labs for this project. This includes “wet lab” experience, dealing with blood samples and their analysis. Most of all, I feel much more confident with the practical side of lab work, particularly instructing participants as well as competency with the laboratory equipment. This also comes from work done outside of the project with some of the professional sports teams that work with UCLan.

Overall, despite the difficulties I have faced this year, I do not regret doing this MSc. I am proud of what I have achieved this year and hope to use what I have learned to benefit me in the future.

APPENDIX 1

Statistical considerations for a cross-over study where the outcome is a measurement

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Request

0.5 Significance Level (%) — 2 sided *(default is 0.05, two-sided)*

3.4 ◉ Within patient standard deviation *(if known)*, or ○ Standard deviation of the difference between the two value for the same patient *(if known)*

Enter two of the following three values and the remaining value will be calculated

1. [ ] Total number of patients
2. 0.8 Power (usually 0.8 or 0.9)
3. 8.5 Minimal detectable difference in means

Response

Calculation performed at: Fri Apr 15 2011 21:04:01 GMT+0100 (GMT Daylight Time)

The provided parameters were: significance level (adjusted for sidedness) = 0.025, standard deviation within patients = 3.4, standard deviation of the difference = undefined, number of patients = undefined, power = 0.8, difference in means = 8.5.

The variable calculated was the total number of patients.

A total of 5 patients will enter this two-treatment crossover study. The probability is 83 percent that the study will detect a treatment difference at a two-sided 0.05 significance level, if the true difference between treatments is 8.500 units. This is based on the assumption that the within-patient standard deviation of the response variable is 3.4.

This software developed by David Schoenfeld, Ph.D. (dschoenfeld@partners.org), with support from the MGH Mallinckrodt General Clinical Research Center. Javascript version developed by REMorse.

APPENDIX 2

Do you want to get Wii Fit™?

Do you fancy a change to conventional exercise methods? As part of our MSc project, we are offering a FREE health screen and a four week step aerobics programme using the Nintendo Wii. Perfect if you are keen to improve your health, fitness and overall well-being.

Interested? Please contact either Francesca or Matt on:

Francesca: 07878929049
Matt: 07898853727

FPell@uclan.ac.uk
MPDuckham@uclan.ac.uk

APPENDIX 3

University of Central Lancashire

School of Psychology

CONSENT FORM

Title of Project: The effect of step aerobics using Nintendo Wii Fit on immune function and blood lipids

Name of Researcher(s): Matthew Duckham & Francesca Pell (MSc students)

Name of Supervisor(s): Steve Atkins, Stephanie Dillon, & David Fewtrell

Please Tick box

I confirm that I have read and understand the participant information sheet for this study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.	☐
I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason.	☐
I agree to take part in this study.	☐

Name of Participant	Date	Signature

I confirm that I have explained to the above individual the nature, purpose and possible risk associated with the participation in this research study, and have answered any questions that have been raised.

Researcher(s)	Date	Signature

APPENDIX 4

Participant Information Sheet

We (Francesca Pell and Matthew Duckham) are currently masters (MSc) students undertaking a masters research project. The following information will indicate why we are conducting this research and what participation will entail. Please read the following information carefully. You may ask any questions if you are not clear on anything or if you would like more information.

Study title

The effect of a four week Nintendo Wii Fit training programme on blood lipids and immune function.

What is the purpose of the study?

To investigate the effect of a four week Nintendo Wii programme (step aerobics) on blood lipids (e.g. cholesterol) and immune function.

Why have I been chosen?

You have kindly volunteered to take part in this study.

Do I have to take part?

Taking part in this study is entirely voluntary. You may withdraw at any time (see contact details at the bottom of the page) before completing your final testing session, at which point your data will be anonymised (for the purpose of analysis) and therefore we cannot trace your results back to you personally after this time.

What do I have to do?

Participants are invited to attend the sports physiology lab at the University of Central Lancashire. Participants will be asked to give both a blood and two saliva samples, along with some basic data (height, weight etc). After four weeks have passed, these measures will be repeated and participants will begin their four week step aerobics programme, using the Nintendo Wii. Each training session will be about 20 minutes long and it is hoped participants will attend three sessions per week for the entire four week training programme. When the four week training has been completed, a third and final measure of blood, saliva and the body (height, weight etc) will be taken. In addition, participants will be asked to wear a metalyser (meta-max) during the first and last training session of the Nintendo Wii

programme. This involves placing a face mask over the mouth and nose, whilst wearing a device that rests on the shoulders, this allows measures of heart rate, oxygen consumption and other gas analyses to be obtained. Blood samples will be taken using a lancet to make a small puncture (finger prick) at the end of a finger of choice (index or middle finger), whilst the blood is collected. In addition, a saliva sample will be taken by passively dribbling through a straw.

What are the possible risks of taking part?

The study will include some moderate-to-vigorous physical activity and as such, a questionnaire (PAR-Q) is used to assess participants' suitability. In subsequent exercise sessions, you will be asked if your health has changed so that you now answer 'YES' to any of the questions on the PAR-Q. Participation will be dependent on this response. Moreover, a comprehensive risk assessment has been undertaken to identify and control any potential risks, in order to help ensure the safety of all participants.

What are the possible benefits of taking part?

You may enjoy this exciting new way in which to exercise on the Nintendo Wii, not to mention the health benefits that are commonly associated with participation in physical activity. You will receive a free health screen (e.g. blood pressure and physical fitness test) and will also be taking part in an innovative study, which can contribute to the limited research in this area.

Will the results be confidential?

The results of the study will be anonymous in that no results can be linked the participants' name. This will be achieved by identifying participants by a unique number rather than their name, a record of which will be stored separate from any of the participants' results. In no instance will individual data be presented, only group averages.

What will happen to the results of the research study?

The data will be saved on a password protected laptop and then incorporated into a written report and presentation, which will be assessed by internal and external staff at UCLAN. Thereafter, it is possible that the results may be published in an academic journal.

Student Contact Details

For further information or if you wish to withdraw, please do not hesitate to contact:

Matthew Duckham (BSc Hons)
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MPDuckham@uclan.ac.uk

Francesca Pell (BSc Hons)
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Further Information

Please let us know if we can be of assistance in directing you to any further information sources relating to health and fitness.

APPENDIX 5

School of Psychology RISK ASSESSMENT FORM (Medium & High Risk, Student Version)

Use this form to risk-assess:
Off-campus student activities (research, fieldwork, educational visits etc) in medium/high risk environments such as factories, farms, prisons, remote areas or participants' homes.
All student activities involving medium/high risk procedures or use of specialist equipment.
For low risk locations and activities, use the appropriate low risk form.

This form should be completed by the staff member responsible for the activity (e.g. the project supervisor), in consultation with the student and a qualified or otherwise competent person (normally a technician or Faculty HSE officer). Completed forms must be countersigned by the Head of School or the Chair of the School Health & Safety Committee.

Students:	**Assessment Undertaken By: (Staff member)**	**Assessment Verified By: (Technician or other competent person)**
Names: Francesca Pell Matthew Duckham	Name:	Name:
Signed:	Signed:	Signed:
Date: 22/03/10	Date:	Date*:
Note: Risk Assessment is valid for **one year from the date given above. Risk Assessments for activities lasting longer than one year should be reviewed annually.*		
Countersigned by Head of School or Chair of H&S Committee:		
Date:		

Risk Assessment For:
Activity: Four week step aerobics programme using the Nintendo Wii Fit. Blood and saliva sampling
Location of Activity: *University of Central Lancashire* *Darwin Room 026 (Physiology Laboratory)* *Preston* *Lancashire* *PR1 2HE*

List significant hazards here:	List groups of people who are at risk:	List existing controls, or refer to safety procedures etc:	For risks which are not adequately controlled, list the action needed:	Remaining level of risk (high, medium or low):
Obstacles	Participants, Investigators,	Check area before and throughout testing		Low
Injury	Participants	Qualified First Aider present, equipped with First Aid Kit and defibrillator.	Phones available	Med
Slippery/wet surfaces	Participants, Investigators,	Warning signs	Assess prior to testing and re-assess throughout testing	Med
Equipment	Participants, Investigators	Equipment regularly checked and maintained	Test before use	Med
Inappropriate footwear and/or clothing	Participants	Participants advised to come wearing the correct clothing and footwear for physical activity	Check clothing/footwear and exclude participants from the study if it is inappropriate	Low
Trails not appropriate for the health of the	Participants	Screening (PAR-Q)	Inability to satisfy a health questionnaire will result in exclusion	High

participant			from testing	
Fire	Participants, Investigators	Alarms, knowledge of fire exits and drills		High
Electrical Items	Participants, Investigators	Cover/tape any trailing cables, check that it is well maintained	Check before use and use in accordance with instructions	Med
Jewellery	Participants, Investigators	Advise participants to remove or cover any jewellery prior to the testing		Low
Untied long hair	Participants	Provide bobbles so participants can tie back hair		Low
Blood Collection	Participants, Investigators	Investigator will be familiar with the appropriate procedure (see below). Latex gloves and a plastic bib will be worn. The finger will be sterilised using alcohol wipes. New gloves and lancets will be used for each		High

		participant.		
Saliva Collection	Participants, Investigators	Investigator will be familiar with the appropriate procedure (see below). Latex gloves and a plastic bib will be worn. Each participant will be provided with an individual straw and cryovial for their saliva sample.		Med
Bodily Waste Products		Sharps will be disposed of appropriately in a sharp bin, whilst contaminated tissues/gloves etc will be disposed of in a clinical waste bag	Subsequently, these will be collected by the appropriate professionals and disposed of in accordance with relevant guidelines	High
Sample Storage		Samples will be labelled and appropriately stored (frozen) in preparation for analysis		Low

APPENDIX 6

Physical Activity Readiness
Questionnaire - PAR-Q
(revised 2002)

PAR-Q & YOU

(A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.

YES	NO	
☐	☐	**1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?**
☐	☐	**2. Do you feel pain in your chest when you do physical activity?**
☐	☐	**3. In the past month, have you had chest pain when you were not doing physical activity?**
☐	☐	**4. Do you lose your balance because of dizziness or do you ever lose consciousness?**
☐	☐	**5. Do you have a bone or joint problem (for example, back, knee or hip) that could be made worse by a change in your physical activity?**
☐	☐	**6. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?**
☐	☐	**7. Do you know of any other reason why you should not do physical activity?**

If you answered

YES to one or more questions

Talk with your doctor by phone or in person BEFORE you start becoming much more physically active or BEFORE you have a fitness appraisal. Tell your doctor about the PAR-Q and which questions you answered YES.

- You may be able to do any activity you want — as long as you start slowly and build up gradually. Or, you may need to restrict your activities to those which are safe for you. Talk with your doctor about the kinds of activities you wish to participate in and follow his/her advice.
- Find out which community programs are safe and helpful for you.

NO to all questions

If you answered NO honestly to all PAR-Q questions, you can be reasonably sure that you can:

- start becoming much more physically active – begin slowly and build up gradually. This is the safest and easiest way to go.
- take part in a fitness appraisal – this is an excellent way to determine your basic fitness so that you can plan the best way for you to live actively. It is also highly recommended that you have your blood pressure evaluated. If your reading is over 144/94, talk with your doctor before you start becoming much more physically active.

DELAY BECOMING MUCH MORE ACTIVE:

- if you are not feeling well because of a temporary illness such as a cold or a fever – wait until you feel better; or
- if you are or may be pregnant – talk to your doctor before you start becoming more active.

PLEASE NOTE: If your health changes so that you then answer YES to any of the above questions, tell your fitness or health professional. Ask whether you should change your physical activity plan.

Informed Use of the PAR-Q: The Canadian Society for Exercise Physiology, Health Canada, and their agents assume no liability for persons who undertake physical activity, and if in doubt after completing this questionnaire, consult your doctor prior to physical activity.

No changes permitted. You are encouraged to photocopy the PAR-Q but only if you use the entire form.

NOTE: If the PAR-Q is being given to a person before he or she participates in a physical activity program or a fitness appraisal, this section may be used for legal or administrative purposes.

"I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction."

NAME _____

SIGNATURE _____ DATE _____

SIGNATURE OF PARENT _____ WITNESS _____
or GUARDIAN (for participants under the age of majority)

Note: This physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if your condition changes so that you would answer YES to any of the seven questions.

Supported by: Health Canada Santé Canada

continued on other side...

APPENDIX 7

STAFF, MPhil/PhD & MSc RESEARCH PROJECTS

Before completing this form you should read the UCLAN *Code of Conduct* and the British Psychological Society *Code of Conduct* (both online at *www.uclan.ac.uk/scitech/psychology/research/ethics.php)*. In addition, for questions 4-22, please see the attached guidance notes. PhD & MSc students should discuss the completion of this form with their supervisor.

All researchers MUST obtain ethical approval BEFORE collecting any data.

Research Team
Researcher name(s) & email

Matthew Duckham - MPDuckham@uclan.ac.uk

Researcher type: MSc Student

Supervisor name(s) & email (if applicable)

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Project details (please see attached guidance notes)
What is the project title?

The effect of Active Gaming on Blood Lipids as a Measure of CVD risk

What is the likely duration of project?

One Year

Please provide a brief summary of the project aims (Max 250 words)

The aim of this research project is to investigate the effect of a four week training programme using the Nintendo Wii Fit on blood lipid levels. Specifically, examining the impact the "Wii fit" programme may have on cardiovascular risk factors such as plasma triglyceride and lipoprotein levels. In particular, low density lipoproteins and high density lipoproteins. Currently, the literature surrounding the Nintendo Wii focuses primarily on the energy expenditure and weight loss derived from game play (Graves et al., 2008, Bausch et al., 2008). Currently, there are no studies that examine the effect of Nintendo Wii game play at a biochemical level. Furthermore, methodologies in current literature have only used a single bout of exercise as opposed to a four week intervention intended in this research.

Please provide a brief summary of the project methods (Max 250 words)

With regards to the methodology a single subjects design will be employed. The experimental design will be eight weeks in total. Baseline measurements of blood lipids will be taken on week one of the programme. Subsequently, post control measures will be taken on week four. Participants will then

take part in a four week training programme using the Nintendo Wii Fit Plus hardware on the Nintendo Wii console. Each participant will take part in 3 sessions of moderate/vigorous intensity exercise per week, for approximately 30 minutes each session. This will be done for the duration of the four week programme. Following the programme, participants will have blood samples taken again (week 8) to assess lipid and plasma triglyceride levels, thus examining the effect of the "Wii Fit" training programme on these cardiovascular disease markers compared to the control condition.

The blood sample will be taken intravenously using a vacutainer, taking blood from a vain in the arm. The blood will then be centrifuged to seperate the plasma from the whole blood. Next the plasma will be pipetted into safe seal tubes and then stored (frozen) for analysis. Approximately 2 mL of blood will be taken. Analysis of the blood will be done using a Dr Lange miniphotometer.

Does the research involve contact with any other organisation or group (e.g. schools, companies, charities, hospitals, sports clubs)? If **yes**, please give details.

No

Is the research to be funded externally? If **yes**, please give details.

No

Will ethical approval for the proposed research be sought from any other body (e.g. collaborating departments, Home Office, health authority, education authority)? If **yes**, please give details.

No

Has a Risk Assessment form been completed?

Yes (see attached)

Has permission been obtained to use any copyright materials (e.g. personality tests)? Please also indicate whether particular qualifications or training are needed to administer the tests, and if so, whether the researcher is appropriately qualified.

A basic first aid and defibrillator course has been undertaken to allow unsupervised use of the laboratory and equipment.

Participants (Please see attached guidance notes. Projects without participants may leave this section blank and proceed to Q. 22.)
Who do you propose to use as participants and do they belong to a group unable to provide **informed** consent?

The healthy adults with a sedentary lifestyle recruited for this study will all be able to provide written informed consent

Please indicate exactly how participants will be recruited for the project.

Advertisments will be used in order to recruit participants (see attached advert). These will include e-mails, posters and computer based advertisments (screen savers) which will be viewable by both UCLAN staff and students.

How exactly will consent be given (e.g., verbal or written)?

Written (see attached)

What information will be provided at recruitment and briefing to ensure that consent is **informed**?

A participant information sheet (see attached) will be given to all participants at briefing

Please indicate what information will be provided to participants at debrief.

All relevant information will be included in the participation information sheet which will be given out at briefing.

Please give details of any proposed rewards or incentives to be offered to encourage participation.

None

Is any deception involved? (If **yes**, please give details and explain why deception is necessary.)

No

Does the procedure involve **any** possible distress, discomfort or harm to participants? If so, what measures are in place to reduce it?

The methodology involves taking an intravenous blood sample from a vein in the arm, which may result in some discomfort. However, a trained phlebotomist will take the sample. Additionally, a risk assessment has been undertaken (see attached) to prevent and reduce the risk to participants. All participants will be informed of the procedures prior to testing and will then be given the option to participate (informed consent). In addition, the participants may withdraw at any time.

What mechanism is there for participants to withdraw from the investigation and how is this communicated to participants?

Participants may withdraw at any time up until the data has been analysed. At this point the data will be anonymised and can not be linked to the participants. This information will be explained in the participant information sheet.

How are confidentiality and/or anonymity to be maintained?

The participants names will be stored on a password protected laptop, which will only be accessible by the researchers involved in the study. Each of the participants will be designated a number which will be used to identify their data and thus maintain anonymity. Individual data will not be presented at any time, only group averages will be used. This data will be seen by the professionals marking the

written report and viva and potentially the general public, should the study be published. At no point in time will any individual data be used that may be traceable back to a specific participant.

Additional information
Please give details of any other ethical issues that have been considered.

N/A

Submission checklist:
Please attach any risk assessments, questionnaires, interview schedules, experimental protocols, other relevant research materials, advertisements, introductory letters, letters of approval, consent forms, participant briefing/debriefing materials, etc.
Please do NOT submit unnecessary material (for example, multiple copies of the same questionnaires, risk assessment notes or ethics guidance notes, etc.). Staff and Mphil/PhD students should submit the ethics form and attachments to **Susan Ross** (DB120). MSc students should submit the forms to their project supervisor.
Dates of Ethics Committee meetings and submission deadlines are available at: www.uclan.ac.uk/scitech/psychology/research/ethics.php

Would you like to attend the ethics meeting to discuss your proposal (staff, PhD researchers and MSc *supervisors*, not normally MSc students, are welcome to attend that part of the meeting at which their research is to be discussed)? No

(If you indicate 'yes', please make sure you are available 1-3 pm on the day of the meeting and include a contact number we can reach you on when your proposal is about to be considered. Please leave your office extension number and, if you wish, a mobile number here:)

Please print and sign – remember to print from page 4 onwards only.

Signed ……..……..……...…

(Signing this form certifies that you agree to carry out your research in the manner specified. If you want to deviate from the approved method at any time, you should seek further ethical approval for the change.)

Date …….

Supervisor signature (MSc projects only)……

(**Note to supervisors:** Signing this form certifies that, in your opinion, the project specified here is ethical under Departmental and BPS guidelines. Do not sign if you are unsure, or if the student has not attached final versions of the research materials they are planning to use.)

APPENDIX 8

Group Statistics

	Poor_or_Good	N	Mean	Std. Deviation	Std. Error Mean
Age_yrs	1.00	7	40.0000	12.66228	4.78589
	2.00	8	33.8750	14.05538	4.96933
Mass_kg	1.00	7	74.5714	7.72673	2.92043
	2.00	8	63.7500	6.58461	2.32801
Height_cm	1.00	7	165.7143	6.07493	2.29611
	2.00	8	162.6250	4.86056	1.71847
SBP_mmHg	1.00	7	125.3571	12.34812	4.66715
	2.00	8	118.6250	10.70297	3.78407
DBP_mmHg	1.00	7	83.6429	9.28132	3.50801
	2.00	8	77.2500	9.67323	3.42000
BF_Percent	1.00	6	34.5167	13.39394	5.46805
	2.00	7	27.6286	12.79163	4.83478
Estimated_VO2max_mL_kg_min	1.00	7	28.6714	5.09814	1.92692
	2.00	8	43.3900	6.29571	2.22587

Independent Samples Test

	Levene's Test for Equality of Variances		t-test for Equality of Means						
								95% Confidence Interval of the Difference	
	F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
Age_yrs	.626	.443	.881	13	.394	6.12500	6.95088	-8.89146	21.14146
			.888	12.980	.391	6.12500	6.89920	-8.78217	21.03217
Mass_kg	1.084	.317	2.931	13	.012	10.82143	3.69245	2.84436	18.79849
			2.897	11.922	.013	10.82143	3.73478	2.67813	18.96473
Height_cm	.271	.611	1.094	13	.294	3.08929	2.82309	-3.00964	9.18821
			1.077	11.509	.303	3.08929	2.86797	-3.18917	9.36774
SBP_mmHg	.572	.463	1.132	13	.278	6.73214	5.94746	-6.11655	19.58084
			1.120	12.027	.284	6.73214	6.00845	-6.35594	19.82022
DBP_mmHg	.009	.927	1.301	13	.216	6.39286	4.91380	-4.22276	17.00847
			1.305	12.865	.215	6.39286	4.89924	-4.20265	16.98836
BF_Percent	.033	.859	.947	11	.364	6.88810	7.27084	-9.11491	22.89110
			.944	10.517	.366	6.88810	7.29895	-9.26724	23.04343
Estimated_VO2max_mL_kg_min	.832	.378	-4.925	13	.000	-14.71857	2.98829	-21.17439	-8.26275
			-4.999	12.943	.000	-14.71857	2.94406	-21.08169	-8.35545

APPENDIX 9

1a. During the last 7 days, on how many days did you do **vigorous** physical activities like heavy lifting, digging, aerobics, or fast bicycling,?

Think about *only* those physical activities that you did for at least 10 minutes at a time.

_____ **days per week** ⇨

1b. How much time in total did you usually spend on one of those days doing vigorous physical activities?

_____ **hours** _____ **minutes**

or

☐ **none**

2a. Again, think *only* about those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do **moderate** physical activities like carrying light loads, bicycling at a regular pace, or doubles tennis? Do not include walking.

_____ **days per week** ⇨

2b. How much time in total did you usually spend on one of those days doing moderate physical activities?

_____ **hours** _____ **minutes**

or

☐ **none**

3a. During the last 7 days, on how many days did you **walk** for at least 10 minutes at a time? This includes walking at work and at home, walking to travel from place to place, and any other walking that you did solely for recreation, sport, exercise or leisure.

_____ **days per week** ⇨

3b. How much time in total did you usually spend walking on one of those days?

_____ **hours** _____ **minutes**

or

☐ **none**

The last question is about the time you spent sitting on weekdays while at work, at home, while doing course work and during leisure time. This includes time spent sitting at a desk, visiting friends, reading traveling on a bus or sitting or lying down to watch television.

4. During the last 7 days, how much time in total did you usually spend *sitting* on a **week day?**

____ **hours** _____ **minutes**

This is the end of questionnaire, thank you for participating.

This is the final SHORT LAST 7 DAYS SELF-ADMINISTERED version of IPAQ from the 2000/01 Reliability and Validity Study. Completed May 2001.

APPENDIX 10

HDL Precipitant
BCZ 326

DR LANGE

HDL Cholesterol
Dextran sulfate method

Article No. BCZ 326
1 x 25 ml

For the HDL cholesterol determination a package LCN 350/ 450 or LKM 226 (CHOD/PAP method) is needed in addition.

Method[1]
Precipitation with dextran sulfate/magnesium chloride.
Cholesterol determination based on the CHOD/PAP method.
VLDL (very low-density lipoproteins) and LDL (low-density lipoproteins) are precipitated by dextran sulfate and magnesium ions. After centrifuging, the HDL (high-density lipoproteins) remain in the supernatant and their cholesterol content can be determined enzymatically.

Active components

HDL precipitant	Dextran sulfate:	7.5	g/l
	Magnesium chloride:	0.54	mol/l
	Sodium azide:	< 0.1	%

Classification
No hazardous product as specified in Directive 67/548/EEC.

Preparation of reagent and stability
The reagent is ready-to-use.
The HDL precipitant stored at +2 °C to +8 °C can be used until the stated expiry date.

Sample material
Serum, heparinized or EDTA plasma
The serum should be separated from the blood coagulum as quickly as possible. The HDL cholesterol in the sample is stable at +2 °C to +8 °C for 14 days[2].
The decanted supernatant after precipitation is usable for 24 hours when kept at +2 °C to +8 °C.

Measuring conditions
See package insert LCN 350/450 resp. LKM 226.

Procedure

1. Precipitation

Pipette into a centrifuge tube:		
	Analysis	Analysis*
Serum	500 µl	200 µl
Precipitant	50 µl	20 µl
Mix well and allow to stand for 10 minutes at room temperature. Centrifuge at least at 3000 rpm for 10 minutes.		

* When using this pipetting scheme please use 1.5 ml reaction vessel.

2. HDL cholesterol determination in the supernatant

a) Procedure LCN 350/450

Pipette into semi-micro cuvettes:		
	Blank	Analysis
Reagent	500 µl	500 µl
Supernatant	--	20 µl
Mix well, incubate at room temperature for 10 minutes or 5 minutes at 37 °C and measure analysis against blank within 60 minutes.		

b) Procedure LKM 226

Pipette into round cuvette:	
	Analysis
Supernatant	50 µl
Mix, rotate the start cap and reclose the cuvette. Dissolve the contents by shaking. After 5-15 minutes read one reagent blank (unused cuvette) and measure all analyses against it.	

Calculation

HDL cholesterol : $c = F_I \times A$ [mg/dl]
$c = F_{II} \times A$ [mmol/l]

Wavelength	Kit	F_I [mg/dl]	F_{II} [mmol/l]
520 nm	LCN 350/450	176	4.56
	LKM 226	140	3.63
546 nm	LCN 350/450	244	6.32
	LKM 226	187	4.83
560 nm	LCN 350/450	310	8.03
	LKM 226	238	6.17

Notes
1. The supernatant after centrifuging must be clear. If the supernatant is cloudy or if a part of the precipitated lipoproteins floats on the surface, the sample must be diluted 1:1 with physiological saline. Mix 500 µl serum with 500 µl physiological saline and repeat the precipitation using 500 µl of this dilution. Multiply the result by 2.
2. In case of triglyceride values exceeding 400 mg/dl (4.56 mmol/l) serum, dilute as in 1. above.
3. In case a micro-centrifuge is available, the precipitation can be performed with 200 µl serum and 20 µl precipitant. For the subsequent cholesterol determination 20 µl (LCN 350/450) or 50 µl (LKM 226) supernatant are used.
4. Strongly hemolytic sera (hemoglobin exceeding 100 mg/dl resp. 0.062 mmol/l) cannot be used. Bilirubin disturbs from about 10 mg/dl (171 µmol/l) on.
5. The precipitant contains sodium azide (< 0.1 %) as a preserving agent. Swallowing and contact with the skin or the mucous membranes must be avoided.

Reference ranges[3]

[mmol/l]	desirable	borderline	high risk
Male	> 1.45	1.45 - 0.90	< 0.90
Fermale	> 1.68	1.68 - 1.15	< 1.15

Quality control
You can use LT-SYS (article no. LCQ 710) and all control sera with cholesterol target values of these method.

References

1) Kostner GW. Clin Chem 1976; 22:695
2) Finley PR. Clin Chem 1978; 24:931
3) Assmann G. Der Praktische Arzt 1979; 3922

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Dr. Lange Cuvette Test
LKM 226

DR LANGE

Cholesterol
CHOD-PAP method

Article No. LKM 226
40 cuvettes

Method
Enzymatic colorimetric test, CHOD-PAP method[1)]

Cholesterol esters + H_2O —(Cholesterolesterase)→ Cholesterol + fatty acids

Cholesterol + O_2 —(Cholesteroloxidase)→ Cholestenon + H_2O_2

H_2O_2 + 4-Chlorophenol + 4-Aminophenazone —(Peroxidase)→ Quinoneimine dye + 2 H_2O + HCl

The determination at 520 nm can directly employ whole blood. It is *immediately and completely hemolysed by the buffer solution.*
Results obtained from blood samples also refer to the plasma concentrations as the erythrocytes content of each individual sample is taken into account. Calculation is automatically done by a special evaluation program.

Active components

1. Buffer solution (prepipetted in LKM 226)	Pipes buffer, pH 7.6:	75	mmol/l
	Lipoproteinlipase:	≥ 800	U/l
	4-Chlorphenol:	9	mmol/l
	EDTA:	3.8	mmol/l
	Detergent:	0.15	%
	Sodium cholate:	3.5	mmol/l
	Sodium azide:	< 0.1	%
2. Starting reagent (in start caps)	4-Aminophenazone:	0.2	mmol/l
	Cholesterinoxidase:	≥ 350	U/l
	Peroxidase:	≥ 4.0	kU/l

Classification
1. No hazardous product as specified in Directive 67/548/EEC.
2. Hazardous product as specified in Directive 67/548/EEC and 1999/45/EEC. Classified as harmful with R 42/43 (may cause sensitization by inhalation and skin contact).

Preparation of reagent and stability
The reagents are ready-to-use.
The reagents are stable at +2 °C to +8 °C until the expiry date stated on the packing.
Please note that the lyophilized starting reagent is moisture-sensitive!
At high ambient temperature bring cuvettes with the start caps to room temperature before removing the foil.
Do not freeze the reagent!
Categorically avoid contamination with the reagent.

Sample material
Whole blood, serum, heparinized or EDTA plasma
Transfer blood samples immediately into buffer solution.
Stability of cholesterol in closed LKM 226 (without starting reagent) cuvette before measurement:
at +2 °C to +8 °C: 8 hours
at +15 °C to +30 °C: 4 hours

Measuring conditions

Wavelength:	LP 20, LP 400/420, LP 800 (appropriate software for whole blood needed)	520 nm
	LP 6, LP 6 A/S	Hg 546 nm
	LP 300/S, LP 400/420, LP 450, LP 700, LP 800	546 nm
	LP 1	560 nm
Measuring temperature:	+15 °C to +37 °C	

Procedure

A. Measuring wavelength 520 nm

Pipette into a cuvette test LKM 226:	
	Analysis
Sample	10 µl
Mix and measure A(0). Remove the protective foil, rotate the cap and then reclose the cuvette. Dissolve the contents by shaking. Insert the cuvette immediately into the photometer again for measurement.	

Please also note the instrument-specific instructions.

B. Measuring wavelength 546 nm or 560 nm

Pipette into a cuvette test LKM 226:	
	Analysis
Serum/plasma	20 µl
Mix, rotate the start cap and reclose the cuvette. Dissolve the contents by shaking. After 5-15 minutes read one reagent blank (unused cuvette) and measure all analyses against it.	

Calculation
Cholesterol concentration c in serum/plasma: $c = F_I \times A$ [mg/dl]
$c = F_{II} \times A$ [mmol/l]

Wavelength	F_I [mg/dl]	F_{II} [mmol/l]
546 nm	418	10.8
560 nm	534	13.8

Conversion into mg/dl: mmol/l x 38.7 = mg/dl

Performance description
The test is suited for the determination of cholesterol activity up to values from 25.9 mmol/l (1000 mg/dl).

Clinical interpretation
The cholesterol concentration depends upon e.g. age, sex, nutrition and ethnic group. The following values are recommended to assess the risk factor of hypercholesterolemia[2)]:

	mmol/l	mg/dl
Desirable	< 5.2	< 200
Borderline	5.2 - 6.1	200 - 239
High	≥ 6.2	≥ 240

Notes
The buffer solution contains sodium azide (<0.1 %) as preserving agent. Do not swallow and avoid contact with skin and mucous membranes.

Quality control
You can use LT-SYS (article no. LCQ 710) and all control sera with cholesterol target values of these method.

References
1) Allain CC, et al. Clin Chem 1974; 20:470
2) NCEP Expert Panel. Arch Intern Med 1988; 148:36

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4/05

Dr. Lange Cuvette Test
LKM 227

DR LANGE

Triglycerides

GPO-PAP method

Article No. LKM 227
40 cuvettes

Method
Enzymatic colorimetric test, GPO-PAP method[1)]
without considering free glycerol

Triglycerides + H_2O —(Lipase)→ Glycerol + fatty acids

Glycerol + ATP —(GK)→ Glycerol-3-phosphate + ADP

Glycerol-3-phosphate + O_2 —(GPO)→ Dihydroxyacetone phosphate + H_2O_2

H_2O_2 + 2.4-Dichlorophenol + 4-Aminophenazone —(Peroxidase)→ Quinonimine dye + 2 H_2O + HCl

The determination at 520 nm can directly employ whole blood. It is immediately and completely hemolysed by the buffer solution.
Results obtained from blood samples also refer to the plasma concentrations as the erythrocytes content of each individual sample is taken into account. Calculation is automatically done by a special evaluation program.

Active components

1. Buffer solution (prepipetted in LKM 227)	Pipes buffer, pH 7.5:	50	mmol/l
	Lipoproteinlipase:	≥ 7.5	kU/l
	Peroxidase:	≥ 0.5	kU/l
	Magnesium acetate:	11	mmol/l
	2.4-Dichlorphenol:	4	mmol/l
	Detergent:	0.15	%
	Sodium cholate:	3.5	mmol/l
	Sodium azide:	< 0.1	%
2. Starting reagent (in start caps)	4-Aminophenazone:	0.15	mmol/l
	ATP:	2	mmol/l
	GPO:	≥ 3.5	kU/l
	Peroxidase:	≥ 3.5	kU/l
	Glycerokinase:	1	kU/l

Classification
1. No hazardous product as specified in Directive 67/548/EEC.
2. Hazardous product as specified in Directive 67/548/EEC and 1999/45/EEC. Classified as harmful with R 42/43 (may cause sensitization by inhalation and skin contact).

Preparation of reagent and stability
The reagents are ready-to-use.
The reagents are stable at +2 °C to +8 °C until the expiry date stated on the packing.
Please note that the lyophilized starting reagent is moisture-sensitive!
At high ambient temperature bring cuvettes with the start caps to room temperature before removing the foil.

Sample material
Whole blood, serum, heparinized or EDTA plasma
Transfer blood samples immediately into buffer solution.
Stability of triglycerides in closed LKM 227 (without starting reagent) cuvette before measurement:
at +2 °C to +8 °C: 8 hours
at +15 °C to +30 °C: 4 hours

Measuring conditions

Wavelength:	LP 20, LP 400/420, LP 800 (appropriate software for whole blood needed)	520 nm
	LP 6, LP 6 A/S	Hg 546 nm
	LP 300/S, LP 400/420, LP 450, LP 700, LP 800	546 nm
	LP 1	560 nm
Measuring temperature:	+15 °C to +37 °C	

Procedure

A. Measuring wavelength 520 nm

Pipette into a cuvette test LKM 227:	
	Analysis
Sample	10 µl
Insert the cuvette in the photometer and measure absorbance A(0). Remove the protective foil, rotate the cap and then reclose the cuvette. Dissolve the contents by shaking. Insert the cuvette immediately into the photometer again for measurement.	

Please also note the instrument-specific instructions.

B. Measuring wavelength 546 nm or 560 nm

Pipette into a cuvette test LKM 227:	
	Analysis
Serum/plasma	20 µl
Mix, rotate the start cap and reclose the cuvette. Dissolve the contents by shaking. After 3-10 minutes read one reagent blank (unused cuvette) and measure all analyses against it.	

Calculation
Triglycerides concentration c in serum:
$c = F_I \times A$ [mg/dl]
$c = F_{II} \times A$ [mmol/l]

Wavelength	F_I [mg/dl]	F_{II} [mmol/l]
546 nm	690	7.87
560 nm	897	10.22

Conversion into mg/dl: mmol/l x 87.7 = mg/dl

Performance description
The test is suited for the determination of triglycerides activity up to values from 1000 resp. 2000 mg/dl.
Wavelength 520 nm (10 µl sample): (22.8 mmol/l) 2000 mg/dl
Wavelength 546 nm (20 µl sample): (11.4 mmol/l) 1000 mg/dl
When exceeding these values, specimens should be diluted with physiological saline 1+1 and repeat determination. Multiply the result by 2.

Reference ranges
Recommended normal fasting levels are <1.71 mmol/l.
Levels >2.29 mmol/l should be considered as increased.[2)]

Notes
The buffer solution contains sodium azide (<0.1 %) as preserving agent. Do not swallow and avoid contact with skin and mucous membranes.

Quality control
You can use LT-SYS (article no. LCQ 710) and all control sera with triglycerides target values of these method.

References

1) Fossati P, Prencipe L. Clin Chem 1982; 28:2077
2) Schettler G, Nüssel E. Arb Med Soz Med Präv Med 1975; 10:25

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APPENDIX 11

Participant Number	Pre exercise	Week 1			Week 2			Week 3			Week 4			Post exercise	Pre control	Post control
		1	2	3	1	2	3	1	2	3	1	2	3			
✓ 1	21.06.10 ✓ 12pm	29th JUNE	30th JUNE	2nd JULY	5th JULY	7th JULY	9th JULY	12th JULY	14th JULY	15th JULY	19th JULY	21st JULY	22nd JULY	26.07.10 ✓ 1:00 pm	20.05.10 ✓ 12pm	17.06.10 ✓ 12 pm
✓ 2	17.05.10 ✓ 12pm	24th MAY	26th MAY	28th MAY	1st JUNE	2nd JUNE	[redacted]	7th JUNE	9th JUNE	10th JUNE	14th JUNE	15th JUNE	16th JUNE	21.06.10 ✓ 12pm	5.07.10 ✓ 11:30am	28.7.10 29.07.10 11:30 ~~12:30pm~~
✓ 3	17.05.10 ✓ 4.15pm	20th MAY	21st MAY	[redacted]	26th MAY	27th MAY	28th MAY	2nd JUNE	3rd JUNE	4th JUNE	7th JUNE	9th JUNE	9th JUNE	10.06.10 ? 4pm	1.07.10 ✓ 4:15pm	28.07.10 ✓ 4:00pm
✓ 4	3.06.10 ✓ 1pm	7th JUNE	8th JUNE	9th JUNE	15th JUNE	[redacted]	18th JUNE	23rd JUNE	24th JUNE	25th JUNE	29th JUNE	[redacted]	1st JULY	8.07.10 12:30 4pm	21.07.10 ✓ 11.30	11.08.10 ✓ 4.00
✓ 5	7.06.10 ✓	9th JUNE	10th JUNE	11th JUNE	14th JUNE	16th JUNE	17th JUNE	23rd JUNE	24th JUNE	25th JUNE	28th JUNE	1st JULY	2nd JULY	7.07.10 ✓ 9:30am	15.07.10 ✓ 4pm	13.08.10 ✓ 9am
✓ 6	22.06.10 ✓ 10am	28th JUNE	[redacted]	2nd JULY	5th JULY	7th JULY	9th JULY	13th JULY	14th JULY	16th JULY	19th JULY	20th JULY	21st JULY	26.07.10 ✓ 1:15pm	18.05.10 ✓ 9am ✡	18.06.10 ✓ 9 am ✡
✓ 7	25.06.10 ✓ 10am	28th JUNE	30th JUNE	2nd JULY	[redacted]	8th JULY	9th JULY	13th JULY	14th JULY	[redacted]	[redacted]	22nd JULY	23rd JULY	2.08.10 ✓ 1:30 pm	18.05.10 ✓ 9am	15.06.10 ✓ 9am
✓ 8	21.05.10 ✓ 11am	25th MAY	25th MAY	28th MAY	1st JUNE	3rd JUNE	[redacted]	[redacted]	10th JUNE	11th JUNE	14th JUNE	17th JUNE	18th JUNE	21.06.10 ✓ 3pm	5.07.10 ✓ 11am	23.07.10 ✓ 11am
✓ 9	28.06.10 ✓ 12pm	29th JUNE	30th JUNE	1st JULY	3rd JULY	6th JULY	7th JULY	12th JULY	13th JULY	14th JULY	19th JULY	21st JULY	22nd JULY	26.07.10 ✓ 1pm	20.05.10 ✓ 12pm	28.06.10 ✓ 12 PM
✓ 10	21.05.10 ✓ 1pm	24th MAY	26th MAY	28th MAY	1st JUNE	2nd JUNE	[redacted]	7th JUNE	8th JUNE	10th JUNE	14th JUNE	15th JUNE	16th JUNE	21.06.10 ✓ 12pm	5.07.10 ✓ 11:30am	29.07.10 ✓ 11:30
✓ 11	24.05.10 ✓ 3.30pm	24th MAY	26th MAY	28th MAY	1st JUNE	2nd JUNE	3rd JUNE	9th JUNE	10th JUNE	11th JUNE	16th JUNE	17th JUNE	18th JUNE	21.06.10 ✓ 3pm	5.07.10 ✓ 11.00	2.08.10 ✓ 1:30pm
✓ 12	30.06.10 ✓	5th JULY	7th JULY	8th JULY	13th JULY	14th JULY	16th JULY	20th JULY	21st JULY	22nd JULY	28th JULY	29th JULY	30th JULY	2.08.10 ✓ 2:30pm	18.05.10 ✓ 10.30am	15.06.10 ✓ 9am
✓ 13	30.06.10 ✓	5th JULY	7th JULY	8th JULY	13th JULY	14th JULY	16th JULY	20th JULY	21st JULY	22nd JULY	28th JULY	29th JULY	30th JULY	2.08.10 ✓ 2:30 pm	18.05.10 ✓ 10.30am	15.06.10 ✓ 9am
✓ 14	21.07.10 ✓	21st JULY	22nd JULY	23rd JULY	28th JULY	29th JULY	30th JULY	4th AUG	5th AUG	6th AUG	10th AUG	11th AUG	12th AUG	13.08.10 ✓ 9.30am	7.06.10 ✓ 1.25pm	7.07.10 ✓ 9.00
✓ 15	2.06.10 ✓ 12:30 pm	8th JUNE	9th JUNE	10th JUNE	15th JUNE	16th JUNE	17th JUNE	21st JUNE	22nd JUNE	23rd JUNE	29th JUNE	30th JUNE	1st JULY	8.07.10 ✓ 12:30 pm	22.07.10 ✓ 12:30	1.09.10 ✓ 12:30
✓ 16	2.06.10 ✓ 12:30pm	8th JUNE	9th JUNE	10th JUNE	15th JUNE	16th JUNE	17th JUNE	21st JUNE	[redacted]	23rd JUNE	29th JUNE	30th JUNE	1st JULY	8.07.10 ✓ 12:30pm	[redacted]	1.09.10 ✓ 12:30
✓ 17	11.06.10 ✓ 1:00pm	16th JUNE	16th JUNE	17th JUNE	23rd JUNE	24th JUNE	25th JUNE	29th JUNE	1st JULY	2nd JULY	6th JULY	7th JULY	8th JULY	16.07.10 ✓ 3:30pm	30.07.10 ✓ 1 PM	2.09.10 ✓ 11.00

APPENDIX 12

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	PreExerciseTC	5.5486	7	1.19791	.45277
	PostExerciseTC	4.7429	7	.82133	.31044
Pair 2	PreControlTC	5.1357	7	1.00372	.37937
	PostControlTC	5.3729	7	.90855	.34340
Pair 3	PreExerciseTG	1.1349	7	.53820	.20342
	PostExerciseTG	1.0061	7	.62916	.23780
Pair 4	PreControlTG	.9906	7	.29764	.11250
	PostControlTG	1.0393	7	.23273	.08797
Pair 5	PreExerciseHDL	1.2414	7	.32921	.12443
	PostExerciseHDL	2.1400	7	.77326	.29227
Pair 6	PreControlHDL	1.3471	7	.38629	.14601
	PostControlHDL	1.4314	7	.37280	.14091
Pair 7	PreExerciseLDL	2.8557	7	1.08523	.41018
	PostExerciseLDL	2.6929	7	1.07060	.40465
Pair 8	PreControlLDL	2.7300	7	.76746	.29007
	PostControlLDL	2.8929	7	.84253	.31845

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	PreExerciseTC & PostExerciseTC	7	.683	.091
Pair 2	PreControlTC & PostControlTC	7	.964	.000
Pair 3	PreExerciseTG & PostExerciseTG	7	.659	.108
Pair 4	PreControlTG & PostControlTG	7	.988	.000
Pair 5	PreExerciseHDL & PostExerciseHDL	7	.770	.043
Pair 6	PreControlHDL & PostControlHDL	7	.954	.001
Pair 7	PreExerciseLDL & PostExerciseLDL	7	.717	.070
Pair 8	PreControlLDL & PostControlLDL	7	.821	.024

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	PreExerciseTC - PostExerciseTC	.80571	.87527	.33082	-.00377	1.61520	2.436	6	.051
Pair 2	PreControlTC - PostControlTC	-.23714	.27476	.10385	-.49125	.01696	-2.284	6	.062
Pair 3	PreExerciseTG - PostExerciseTG	.12871	.48920	.18490	-.32372	.58115	.696	6	.512
Pair 4	PreControlTG - PostControlTG	-.04871	.07658	.02894	-.11954	.02211	-1.683	6	.143
Pair 5	PreExerciseHDL - PostExerciseHDL	-.89857	.56079	.21196	-1.41721	-.37993	-4.239	6	.005
Pair 6	PreControlHDL - PostControlHDL	-.08429	.11588	.04380	-.19146	.02289	-1.924	6	.103
Pair 7	PreExerciseLDL - PostExerciseLDL	.16286	.81043	.30631	-.58666	.91238	.532	6	.614
Pair 8	PreControlLDL - PostControlLDL	-.16286	.48730	.18418	-.61353	.28782	-.884	6	.411

Paired Samples Test

		Paired Differences					t	Df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	SBP_Pre_E - SBP_Post_E	6.7857	6.7507	2.5515	.5424	13.0290	2.659	6	.038
Pair 2	SBP_Pre_C - SBP_Post_C	-19.4286	38.5253	14.5612	-55.0586	16.2014	-1.334	6	.231
Pair 3	DBP_Pre_E - DBP_Post_E	-.5000	9.3274	3.5254	-9.1264	8.1264	-.142	6	.892
Pair 4	DBP_Pre_C - DBP_Post_C	-14.7143	31.2221	11.8009	-43.5899	14.1614	-1.247	6	.259

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Fat_Percent_Pre_E	34.517	6	13.3939	5.4681
	Fat_Percent_Post_E	33.100	6	12.5319	5.1161
Pair 2	Fat_Percent_Pre_C	31.775	4	14.4590	7.2295
	Fat_Percent_Post_C	32.100	4	17.9114	8.9557
Pair 3	FatFree_Percent_Pre_E	65.483	6	13.3939	5.4681
	FatFree_Percent_Post_E	66.900	6	12.5319	5.1161
Pair 4	FatFree_Percent_Pre_C	68.175	4	14.4654	7.2327
	FatFree_Percent_Post_C	67.900	4	17.9114	8.9557
Pair 5	TotalMass_kg_Pre_E	74.214	7	8.0172	3.0302
	TotalMass_kg_Post_E	75.043	7	8.4069	3.1775
Pair 6	TotalMass_kg_Pre_C	73.020	5	9.3663	4.1887
	TotalMass_kg_Post_C	73.620	5	9.1040	4.0714

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Fat_Percent_Pre_E & Fat_Percent_Post_E	6	.993	.000
Pair 2	Fat_Percent_Pre_C & Fat_Percent_Post_C	4	.997	.003
Pair 3	FatFree_Percent_Pre_E & FatFree_Percent_Post_E	6	.993	.000
Pair 4	FatFree_Percent_Pre_C & FatFree_Percent_Post_C	4	.997	.003
Pair 5	TotalMass_kg_Pre_E & TotalMass_kg_Post_E	7	.944	.001
Pair 6	TotalMass_kg_Pre_C & TotalMass_kg_Post_C	5	.991	.001

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	Fat_Percent_Pre_E - Fat_Percent_Post_E	1.4167	1.7804	.7268	-.4517	3.2850	1.949	5	.109
Pair 2	Fat_Percent_Pre_C - Fat_Percent_Post_C	-.3250	3.6527	1.8264	-6.1373	5.4873	-.178	3	.870
Pair 3	FatFree_Percent_Pre_E - FatFree_Percent_Post_E	-1.4167	1.7804	.7268	-3.2850	.4517	-1.949	5	.109
Pair 4	FatFree_Percent_Pre_C - FatFree_Percent_Post_C	.2750	3.6619	1.8309	-5.5518	6.1018	.150	3	.890
Pair 5	TotalMass_kg_Pre_E - TotalMass_kg_Post_E	-.8286	2.7723	1.0478	-3.3925	1.7354	-.791	6	.459
Pair 6	TotalMass_kg_Pre_C - TotalMass_kg_Post_C	-.6000	1.2981	.5805	-2.2118	1.0118	-1.034	4	.360

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	PreExercise	28.6714	7	5.09814	1.92692
	PostExercise	35.0987	7	8.82394	3.33514
Pair 2	PreControl	42.9236	6	19.20438	7.84015
	PostControl	33.8791	6	7.55597	3.08471

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	PreExercise & PostExercise	7	.618	.139
Pair 2	PreControl & PostControl	6	.433	.391

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	PreExercise - PostExercise	-6.42727	6.94750	2.62591	-12.85264	-.00191	-2.448	6	.050
Pair 2	PreControl - PostControl	9.04452	17.32593	7.07328	-9.13793	27.22697	1.279	5	.257

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	PreLDLHDLFair & PostLDLHDLFair	7	.590	.163
Pair 2	PreLDLHDLGood & PostLDLHDLGood	8	.748	.033
Pair 3	PreHDLLDLFairCon & PostHDLLDLFairCon	7	.808	.028
Pair 4	PreLDLHDLGoodCon & PostLDLHDLGoodCon	8	-.755	.030

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	PreLDLHDLFair - PostLDLHDLFair	.97963	.74293	.28080	.29253	1.66672	3.489	6	.013
Pair 2	PreLDLHDLGood - PostLDLHDLGood	.28780	.34114	.12061	.00259	.57300	2.386	7	.048
Pair 3	PreHDLLDLFairCon - PostHDLLDLFairCon	.00249	.51322	.19398	-.47216	.47714	.013	6	.990
Pair 4	PreLDLHDLGoodCon - PostLDLHDLGoodCon	.29301	.39019	.13795	-.03320	.61922	2.124	7	.071

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	METExePre	4.7286	7	.56484	.21349
	METSExePost	3.7043	7	.40873	.15449
Pair 2	METSRestPre	.8429	7	.43150	.16309
	METSRestPost	1.0414	7	.95293	.36017
Pair 3	HRExePre	124.5714	7	12.48809	4.72005
	HRExePost	115.5714	7	16.58169	6.26729
Pair 4	HRRestPre	70.7143	7	4.78589	1.80890
	HRRestPost	68.1429	7	7.17469	2.71178
Pair 5	VO2KGExePre	16.4286	7	1.90238	.71903
	VO2KGExePost	12.9500	7	1.48520	.56135
Pair 6	VO2KGRestPre	2.7143	7	1.79947	.68014
	VO2KGRestPost	2.1767	7	.90379	.34160
Pair 7	EE_Exercise_Pre	334.1465	7	39.28983	14.85016
	EE_Exercise_Post	263.4943	7	24.28463	9.17873
Pair 8	EE_Rest_Pre	60.2644	7	32.13338	12.14528
	EE_Rest_Post	49.0321	7	15.62020	5.90388

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	METExePre & METSExePost	7	.045	.924
Pair 2	METSRestPre & METSRestPost	7	-.046	.921
Pair 3	HRExePre & HRExePost	7	.801	.031
Pair 4	HRRestPre & HRRestPost	7	.516	.236
Pair 5	VO2KGExePre & VO2KGExePost	7	.059	.900
Pair 6	VO2KGRestPre & VO2KGRestPost	7	-.033	.945
Pair 7	EE_Exercise_Pre & EE_Exercise_Post	7	.006	.990
Pair 8	EE_Rest_Pre & EE_Rest_Post	7	.121	.796

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	METExePre - METSExePost	1.02429	.68220	.25785	.39336	1.65521	3.972	6	.007
Pair 2	METSRestPre - METSRestPost	-.19857	1.06415	.40221	-1.18275	.78560	-.494	6	.639
Pair 3	HRExePre - HRExePost	9.00000	9.96661	3.76702	-.21758	18.21758	2.389	6	.054
Pair 4	HRRestPre - HRRestPost	2.57143	6.24118	2.35895	-3.20070	8.34356	1.090	6	.318
Pair 5	VO2KGExePre - VO2KGExePost	3.47857	2.34341	.88572	1.31128	5.64586	3.927	6	.008
Pair 6	VO2KGRestPre - VO2KGRestPost	.53757	2.03988	.77100	-1.34900	2.42415	.697	6	.512
Pair 7	EE_Exercise_Pre - EE_Exercise_Post	70.65225	46.06410	17.41059	28.05006	113.25444	4.058	6	.007
Pair 8	EE_Rest_Pre - EE_Rest_Post	11.23239	33.98940	12.84679	-20.20256	42.66734	.874	6	.416

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	PreExercise	1.2857	7	.48795	.18443
	PostExercise	2.0000	7	.81650	.30861
Pair 2	PreControl	1.6667	6	.81650	.33333
	PostControl	1.8333	6	.75277	.30732

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	PreExercise & PostExercise	7	.000	1.000
Pair 2	PreControl & PostControl	6	.542	.266

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	PreExercise - PostExercise	-.71429	.95119	.35952	-1.59399	.16542	-1.987	6	.094
Pair 2	PreControl - PostControl	-.16667	.75277	.30732	-.95665	.62332	-.542	5	.611

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	METSRestPre	.8429	7	.43150	.16309
	METExePre	4.7286	7	.56484	.21349
Pair 2	HRRestPre	70.7143	7	4.78589	1.80890
	HRExePre	124.5714	7	12.48809	4.72005
Pair 3	VO2KGRestPre	2.7143	7	1.79947	.68014
	VO2KGExePre	16.4286	7	1.90238	.71903
Pair 4	EE_Rest_Pre	60.2644	7	32.13338	12.14528
	EE_Exercise_Pre	334.1465	7	39.28983	14.85016
Pair 5	METSRestPost	1.0414	7	.95293	.36017
	METSExePost	3.7043	7	.40873	.15449
Pair 6	HRRestPost	68.1429	7	7.17469	2.71178
	HRExePost	115.5714	7	16.58169	6.26729
Pair 7	VO2KGRestPost	2.1767	7	.90379	.34160
	VO2KGExePost	12.9500	7	1.48520	.56135
Pair 8	EE_Rest_Post	49.0321	7	15.62020	5.90388
	EE_Exercise_Post	263.4943	7	24.28463	9.17873

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	METSRestPre & METExePre	7	.589	.164
Pair 2	HRRestPre & HRExePre	7	.714	.071
Pair 3	VO2KGRestPre & VO2KGExePre	7	.675	.096
Pair 4	EE_Rest_Pre & EE_Exercise_Pre	7	.796	.032
Pair 5	METSRestPost & METSExePost	7	-.499	.255
Pair 6	HRRestPost & HRExePost	7	.681	.092
Pair 7	VO2KGRestPost & VO2KGExePost	7	.014	.976
Pair 8	EE_Rest_Post & EE_Exercise_Post	7	.338	.459

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	SBP_Pre_E	118.625	8	10.7030	3.7841
	SBP_Post_E	109.125	8	7.2789	2.5735
Pair 2	SBP_Pre_C	119.250	8	7.6765	2.7141
	SBP_Post_C	111.750	8	7.5829	2.6810
Pair 3	DBP_Pre_E	77.250	8	9.6732	3.4200
	DBP_Post_E	72.188	8	5.0634	1.7902
Pair 4	DBP_Pre_C	81.688	8	4.2421	1.4998
	DBP_Post_C	73.813	8	8.7421	3.0908

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	SBP_Pre_E & SBP_Post_E	8	.431	.287
Pair 2	SBP_Pre_C & SBP_Post_C	8	.392	.337
Pair 3	DBP_Pre_E & DBP_Post_E	8	.277	.507
Pair 4	DBP_Pre_C & DBP_Post_C	8	.410	.313

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	SBP_Pre_E - SBP_Post_E	9.5000	10.0214	3.5431	1.1219	17.8781	2.681	7	.031
Pair 2	SBP_Pre_C - SBP_Post_C	7.5000	8.4134	2.9746	.4662	14.5338	2.521	7	.040
Pair 3	DBP_Pre_E - DBP_Post_E	5.0625	9.5970	3.3931	-2.9608	13.0858	1.492	7	.179
Pair 4	DBP_Pre_C - DBP_Post_C	7.8750	7.9989	2.8280	1.1878	14.5622	2.785	7	.027

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Fat_Percent_Pre_E	27.629	7	12.7916	4.8348
	Fat_Percent_Post_E	27.986	7	11.9910	4.5322
Pair 2	Fat_Percent_Pre_C	27.213	8	11.7005	4.1367
	Fat_Percent_Post_C	26.987	8	11.5470	4.0825
Pair 3	FatFree_Percent_Pre_E	72.371	7	12.7916	4.8348
	FatFree_Percent_Post_E	72.014	7	11.9910	4.5322
Pair 4	FatFree_Percent_Pre_C	72.788	8	11.7005	4.1367
	FatFree_Percent_Post_C	73.013	8	11.5470	4.0825
Pair 5	TotalMass_kg_Pre_E	63.943	7	5.8366	2.2060
	TotalMass_kg_Post_E	63.557	7	6.3416	2.3969
Pair 6	TotalMass_kg_Pre_C	63.075	8	6.3281	2.2373
	TotalMass_kg_Post_C	63.238	8	6.3644	2.2502

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Fat_Percent_Pre_E & Fat_Percent_Post_E	7	.979	.000
Pair 2	Fat_Percent_Pre_C & Fat_Percent_Post_C	8	.951	.000
Pair 3	FatFree_Percent_Pre_E & FatFree_Percent_Post_E	7	.979	.000
Pair 4	FatFree_Percent_Pre_C & FatFree_Percent_Post_C	8	.951	.000
Pair 5	TotalMass_kg_Pre_E & TotalMass_kg_Post_E	7	.996	.000
Pair 6	TotalMass_kg_Pre_C & TotalMass_kg_Post_C	8	.996	.000

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	Fat_Percent_Pre_E - Fat_Percent_Post_E	-.3571	2.6894	1.0165	-2.8444	2.1301	-.351	6	.737
Pair 2	Fat_Percent_Pre_C - Fat_Percent_Post_C	.2250	3.6378	1.2862	-2.8163	3.2663	.175	7	.866
Pair 3	FatFree_Percent_Pre_E - FatFree_Percent_Post_E	.3571	2.6894	1.0165	-2.1301	2.8444	.351	6	.737
Pair 4	FatFree_Percent_Pre_C - FatFree_Percent_Post_C	-.2250	3.6378	1.2862	-3.2663	2.8163	-.175	7	.866
Pair 5	TotalMass_kg_Pre_E - TotalMass_kg_Post_E	.3857	.7313	.2764	-.2906	1.0620	1.396	6	.212
Pair 6	TotalMass_kg_Pre_C - TotalMass_kg_Post_C	-.1625	.5780	.2044	-.6457	.3207	-.795	7	.453

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	PreExercise	43.3900	8	6.29571	2.22587
	PostExercise	57.1426	8	15.40763	5.44742
Pair 2	PreControl	50.4193	8	8.92279	3.15468
	PostControl	55.9005	8	12.25591	4.33312

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	PreExercise & PostExercise	8	.228	.587
Pair 2	PreControl & PostControl	8	.267	.523

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	PreExercise - PostExercise	-13.75258	15.25863	5.39474	-26.50911	-.99604	-2.549	7	.038
Pair 2	PreControl - PostControl	-5.48111	13.09413	4.62948	-16.42808	5.46586	-1.184	7	.275

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	METSRestPre	1.2750	8	.77229	.27304
	METSRestPost	.9338	8	.15847	.05603
Pair 2	METExePre	5.0375	8	1.10704	.39140
	METSExePost	4.3213	8	1.04316	.36881
Pair 3	HRRestPre	75.8750	8	12.15892	4.29883
	HRRestPost	71.1250	8	5.48862	1.94052
Pair 4	HRExePre	116.7500	8	16.22828	5.73756
	HRExePost	109.8473	8	9.25973	3.27381
Pair 5	VO2KGRestPre	4.3750	8	2.61520	.92461
	VO2KGRestPost	3.4166	8	1.15227	.40739
Pair 6	VO2KGExePre	17.7500	8	3.84522	1.35949
	VO2KGExePost	15.1375	8	3.69843	1.30759
Pair 7	EE_Rest_Pre	92.9727	8	54.84226	19.38967
	EE_Rest_Post	66.2078	8	34.43265	12.17378
Pair 8	EE_Exercise_Pre	360.6935	8	77.10697	27.26143
	EE_Exercise_Post	316.2261	8	75.55323	26.71210

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	METSRestPre & METSRestPost	8	.158	.708
Pair 2	METExePre & METSExePost	8	.589	.124
Pair 3	HRRestPre & HRRestPost	8	.142	.738
Pair 4	HRExePre & HRExePost	8	.561	.148
Pair 5	VO2KGRestPre & VO2KGRestPost	8	.257	.539
Pair 6	VO2KGExePre & VO2KGExePost	8	.623	.099
Pair 7	EE_Rest_Pre & EE_Rest_Post	8	.249	.552
Pair 8	EE_Exercise_Pre & EE_Exercise_Post	8	.556	.153

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	METSRestPre - METSRestPost	.34125	.76338	.26990	-.29696	.97946	1.264	7	.247
Pair 2	METExePre - METSExePost	.71625	.97620	.34514	-.09987	1.53237	2.075	7	.077
Pair 3	HRRestPre - HRRestPost	4.75000	12.61235	4.45914	-5.79419	15.29419	1.065	7	.322
Pair 4	HRExePre - HRExePost	6.90275	13.42980	4.74815	-4.32485	18.13035	1.454	7	.189
Pair 5	VO2KGRestPre - VO2KGRestPost	.95838	2.57246	.90950	-1.19225	3.10900	1.054	7	.327
Pair 6	VO2KGExePre - VO2KGExePost	2.61250	3.27760	1.15881	-.12764	5.35264	2.254	7	.059
Pair 7	EE_Rest_Pre - EE_Rest_Post	26.76495	57.03450	20.16474	-20.91709	74.44698	1.327	7	.226
Pair 8	EE_Exercise_Pre - EE_Exercise_Post	44.46741	71.95557	25.44013	-15.68895	104.62377	1.748	7	.124

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	PreExercise	2.5000	8	.53452	.18898
	PostExercise	2.3750	8	.51755	.18298
Pair 2	PreControl	2.1250	8	.35355	.12500
	PostControl	2.2500	8	.88641	.31339

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	PreExercise & PostExercise	8	.775	.024
Pair 2	PreControl & PostControl	8	.342	.407

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	PreExercise - PostExercise	.12500	.35355	.12500	-.17058	.42058	1.000	7	.351
Pair 2	PreControl - PostControl	-.12500	.83452	.29505	-.82268	.57268	-.424	7	.685

APPENDIX 13

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Fat_Percent_Pre_E	28.833	6	15.2930	6.2433
	Fat_Percent_Pre_C	28.433	6	13.9609	5.6995
Pair 2	Fat_kg_Pre_E	20.050	6	10.6577	4.3510
	Fat_kg_Pre_C	19.600	6	9.6503	3.9397
Pair 3	FatFree_Percent_Pre_E	71.167	6	15.2930	6.2433
	FatFree_Percent_Pre_C	71.533	6	13.9745	5.7051
Pair 4	FatFree_kg_Pre_E	48.967	6	9.7770	3.9915
	FatFree_kg_Pre_C	48.750	6	8.7817	3.5851
Pair 5	TotalMass_kg_Pre_E	69.017	6	4.0425	1.6503
	TotalMass_kg_Pre_C	68.350	6	3.8775	1.5830

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Fat_Percent_Pre_E & Fat_Percent_Pre_C	6	.992	.000
Pair 2	Fat_kg_Pre_E & Fat_kg_Pre_C	6	.992	.000
Pair 3	FatFree_Percent_Pre_E & FatFree_Percent_Pre_C	6	.992	.000
Pair 4	FatFree_kg_Pre_E & FatFree_kg_Pre_C	6	.987	.000
Pair 5	TotalMass_kg_Pre_E & TotalMass_kg_Pre_C	6	.975	.001

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	Fat_Percent_Pre_E - Fat_Percent_Pre_C	.4000	2.3134	.9445	-2.0278	2.8278	.424	5	.690
Pair 2	Fat_kg_Pre_E - Fat_kg_Pre_C	.4500	1.6610	.6781	-1.2931	2.1931	.664	5	.536
Pair 3	FatFree_Percent_Pre_E - FatFree_Percent_Pre_C	-.3667	2.3166	.9458	-2.7978	2.0645	-.388	5	.714
Pair 4	FatFree_kg_Pre_E - FatFree_kg_Pre_C	.2167	1.8104	.7391	-1.6833	2.1166	.293	5	.781
Pair 5	TotalMass_kg_Pre_E - TotalMass_kg_Pre_C	.6667	.9026	.3685	-.2805	1.6139	1.809	5	.130

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	PreExercise	37.3388	8	9.98661	3.53080
	PreControl	41.0710	8	8.99614	3.18061

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	PreExercise & PreControl	8	.325	.433

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	PreExercise	2.0000	8	.75593	.26726
	PreControl	1.8750	8	.64087	.22658

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	PreExercise & PreControl	8	.885	.004

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	PreExercise - PreControl	.12500	.35355	.12500	-.17058	.42058	1.000	7	.351

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	SBP_Pre_E	118.625	8	11.1795	3.9526
	SBP_Pre_C	119.750	8	7.6485	2.7042
Pair 2	DBP_Pre_E	77.938	8	10.9982	3.8884
	DBP_Pre_C	83.438	8	6.4056	2.2647

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	SBP_Pre_E & SBP_Pre_C	8	.826	.012
Pair 2	DBP_Pre_E & DBP_Pre_C	8	.105	.804

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	SBP_Pre_E - SBP_Pre_C	-1.1250	6.5014	2.2986	-6.5603	4.3103	-.489	7	.639
Pair 2	DBP_Pre_E - DBP_Pre_C	-5.5000	12.1302	4.2887	-15.6411	4.6411	-1.282	7	.241

APPENDIX 14

Group Statistics

	Fitness	N	Mean	Std. Deviation	Std. Error Mean
HDLChange	Fair and Below	7	.8986	.56079	.21196
	Good and Below	8	.3188	.24180	.08549

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
HDLChange	Equal variances assumed	6.524	.024	2.666	13	.019	.57982	.21751	.10992	1.04973
	Equal variances not assumed			2.537	7.931	.035	.57982	.22855	.05199	1.10765