The Impact of Four Weeks of High Intensity Interval Training on Cardiac Autonomic Function in Physically Inactive Males and Females

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Contents

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Abstract

Introduction: Physical inactivity is the fourth largest risk factor of premature mortality, responsible for 6% of deaths each year worldwide. Autonomic dysfunction has shown to be an indicator of cardiovascular diseases. High intensity interval training has shown to reduce traditional cardiovascular risk factors in less time than traditional aerobic exercise training. However, the adaptations following a high intensity interval training programme to the cardiac autonomic nervous system have not yet been fully researched.

Method: Using an independent-measures design, 45 physically inactive males and females (22.8 ± 2.7 years) were placed into either a high intensity interval training or control group. The subjects in the high intensity interval training group completed 3 x 30 seconds all out maximal cycle sprints, 3 times a week on a Wattbike cycle ergometer at a resistance of 7.5% of their body weight, with two minutes of active recovery between bouts, for four weeks. Cardiac autonomic function was measured using the task force monitor.

Results: This study found that four weeks of high intensity interval training led to resting heart rate significantly reducing (65.59 ± 10.15 to 63.05 ± 13.42 $b \cdot min^{-1}$; $p < 0.05$) and significant improvements in total power spectrum density (498.8 ± 239.8 ms2; $p < 0.05$) and high frequency power (389.4 ± 123 ms^2; $p < 0.01$) against the control group. There was a non-significant increase in low frequency power (189.7 ± 91.2 ms^2) and no significant reduction in LF/HF ratio compared to the control group.

Conclusion: Four weeks of high intensity interval training led to a significant reduction in resting heart rate and a significant improvement to cardiac autonomic function.

1.0: Introduction

The cardiovascular system (or circulatory system) is comprised of three components: the heart, blood and the blood vessels (Tortora & Derrickson, 2011), and has three main functions. The first function is to transport oxygen and nutrients, such as glucose, vitamins, amino acids and fatty acids, to the cells of the body and to transport toxic metabolic waste products and carbon dioxide out of the cells (Levick, 2010). Secondly, the cardiovascular system regulates body temperature and pH levels as heat produced by the working muscles and from metabolic activity is directed to the skin (Tortora & Derrickson, 2011). Lastly, the cardiovascular system protects the body from infection as it produces immune cells called lymphocytes, which originate in the bone marrow and help protect the body against antigens (Gavin, 2003). The cardiovascular system relies on the cardiac cycle functioning at optimal levels. The cardiac cycle consists of all the mechanical and electrical events that take place during a single heartbeat (Levick, 2010). However, individuals who are physically inactive are at a greater risk of having an unhealthy cardiovascular system and leading to a higher risk of developing cardiovascular disease (Kobirumaki-Shimozawa, Inoue, Shintani, Oyama, Terui *et al* 2014).

Physical inactivity is the fourth largest risk factor of mortality, causing 6% of all deaths each year worldwide (Lee, Shiroma, Lobelo, Puska, Blair *et al* 2012). Lee *et al* (2012) estimate that 6 - 10% of all major non-communicable diseases, such as type 2 diabetes, CHD, obesity and some forms of cancer, are related to a lack of physical activity. Physical inactivity has been attributed to nearly 240,000 individuals developing chronic diseases each year (Martin, Church, Thompson, Earnest & Blair, 2009). Due to the high morbidity and mortality rates

associated with physical inactivity, the indirect cost of physical inactivity in England is estimated to be £8.2 billion, including expenditure on medical costs and work absences (Allender, Foster, Scarborough, & Rayner, 2007). Physical inactivity is one of the leading risk factors for cardiovascular disease (Lee *et al* 2012).

Cardiovascular disease is known to be the leading cause of death in developing nations, exceeding infectious diseases (Okwuosa, Lewsey, Adesiyun, Blumenthal & Yancy, 2016). Furthermore, in 2011, cardiovascular disease caused 17.3 million deaths worldwide (Laslett, Alagona, Clark, Drozda, Saldivar, *et al.* 2012) and it is predicted that by the year 2030 cardiovascular disease will be responsible for approximately 25 million deaths per year (WHO, 2011). Cardiovascular disease was the second leading cause of death in the United Kingdom in 2014, responsible for 27% of all deaths, with medical services, such as the NHS, incurring costs of £4.3 billion (Townsend, Bhatnagar, Wilkins Wickramasinghe & Rayner, 2015). The main form of cardiovascular disease is coronary artery disease, which is the leading single cause of death in the UK, causing 73,680 deaths in 2012 and 45% of all cardiovascular disease deaths (Townsend, Williams, Bhatnagar, Wickramasinghe & Rayner, 2014). The remaining cardiovascular disease deaths are from strokes, responsible for 25%, and peripheral arterial and aortic diseases, responsible for the remaining 30% of deaths (Townsend *et al* 2014). There are many risk factors for developing cardiovascular disease, such as hypertension, smoking, drinking and obesity. However, a significant risk factor for cardiovascular disease is cardiac autonomic dysfunction (Lahiri, Kannankeril & Goldberger, 2008).

The autonomic nervous system is part of the peripheral nervous system, which is vital for maintaining homeostasis within the body by controlling involuntary internal function, such

as heart rate, breathing, blood pressure and digestion (Nesvold, Fagerland, Davanger, Ellingsen, Solberg *et al* 2012). Therefore, a cardiac autonomic imbalance is when there is a depressed level of parasympathetic activity and/or a higher level of sympathetic activity in the cardiac autonomic nervous system; this is known as autonomic dysfunction (Hautala, Mäkikallio, Kiviniemi, Laukkanen, Nissilä *et al* 2003). Autonomic dysfunction has been evidenced to be a better predicator of mortality rates than conventional measures, such as left ventricle size due to progressive heart failure (Routledge, Chowdhary & Townend, 2002). This is because, the greater the autonomic imbalance gets, the higher the risk of developing a chronic disease (Lahiri *et al* 2008). Whereas, left ventricle size predicator of mortality has been inconsistent due to an increase in relative wall thickness in the absence of left ventricle hypertrophy, meaning results are not always accurate (Shah & Pfeffer, 2014). The autonomic imbalance can be investigated by examining heart rate variability (Tsai, Wei-Chu, Kuo & Ming-Fong, 2006).

Heart rate variability is the beat-to-beat variability between the intervals of the heartbeat, meaning it is a marker of neural outflow modulation. Therefore, it is a reproducible and non-invasive measure of quantitatively assessing cardiac autonomic function (Pumprla, Howorka, Groves, Chester & Nolan, 2002; Floras, 2009). Neural influences can cause the rhythm of the myocardium to change. These neural influences originate in the cardiovascular centre in the medulla oblongata and flow through the parasympathetic and sympathetic nervous systems. The parasympathetic and sympathetic nervous systems function almost antagonistically but use different structural pathways and transmitter systems to maintain homeostasis (Kingsley & Figueroa, 2014). By examining heart rate variability, information can be gathered concerning the amount of activity occurring through the parasympathetic and sympathetic

nervous systems. Decreased heart rate variability indicates the presence of autonomic dysfunction due to reduced parasympathetic control of the heart rate, increasing the risk of cardiovascular disease and all causes of morbidity and mortality (Pop-Busui, Evans, Gerstein, Fonseca, Fleg *et al* 2010). Research shows that physical activity can be used to improve an individual's autonomic control if they have an autonomic imbalance or autonomic dysfunction (Thayer, Yamamoto & Brosschot, 2010). Physical activity is evidenced to significantly increase heart rate variability and decrease resting heart rate, which reduces the risk of developing cardiovascular disease (Jurca, Church, Morss, Jordan & Earnest, 2004).

Physical activity has also been found to improve anxiety and depression levels, reduce constipation, delay cognitive impairment and improve sleep (Nelson, Rejeski, Blair, Duncan, Judge *et al* 2007). A study by Wen, Wai, Tsai, Yang, Cheng *et al* (2011) found that individuals who exercised for 90 minutes a week reduced the risk of premature mortality by 14%, when compared to those who were physically inactive. Furthermore, they found that, for every additional 15 minutes of physical activity, all causes of mortality were further reduced by 4%. Despite this, Hallal, Andersen, Bull, Guthold, Haskell *et al* (2012) suggest that around 31% of the world's population are not meeting the recommended guidelines for physical activity, with many citing a lack of enjoyment and time as reasons why they do not exercise regularly. The World Health Organisation recommends that individuals should participate in either 150 minutes of moderate intensity exercise, using approximately 3-6 METs, a week, such as cycling or pushing a lawn mower; or 75 minutes of vigorous exercise, using above 6 METS, a week, such as running or swimming (WHO, 2011). However, the current physical activity guidelines are not evidence based (Weed, 2016) and they do not consider the effects of high intensity interval training (HIIT) programmes, which have shown to have equal or, in some

cases, greater health adaptations in significantly less time (Fletcher, Balady, Amsterdam, Chaitman, Eckel *et al* 2001).

Traditional aerobic exercise training (TAET) is continuous training at a moderate intensity, for a long period of time. In contrast, HIIT is exercising at a high intensity for short intervals, but for many repetitions. HIIT has seen to be more enjoyable due to its more varied nature and takes significantly less time than TAET, which can be deemed to be boring and tedious (Tjønna, Lee, Rognmo, Stølen, Bye *et al* 2008). HIIT has been shown to significantly improve aerobic capacity, reduce blood pressure and vascular heath when compared to TAET, thereby having a greater impact in reducing the risk factors of cardiovascular disease (Gibala, Little, MacDonald & Hawley, 2012). However, as HIIT has not been around as long as TAET, the effects of a HIIT programme have not been fully researched in all physiological parameters. Nevertheless, TAET has shown to significantly improve autonomic function, whereas adaptions following a HIIT programme are less well known.

2.0: Literature Review

2.1: *Physical Activity*

Physical activity is any bodily movement produced by a skeletal muscle that uses energy and is imperative to staying healthy (Janssen & LeBlanc, 2010). Physical activity has been shown to cause significant reductions in non-communicable diseases, thus preventing premature mortality (Knight, 2012). It has been evidenced that there is an inverse linear relationship between the amount of physical activity that an individual engages in and all

causes of mortality, in both the young and old and in males and females (Lee & Skerrett, 2001). A meta-analysis conducted by Samitz, Egger & Zwahlen (2011) analysed 1,338,143 participants from 80 studies. They found that individuals who adhered to the minimum recommended amount of exercise per week had a reduction, ranging from 7-14%, in all causes of mortality. Therefore, it has been estimated that if physical activity levels increased by 10%, this would prevent 500,000 premature deaths a year worldwide as it would inhibit cardio-metabolic diseases (Lee, Shiroma, Lobelo, Puska, Blair *et al* 2012).

Regular physical activity has been shown to have significant health benefits, causing the following physiological adaptations to occur; improved maximal aerobic consumption ($VO_{2\ max}$) (Whyte, Gill, & Cathcart, 2010) and vascular function (Warburton, Nicol & Bredin, 2006); reduced blood pressure (Metcalfe, Babraj, Fawkner, & Vollaard, 2012; Fletcher, Ades, Kligfield, Arena, Balady *et al* 2013) and body composition (Zhou, Zhang, Liu, Guo, Wang *et al* 2017); improved heart rate variability; and reduced resting heart rate (Tjønna, Stølen, Bye, Volden, Slørdahl *et al* 2009; Fletcher, Ades, Kligfield, Arena, Balady *et al* 2013), which contributes to a lower risk of premature mortality. A meta-analysis by Maddison, Jiang, Foley, Scragg, Direito *et al* (2015), investigated 33 studies, discovering that regular exercise was responsible for a 35% reduction in the risk of cardiovascular disease and a 33% reduction in the risk of mortality compared to individuals who were physically inactive. Research suggests that physically inactive individuals who improve cardiac autonomic function through physical activity improved heart rate variability and resting heart rate, preventing cardiac events (Grant, Viljoen, Janse van Rensburg & Wood, 2012).

Cardiac autonomic function can be significantly enhanced by regular physical exercise (Goldsmith, Bloomfield, & Rosenwinkel, 2000; Hautala, Mäkikallio, Kiviniemi, Laukkanen, Nissilä *et al* 2003). Laing, Gluckman, Weinberg, Lahiri, Ng *et al* (2011) state that physical activity reduces catecholamine levels at rest in the blood and urine, causing a decrease in sympathetic nervous system activity. Fletcher *et al* (2013) suggest that healthy individuals who regularly participate in physical activity have higher parasympathetic activity than physically inactive individuals, so are therefore at a lower risk of developing a non-communicable disease. Thus, individuals who partake in physical activity are likely to have a greater vagal modulation of heart rate and improved heart rate variability Jurca, Church, Morss, Jordan & Earnest (2004).

When questioned about why individuals do not meet these guidelines, most people stated due to a lack of time as they have other commitments, such as work and looking after their children, while others stated they do not enjoy exercise as they find running boring (Bartlett, Close, MacLaren, Gregson, Drust *et al* 2011). Therefore, retention and adherence to TAET is extremely poor. Bartlett *et al* (2011) found that around 50% do not adhere to training programmes. Feedback from participants who have undertaken both HIIT and TAET stated that HIIT was more enjoyable due to its more varied nature in contrast to TAET, which was deemed to be boring (Tjønna, Leinan, Bartnes, Jenssen, Gibala *et al* 2013). Despite HIIT being a more favourable form of physical activity in terms of variety and time, Bartlett *et al* (2011) found that HIIT has not been directly recommended by the World Health Organisation, despite it resulting in similar, and, in some cases, greater physiological adaptations than TAET (Little, Gillen, Percival, Safdar, Tarnopolsky *et al* 2011). Ramos, Dalleck, Tjonna, Beetham & Coombes, (2015) conducted a meta-analysis comparing the effects of HIIT to TAET. They used

seven studies, with the most common form of HIIT being four intervals of four minutes (4 x 4 HIIT) at 95% HR_{peak}, with three minutes of active recovery between bouts, and TAET being a 30 - 45 minute run at 60% of HR_{peak}. Depending on the study the training intervention varied from 12 – 16 weeks. They found that HIIT led to greater improvements than TAET in a number of physiological parameters. Vascular function, assessed via brachial artery flow-mediated dilation (FMD), improved by 2.26% more in HIIT compared to TAET ($P < 0.05$), with HIIT improving FMD by 4.31%, compared to TAET at 2.15%. HIIT was also more effective at improving $VO_{2\ peak}$ as HIIT increased it by 6% more than TAET, with HIIT averaging a 15% improvement and TAET averaging a 9% improvement. It was also found that individuals who undertook a HIIT programme were more likely to see positive adaptations in oxidative stress, inflammation, and insulin sensitivity compared to TAET. This is supported by Jung, Bourne, Beauchamp, Robinson & Little (2015), who found that HIIT leads to greater improvements than TAET in $VO_{2\ peak,}$ cardiac output and stroke volume and has a greater reduction in systolic blood pressure. The HIIT group showed a greater time efficiency as the total training time was 75 minutes a week, compared to the TAET group that exercised for 150 minutes a week. These results show that HIIT is more time effective than TAET as it leads to greater physiological adaptations with less time commitment.

Physical inactivity rates are high in the UK with more than 20 million people not meeting minimum physical activity recommendations (Townsend, 2012)). Physical activity is one of the markers for health, so it is important to find a type of exercise which is more realistic for people to meet. As less time is required to complete a HIIT session, this may produce an increase in adoption and adherence of HIIT compared to a TAET programmes (Shiraev & Barclay, 2012). This could therefore reduce the levels of physically inactive

individuals and could be a key strategy in improving lifestyles and reducing morbidity and mortality. As HIIT been proven to reduce traditional cardiovascular risk factors, it is important to see what affect it can have upon the cardiac autonomic nervous system. However, it is not currently known what the minimum amount of physical activity is needed to make significant health changes. Therefore, this study will aim to address the minimum amount of time need to cause significant health changes.

2.2: *Cardiac Autonomic Nervous System*

The cardiac autonomic nervous system works by using the parasympathetic and sympathetic nervous systems to maintain homeostasis within the body. The sinoatrial node and atrioventricular node in the atria are densely innervated by both parasympathetic and sympathetic fibres, which affect the firing rate of the pacemaker cells, whereas the ventricles only receive sympathetic fibres (Kingsley & Figueroa, 2014). The sympathetic nervous system is known as the 'fight or flight' system as it is predominantly used during a state of crisis, so can therefore increase heart rate and contractility of the myocardium and decrease venous return. This means more blood can be ejected by the left ventricle per minute, as it can spontaneously activate a cardiovascular response from the body (Sztajzel, 2004). This occurs through the secretion of catecholamines adrenaline and noradrenaline through the cardioaccelerator nerves. These processes are known as the chronotropic effect and inotropic effect (Kobirumaki-Shimozawa *et al* 2014). The release of these catecholamines causes the sinoatrial node to depolarise at a quicker rate, meaning the heart beats faster. The catecholamines are then reabsorbed and metabolised slowly, causing sympathetic activation to occur over an extended period of time (Myers, Tan, Abella, Aleti, & Froelicher, 2007). This

increases myocardial inotropy, lusitropy, chronotrophy and dromotropy as the firing rate of the sinoatrial node increases, so there is a greater conduction velocity of the atrioventricular node, increasing stroke volume and cardiac output as the myocardium has greater contractibility (Thomas, 2011).

Parasympathetic nervous system activity slows the heart rate and reduces contractility of the myocardium through the synaptic release of acetylcholine, through the vagus X nerve (Olshansky, Sabbah, Hauptman & Colucci, 2008). The release of acetylcholine retards the rate of sinus discharge, which inhibits sympathetic nervous system activity, slowing the heart rate down. Therefore, parasympathetic nervous system activity most commonly occurs while the body is at rest (Sztajzel, 2004). Parasympathetic nervous system activity occurs very quickly over a short period, allowing it to control the heart on a beat-to-beat basis (Pumprla *et al* 2002). The parasympathetic nervous system predominates over sympathetic activation at rest in healthy individuals (Olshansky *et al* 2008). However, individuals who are physically inactive may have higher sympathetic activation at rest, causing there to be an imbalance in their autonomic function. Having a lower parasympathetic activation and higher sympathetic activation at rest can result in having a greater catecholamine levels due to a cytotoxic effect, meaning they will be negative effects of the myocardium (Rubart & Zipes, 2005). Therefore, the myocardium will have to work harder, increasing the strain upon the cardiovascular system. This can put that individual at a higher risk of having cardiovascular disease (Kobirumaki-Shimozawa *et al* 2014).

The autonomic nervous system is responsible for the systemic circulatory regulation of the body and does so by effecting changes in heart rate, arterial blood pressure and

vascular tone (Levick, 2010). Sandercock, Bromley & Brodie (2005) showed that modulation of the cardiac autonomic nervous system improved following an exercise training programme. Therefore, physical activity can reduce resting heart rate, arterial blood pressure and vascular tone (Gibala & McGee, 2008). After partaking in reoccurring physical activity, the autonomic nervous system begins to work more predominantly, using the parasympathetic nervous system at rest as there is an increase in vagal modulation of the heart rate due to a decrease in sympathetic nervous system activity. Regular physical activity has also shown to affect the arterial baroreflex, this is responsible for short term regulation of blood pressure and is an effective indicator of vagal activity (Thayer *et al* 2010). After exercise the, arterial baroreflex is 'reset' causing arterial blood pressure to reduce, as a result there is a reduction in sympathetic activity and increased parasympathetic tone (Sheriff, 2006; Fletcher *et al* 2013).

Frequency domain analysis of heart rate variability has shown to be a reliable way of measuring the cardiac autonomic nervous system activity as it works at different frequencies. These frequencies are: low frequency and high frequency, which reflects both parasympathetic and sympathetic nervous system activation (Ruediger, Seibt, Scheuch, Krause & Alam, 2004). Previous research has shown that high frequency oscillation (0.15 – 0.4 Hz) of R–R intervals, mainly reflects parasympathetic modulation. Low frequency oscillation (0.04 – 0.15 Hz) of R–R intervals predominately reflects sympathetic modulation, although parasympathetic modulation can also be detected (Bilchick & Berger, 2006; Hillebrand, Gast, de Mutsert, Swenne, Jukema *et al* 2013). By analysing these frequencies, tonic baseline autonomic function can be measured (Routledge *et al* 2002). Normalised units of heart rate variability can also be used to quantify the modulation of the sympathetic and

parasympathetic branches of the cardiac autonomic nervous system. This represents the relative values of either low frequency or high frequency of the power components in proportion to the total power minus very low frequency (Burr, 2007). To examine the changing relationship between the sympathetic and parasympathetic nervous system activities, LF/HF ratio can be assessed, which represents the cardiac sympathovagal balance (Ditor, Kamath, MacDonald, Bugaresti, McCartney *et al* 2005).

High heart rate variability, if the intervals of the heartbeat are of varying length, is considered normal as the heart responds to the demands of the body (Hillebrand *et al* 2013). Evidence suggests that individuals with autonomic dysfunction can be associated with an impairment in parasympathetic cardiac control and/or high levels of sympathetic activity, which can cause an increased risk of cardiovascular disease (Thayer *et al* 2010; Routledge, Campbell, McFetridge-Durdle & Bacon, 2010) and atherosclerosis due to an impairment in endothelial function (Kaufman, Kaiser, Steinberger & Dengel, 2007). Research suggests that this is caused by extended catecholamine stimulation, promoting more low-density lipoproteins to be absorbed into the endothelium (Zhang, Chalothorn, Jackson, Lee & Faber, 2004). Goldsmith, Bigger, Steinman & Fleiss (1992) conducted a cross-sectional study of autonomic function in healthy men. They found that individuals who regularly exercised had higher parasympathetic activity than individuals who were not physically active. Previous research has found that heart rate variability is inversely related to physical inactivity and systolic blood pressure (Zhou, Xie, Wang & Yang, 2012). This is supported by Farah, Barros, Balagopal & Ritti-Dias (2014) who found, in a cross-sectional study of 1,152 adolescent males, that poor heart rate variability is associated with cardiovascular risk factors. They found that 14% of adolescent boys, who had two or more risk factors of cardiovascular disease had

significantly lower heart rate variability than individuals with one or no cardiovascular risk factors. Studies by both Monahan, Dinenno, Tanaka, Clevenger, DeSouza *et al* (2000) and Rosenwinkel, Bloomfield, Arwady & Goldsmith (2001) suggest that physical activity has a positive effect on autonomic function, with an exercise training programme significantly improving heart rate variability, meaning individuals are at a lower risk of suffering from cardiovascular disease.

It is known that both low and high frequency reflect both parasympathetic and sympathetic nervous system activation. Therefore, it is important to measure these parameters to see what impact HITT has on these and how it affects heart rate variability. However, from the research it is still unclear what very low frequency measures (Usui & Nishida, 2017).

2.3: *Traditional Aerobic Exercise Training*

The most common form of TAET is moderate intensity continuous training, which evidence has shown to promote beneficial physiological adaptations to improve health (Azevedo & Dos Santos, 2014). This form of exercise training is highly recommended by the World Health Organisation for benefiting both healthy and diseased populations (Weston, Wisløff & Coombes, 2014). A TAET session takes place over an extended period of time, lasting between 30 – 60 minutes, using 3 – 6 METS (Thompson, Buchner & Pina, 2003). Garber, Blissmer, Deschenes, Franklin, Lamonte, (2011) conducted a research study looking at the quality and quantity of exercise needed to maintain cardiorespiratory health in adults. They found that if participating in a TAET session, individuals needed to train at 64 – 76% of their maximum heart rate, which is 46 – 64 % of their $VO_{2\ max}$. Therefore, participating in regular

physical activity for the recommended time and intensity for TAET can help prevent all causes of morbidity and mortality (Knight, 2012).

Research has found that undertaking a TAET programme has significant health benefits for individuals. Schjerve, Tyldum, Tjønna, Stølen, Loennechen *et al* (2008) conducted a study looking at the effects of continuous training in overweight but healthy middle-aged males, specifically on their $VO_{2\ max}$ and body weight, as both are strong independent factors for cardiovascular mortality (Lee, Sui, Ortega, Kim, Church *et al* 2010). A study by Schjerve *et al* (2008) continuously trained participants at 60% HR_{peak} for 47 minutes. They found that $VO_{2\ max}$ significantly improved by 16% ($P < 0.01$). They also found that BMI decreased from 36.7 ± 1.4 to 35.6 ± 1.4 kg/m^2 ($P < 0.007$) and body fat percentage decreased from 43.6% ± 1.5 to 42.51 ± 1.5% ($P < 0.03$). These results are supported by Cornelissin & Fagard (2005), who conducted a meta-analysis of 3936 participants from 72 studies. They examined the effects of endurance training on normotensive, pre-hypertensive and hypertensive individuals. Both males and females were studied, with the age ranging from 21 – 83 years. They found that both resting systolic ($p < 0.001$) and diastolic ($p < 0.001$) blood pressure significantly reduced. People's weight ($p < 0.001$), body fat percentage ($p < 0.05$) and resting heart rate ($p < 0.001$) all significantly reduced. Finally, they found the $VO_{2\ max}$ significantly improved ($p < 0.0001$). These reductions and improvements signify a healthier cardiovascular system, preventing early morbidity and mortality (Keteyian, Brawner, Savage, Ehrman *et al* 2008). It has been suggested that TAET can also lead to adaptations in autonomic function.

TAET training has also been shown to improve cardiac autonomic function. Jurca *et al* (2004) conducted a study looking at the effects of continuous training on cardiac autonomic

function in sedentary females. They used 88 females with n=49 in the exercise training group and n=39 in the control group. The participants trained at 50% $VO_{2\ peak}$ for 60 minutes, three times a week for eight weeks. They found that high-frequency spectral power significantly increased from 4.73 ± 1.03 ms^2 to 5.23 ± 1.07 ms^2 ($P < 0.002$); low-frequency spectral power significantly increased from 4.72 ± 0.78 ms^2 to 5.13 ± 0.86 ms^2 ($P < 0.03$); and total frequency spectral power significantly improved from 6.40 ± 0.72 ms^2 to 6.79 ± 0.79 ms^2 ($P < 0.001$). It is suggested that these improvements reduce the risk of mortality, as individuals with improved heart rate variability are less likely to develop risk factors associated with cardiovascular disease. The improvements in heart rate variability could have been caused by an enhancement in parasympathetic tone, as there was greater high frequency power and low frequency power recorded after the training programme. This is supported by Voulgari, Pagoni, Vinik & Poirier (2013), who conducted a review of exercise intervention on cardiac autonomic function. They found that TAET improved cardiac autonomic function and heart rate variability as parasympathetic activity becomes more predominant at rest, due to a rise in vagal modulation of the heart rate. Neither of the studies reported the LF/HF ratio; therefore, cardiac sympathovagal balance cannot be interpreted. These results show that after a TAET programme, positive physiological adaptations to the autonomic nervous system were seen. This could have been caused by greater parasympathetic activity, leading to greater vagal modulation of the heart rate (Hautala, Kiviniemi & Tulppo, 2009).

Although physical activity through TAET has been proven to have many health benefits, not all studies have found TAET programmes to significantly improve cardiac autonomic function. Both Perini, Fisher, Veicsteinas & Pendergast (2002) and Loimaala, Huikuri, Oja, Pasanen & Vuori (2000) found that heart rate variability parameters were not

statistically significant following a TAET programme. Participants in Perini *et al.'s* (2002) study were required to be over 70 years old and trained at 40% - 60% of $VO_{2\ max}$ for eight weeks, and in Loimaala *et al's* (2000) study, subjects had to be males aged 35 – 55 and trained at 55% of $VO_{2\ max}$ for five months. After the completion of their TAET programme, neither study saw any significant improvements in heart rate variability parameters or resting heart rate. However, significant improvement was seen in $VO_{2\ max}$, improving from 37.3 ± 4.0 mL · kg^{-1} · min^{-1} to 42.9 ± 6.1 mL · kg^{-1} · min^{-1} ($p < 0.05$). These findings are supported by Figueroa, Baynard, Fernhall, Carhart & Kanaley (2007), who found that after 16 weeks of TAET, there were no significant improvements in high frequency or low frequency power or in LF/HF ratio in obese individuals or individuals with type 2 diabetes. Figueroa *et al* (2007) reported a LF/HF ratio which was not significant, suggesting that parasympathetic activation had increased equally in in both low frequency and high frequency power, while there was no change in sympathetic activity. These findings illustrate that a TAET programme can evoke significant health adaptations but has no beneficial effect on autonomic function.

Research on TAET and autonomic function has produced mixed results, with some studies finding that a TAET programme does result in significant adaptations to autonomic function via heart rate variability (Jurca *et al* 2004; Voulgari *et al* 2013). However, a number of studies found that TAET did not evoke a significant change in autonomic function (Loimaala *et al* 2000; Perini *et al* 2002; Figueroa *et al* 2007). Adherence to long term TAET programmes has proved to be poor in many different populations, with many attesting this to the time commitment needed to complete a session and a lack of enjoyment when exercising for extended periods of time (Stutts, 2002). Therefore, the current guidelines of physical activity

may be unrealistic for some populations, which contributes to physical inactivity. A training programme that could be more beneficial for these populations could be HIIT.

The literature has shown that TAET improves many different health parameters; however, this is poorly adhered to for many different reasons, but most commonly due to time and the tedious nature of TAET. What is not known is what the minimum amount of time is need to incur significant health adaptations. To explore this further HIIT will be examined.

2.4: *High Intensity Interval Training*

A training method that has been shown to have similar, and, in some cases, better results than TAET, in significantly less time, is HIIT (Whyte *et al* 2010). HIIT is training at 77 – 95% of maximal heart rate, which is 64 – 90% of $VO_{2\ max}$, with periods of low intensity for active recovery (Garber *et al* 2011). HIIT has a vigorous exercise time of under 10 minutes, with sessions lasting less than 30 minutes, using over 6 METs (Thompson *et al* 2003). There are many different protocols for HIIT; however, the most common consists of three to seven bouts of 30 seconds of cycling sprint Wingate tests, three times a week. Two to five minutes of active recovery is normally taken between bouts (Sloth, Sloth, Overgaard & Dalgas, 2013). Feedback from participants who have undertaken both HIIT and TAET suggests that HIIT is more enjoyable due to its more varied nature, in contrast to TAET, which was deemed to be boring (Tjønna *et al* 2008). HIIT may therefore be recommended as a form of physical activity that is more enjoyable and requires less time to elicit physiological adaptations to the cardiovascular system and improve cardiac autonomic function to lessen the risk of early morbidity and mortality (Gillen & Gibala, 2013).

HIIT has been shown to lead to significant physiological adaptations when performed as part of a training programme. For example, Gillen & Gibala (2013) found that three, 30 minute HIIT sessions a week, comprising of no more than 10 minutes of intense exercise time, significantly improved health. HIIT has also shown to be a safe training method, as the protocol can be easily adapted to include longer or short intense intervals or longer or shorter periods of active recovery (Millar, Rakobowchuk, McCartney & MacDonald, 2009). However, it has been suggested that the duration of the intense intervals is the factor that causes the physiological adaptations (Kemi & Wisløff, 2010).

The vigorous intensity of HIIT has been shown to challenge the metabolic system. During HIIT, there is a larger production of catecholamines, increased sympathetic activity and enhanced anaerobic metabolism in comparison to TAET. When an individual is performing HIIT, their metabolic rate is raised for a longer period than when conducting TAET (Oliveira, Alvarenga, Mattos, Silva, Rezende *et al* 2013). This has been suggested as a reason why HIIT has been shown to lead to greater physiological adaptations in $VO_{2\ max}$, blood pressure, lactate level, endothelial function and autonomic function in a short duration of exercise time (Guiraud, Labrunee, Gaucher-Cazalis, Despas, Meyer *et al* 2013).

The effects of HIIT on $VO_{2\ max}$ and blood pressure have been documented. Ramos *et al* (2015) conducted a meta-analysis comparing the effects of HIIT to TAET. They used seven studies, with the most common form of HIIT being four intervals of four minutes (4 x 4 HIIT) at 95% HR_{peak}, with three minutes of active recovery between bouts. The TAET group did a 30 - 45 minute run at 60% of HR_{peak}. They found that HIIT led to greater improvements than TAET in a number of areas. Vascular function, assessed via brachial artery flow-mediated

dilation (FMD), improved by 2.16% more in HIIT compared to TAET ($P < 0.05$), with HIIT improving FMD by 4.31%, compared to TAET at 2.15%. HIIT was also more effective at improving $VO_{2\ peak}$, increasing it by 6% more than TAET, with HIIT averaging a 15% improvement and TAET averaging a 9% improvement. It was also found that individuals who undertook a HIIT programme were more likely to see greater positive effects on oxidative stress, inflammation, and insulin sensitivity compared to TAET. This is supported by Jung *et al* (2015), who found that HIIT leads to greater improvements than TAET in $VO_{2\ peak,}$ cardiac output and stroke volume and has a greater reduction in systolic blood pressure ($p < 0.05$). They suggest that hypotrophy of the myocardium could have occurred, which could increase the cardiac contractility, leading to a greater stroke volume. As circulatory function has been improved, the myocardium can pump more blood out to the working muscles per minute, thus improving VO_2 peak. These findings show that HIIT significantly improves VO_2 peak, vascular function and cardiac output, compared to TAET, in these physiological parameters. The subjects in the HIIT group are at lower risk of cardiovascular disease as HIIT has led to a greater improvement in cardiorespiratory fitness than TAET, meaning they have superior health protection.

Another meta-analysis that compares the effects of HIIT vs TAET was conducted by Weston *et al* (2014). Weston *et al* (2014) included ten papers in their meta-analysis that studied the training effects on cardiometabolic chronic disease patients for whom a poor lifestyle was considered the main cause of the disease. In total, the trials consisted of 237 patients, with 137 being in the HIIT programme and 136 being in the TAET programme. They reported that HIIT led to greater adaptations, even though a HIIT training session was shorter than a TAET session. They reported that VO_2 peak improved by 19.4% in the HIIT group, going

from 22.5 mL · kg^{-1} · min^{-1} to 27.9 mL · kg^{-1} · min^{-1}, compared to a 10.3% increase in the TAET, going from 22.6 mL · kg^{-1} · min^{-1} to 25.2 mL · kg^{-1} · min^{-1} ($P < 0.001$). They also found that high-density lipoproteins, maximum rate of calcium ions reuptake, nitric oxide and cardiac output all significantly improved more in the HIIT group compared to the TAET ($p < 0.05$). In addition, systolic and diastolic blood pressure, oxidative stress and fatty acid synthase are reduced more in the HIIT group than the TAET group. After the training programme finished, they found that quality of life was greater in the HIIT group than the TAET, as anxiety and depression were further reduced. Another study that found similar results is Milanović, Sporiš & Weston (2015). They compared the effects of TAET, HIIT and non-exercising groups in physically inactive, but healthy middle-aged males. They found that both HIIT and TAET elicited large improvements in $VO_{2\ peak}$ when compared to the non-training group. However, the HIIT group improved $VO_{2\ peak}$ to a greater extent. It was suggested that HIIT led to a greater enhancement of oxidative capacity, meaning there was an increase in pyruvate dehydrogenase and citrate synthase. This led to a rise in mitochondrial mass, increasing the amount of ATP that can be produced, meaning the body can use a greater amount of oxygen, thus improving VO_2 peak. It was also found that HIIT had the greatest improvement in quality of life. These studies and meta-analysis have shown that HIIT is a more effective way of eliciting beneficial adaptations to improve health in a shorter amount of time than TAET.

There has been a great deal of research into whether a HIIT programme improves $VO_{2\ peak}$, as improving cardiorespiratory fitness has been shown to decrease all causes of morbidity and mortality (Bize, Johnson, & Plotnikoff, 2007). Sloth *et al* (2013) conducted a meta-analysis of 19 studies, looking at the effects of HIIT on healthy, overweight and obese individuals. The 19 studies used 190 subjects: 121 males and 69 females. The HIIT programmes lasted

between two to eight weeks, with two to three training sessions per week. Despite the low training volume, there were improvements in aerobic-based exercise performance. They found that HIIT significantly improved $VO_{2\ peak}$ ($p < 0.05$), with the lowest improvement in a study being 4.2% and the highest being 13.4%, meaning the average across the studies was a 7.9% improvement. They suggest that this could be due to central adaptations as there was greater oxygen availability as cardiac output increased, meaning functional capacity would also increase. Helgerud, Hoydal, Wang, Karlsen, Berg et al (2007) supported this study as they found that $VO_{2\ peak}$ significantly improved following a HIIT programme, from 55.5 ± 7.4 mL · kg^{-1} · min^{-1} to 60.4 ± 7.3 mL · kg^{-1} · min^{-1}; ($P < 0.001$), although this study was conducted on very fit individuals. HIIT has also been shown to improve $VO_{2\ peak}$ in unhealthy populations. Liou, Ho, Fildes & Ooi (2016) conducted a meta-analysis looking at the effects of HIIT and TAET on patients with coronary heart disease. They analysed ten studies with 472 patients in total, with 218 subjects performing HIIT and 254 performing TAET. They found that HIIT led to an increase of 1.78 mL · kg^{-1} · min^{-1} more than in TAET ($P = 0.009$). The effects of HIIT on $VO_{2\ peak}$ have been highly researched, showing improvements in both males and females, normal and overweight populations and in individuals with chronic diseases. It was suggested that HIIT led to the rapid stimulation of skeletal muscle, which increased the capillary density in the muscles, leading to a greater $VO_{2\ peak}$ (Duscha, Robbins, Jones, Kraus, Lye *et al* 2011). A meta-analysis by Barlow, DeFina, Radford, Berry, Cooper *et al* (2012) found that for everyone improvement in METs in aerobic capacity, which is 3.5 mL · kg^{-1} · min^{-1}, there is a 13% decrease in the risk of mortality and a 15% reduction in cardiovascular events. As HIIT leads to greater physiological adaptations in $VO_{2\ max}$ than TAET, there is a reduced risk of cardiovascular disease, indicating HIIT could provide superior health protection.

A study that assesses the effects of HIIT and TAET on individuals suffering from coronary artery disease is Conraads, Pattyn, De Maeyer, Beckers, Coeckelberghs *et al* (2015). They conducted a large multi-centred study over 12 weeks, when they recruited 200 middle-aged male and female subjects. The subjects were placed into either the HIIT group (N=100) or TAET group (N=100). The HIIT group trained at 95% HR_{peak} for four minutes, with three minutes of active recovery between each of the four sets. The TAET group trained at 70% HR_{peak} for 37 minutes, with a five minute warm up and cool down either side. Both training programmes took place on a cycle ergometer. After the training programmes had concluded, the HIIT group significantly improved $VO_{2\ max}$ by 22.7 ± 17.6%, increasing from 23.5 ± 5.7 mL. $kg^{-1.}$ min^{-1} to 28.6 ± 6.9 mL. $kg^{-1.}$ min^{-} ($p < 0.01$). The TAET group also showed significant improvements in $VO_{2\ max}$, increasing by 20.3 ± 15.3%, from 22.4 ± 5.6 to 26.8 ± 6.7 mL. $kg^{-1.}$ min^{-1} ($p < 0.01$). However, the results show that quality of life of the subjects similarly improved in both the HIIT and TAET groups, shown by the Short Form-12 questionnaire. The quantity of HDL cholesterol ratio also decreased, thus indicating that there was far less fatty material within the coronary arteries. Therefore, atheroma build up was reduced, meaning a lower mortality rate was predicted (Cornelissen & Fagard, 2005). This research has many strengths, including a large sample and being multi-centred, but despite this there are some limitations to the data. For example, there was a lack of long-term follow up to the study. This would have allowed the research to find out which training method, HIIT or TAET, had better event free survival rates as one of the training methods may have plateaued. Additionally, when comparing males and females, males showed greater participation rates, 180:20.

HIIT programmes have also shown to be effective at reducing blood pressure. Molmen-Hansen, Stolen, Tjonna, Aamot, Ekeberg *et al* (2012) investigated whether HIIT and

TAET reduced ambulatory blood pressure in hypertensive patients. They used 49 male and 39 female patients. After three weeks of training, they found that both systolic and diastolic blood pressure significantly reduced more in the HIIT group, compared to the TAET group. They found that systolic ambulatory blood pressure significantly reduced by 12 mmHg ($p < 0.001$) in the HIIT group and 4.5 mmHg ($p < 0.05$) in the TAET group. Diastolic ambulatory blood pressure was reduced by 8 mmHg ($p < 0.001$) in the HIIT group and 3.5 mmHg ($p = 0.02$) in TAET group. This is supported by Whyte *et al* (2010) who found, after two weeks of HIIT, systolic blood pressure reduced by 4.7%, from 127 ± 3 to 121 ± 3 ($p < 0.05$), in prehypertensive subjects. Paoli, Pacelli, Moro, Marcolin, Neri *et al* (2013) investigated the effects of HIIT on overweight, prehypertensive males. They found that systolic blood pressure significantly reduced after 12 weeks, from 138 ± 12 mmHg to 131 ± 9 mmHg ($P < 0.05$). Their research supports the findings of Molmen-Hansen *et al* (2012), whose subjects had a significant reduction in diastolic blood pressure of 85 ± 6 mmHg to 79 ± 7 mmHg ($P < 0.001$). Paoli *et al* (2013) suggest that the duration of the HIIT programme is important to see significant changes in diastolic blood pressure, which could explain why studies that have a shorter duration training programmes have not seen significant changes in blood pressure. Reduction in blood pressure can decrease the work load for the myocardium as there is an improvement in endothelium-dependent responses. This inhibits vasoconstriction, so the blood vessels are vasodilated, meaning there is reduced arterial stiffness, which is a known predictor of cardiovascular disease.

There is a breadth of literature stating that HIIT can significantly improve $VO_{2\ peak}$ and reduce blood pressure in all populations. Although, it has also been inferred that HIIT can improve cardiac autonomic function, the literature is limited and equivocal. Boutcher, Park,

Dunn, & Boutcher (2013) conducted a study to see if heart rate variability was affected by HIIT. They used 32 young overweight females, who were physically inactive (23 ± 4 year of age). The protocol the study used for HIIT was that participants exercised for 20 minutes. The HIIT intervention was made up of 10 minutes of intermittent sprinting on a cycle ergometer, with 8 seconds of sprint at 85% of HR_{peak}, followed by 12 seconds of active recovery, and five minutes warm up and cool down. 85% of HR_{peak} was calculated when each participant carried out a peak maximal oxygen uptake during the pre-testing. The HIIT programme was conducted over a 12-week period. They found that HIIT significantly improved high-frequency power in natural logarithm (0.6 In, $P<0.01$) compared to the control group. However, they found that there was a trend in the data, as there was a non-significant increase in low frequency power, going from 1033 ± 704 ms to 1407 ± 852 ms, and a non-significant, but a trend in the data, increase in high frequency power, going from 1932 ± 1736 ms to 3463 ± 2692 ms. It was further reported that HIIT significantly improved $VO_{2\,peak}$, improving from 27.3 ± 5.2 $mL \cdot kg^{-1} \cdot min^{-1}$ to 32.7 ± 5.2 $mL \cdot kg^{-1} \cdot min^{-1}$ ($P < 0.05$). This study shows that HIIT can improve heart rate variability as the findings suggest that there has been an enhancement in parasympathetic activity and/or a decrease in sympathetic activity, which is beneficial for cardiovascular health. Nevertheless, this study's research cannot be generalised to a male population as they only studied the adaptations of HIIT on physically inactive, overweight females.

Another study that examined the effects of HIIT on cardiac autonomic function is Heydari, Boutcher & Boutcher (2013). They used 38 male volunteers, who were non-smoking, physically inactive, but otherwise healthy individuals of 24.9 ± 4.3 years. Anyone who suffered from cardiovascular disease or a chronic disease was excluded from the study. The

participants were randomly assigned to either the HIIT or the control group. The training programme took place on a cycle ergometer over a 12-week period, with participants performing three HIIT sessions a week. The HIIT programme was eight seconds of cycle sprinting at 90% of HR_{peak} at a cadence of 120-130 revolutions per minute (RPM), followed by 12 seconds of active recovery at a cadence of 40 RPM. This was repeated for 20 minutes, with a five-minute warm up and cool down. They found the HIIT group significantly reduced their resting heart rate by 6.2 $b \cdot min^{-1}$ ($P < 0.001$), which was accompanied by a significant increase of 13.2 ml in stroke volume ($P < 0.001$). It was then postulated that this could have caused a significant improvement in $VO_{2\ peak}$, increasing by 5.2 $mL \cdot kg^{-1} \cdot min^{-1}$ ($P < 0.01$). The heart rate variability of the HIIT group also significantly improved in natural logarithm low frequency power by 0.4 In ($P < 0.5$) and high frequency power by 0.3 In ($P < 0.05$), whereas the control group saw no improvements. It was suggested that improvements in resting heart rate and heart rate variability were due to a combination of factors, including the reduction of intrinsic heart rate, improved parasympathetic tone and/or decrease in sympathetic tone. This supports Boutcher *et al* (2013), who found similar results in overweight females. However, this study fails to report the LF/HF ratio, so cardiac sympathovagal balance cannot be interpreted.

A study that looks into the effects of HIIT on heart rate variability in middle-aged men is Kiviniemi, Tulppo, Eskelinen, Savolainen, Kapanen *et al* (2014). They used males who had a BMI of between 15.5 to 30 $kg.m^2$ who were non-smokers and free from all chronic diseases. Participants recruited were between the ages of 40 - 55 and were split into either the HIIT group or the TAET group. Twenty six individuals volunteered for the study, with 13 people randomly being selected into each group. They participated in a two-week training

programme with four to six bouts of 30 seconds of all-out cycling effort, at a resistance of 7.5% of their body weight. Participants had four minutes of unloaded active recovery between bouts, when they could cycle at any intensity. The TAET group cycled for 40 – 60 minutes at a moderate continuous intensity of 60% of their peak workload. The researchers found that HIIT was more effective at prompting adaptations of heart rate variability than TAET. They reported that ambulatory measures of low frequency power significantly increased ($p = 0.024$) compared to the TAET group. They also found that both ambulatory high frequency power improved, and their 24-hour LF/HF ratio decreased after the HIIT programme; nevertheless, neither of these improvements or reductions were significant, although there was a trend in the results. However, it was found that daytime LF/HF ratio did significantly reduce in the HIIT group ($p = 0.032$). They suggested that this could have been caused by greater baroreflex-mediated modulation of the sinoatrial node throughout the day in the HIIT group. No significant adaptations were seen in the TAET group. It was suggested that an improvement in baroreflex sensitivity and vagal modulation could have caused the significant improvements in low frequency power. The study noted that as some of the measurements were conducted at home, they might not be as standardised as if the procedure was performed in the laboratory, meaning results may not be as valid. The sample size for this study is particularly small, which could have affected the results. Although the researchers found that many measurements improved and decreased how they expected after the training intervention, two weeks may not have been enough time to evoke significant adaptations (Deschenes & Kraemer, 2002).

The effects of HIIT on cardiac autonomic function have also been investigated in sedentary individuals with type 2 diabetes. Parpa, Michaelides & Brown (2009) used 14

sedentary, non-smoking participants, nine females and five males, who were 57 ± 7.6 years of age. Participants had no more than two cardiovascular risk factors in addition to their diabetes. The HIIT intervention was for 12 weeks with six, two-minute runs on a treadmill at 90% of HR_{peak}. They had two minutes of active recovery between bouts. After the intervention they found that heart rate variability significantly improved, from 52.70 ± 8.50 ms to 62.60 ± 11.00 ms ($p < 0.05$). Also, resting heart rate significantly reduced, from 76 ± 9 bpm to 69 ± 9 bpm ($p < 0.05$). Although this study showed that HIIT could significantly improve heart rate variability in sedentary individuals with type 2 diabetes, the study had a small sample size and had no control group so the HIIT may not have been the only reason for the significant changes. In addition, this study calculated heart rate variability manually from the mean R-R interval and its standard deviation from the electrocardiogram (ECG). This means they only used two of the time domain measures to calculate heart rate variability and did not use any frequency measures. Therefore, their calculations may not be accurate.

A study that investigated the effects of HIIT on heart rate variability to see if it was a safe intervention for rehabilitation in patients with chronic heart failure is Guiraud *et al* (2013). They used middle-aged patients aged 53 ± 12 years. 18 people volunteered for the study, 12 being male and six being female. This was a randomised crossover study, comparing the effects of a HIIT programme to a TAET programme. Participants had a BMI of 26.9 ± 7.1 $kg.m^2$ and left ventricular ejection fraction of 33 ± 10.7%. The HIIT sessions consisted of two sets of eight-minute, with each sprint interval being made up of 30 seconds at 100% of peak power output with 30 seconds of active recovery at any intensity. There were four minutes of active recovery between the two sets. The TAET consisted of 22 minutes on a cycle ergometer at 60% of peak power output. They found that HIIT led to greater adaptations than TAET, with

HIIT significantly reducing resting heart rate by 2.1 bpm ($p < 0.01$), whereas TAET only reduced resting heart rate by 1 bpm ($P < 0.01$). This is meaningful data as high resting heart rate is an independent predictor of cardiovascular disease. This shows that HITT can lead to greater health adaptations than TAET. HIIT also led to a greater significant improvement in high frequency power compared to the TAET group ($P < 0.01$). However, they found that low frequency power had a greater significant improvement in the TAET group compared to the HIIT group ($p < 0.05$). They also found that LF/HF ratio significantly reduced in both the HIIT and the TAET groups ($p < 0.01$). They suggested that adaptations were caused by an enhancement of vagal modulation due to there being a greater parasympathetic tone as high frequency power increases. They proposed that the exercise training can suppress Angiotensin II expression, as Angiotensin II has been shown to inhibit vagal activity and increase nitric oxide, which can inhibit sympathetic activity, increasing vagal control. However, they do not address why TAET may have led to greater improvements in low frequency power. These results suggest HIIT is an effective exercise programme to significantly improve cardiac autonomic function in patients with stable chronic heart failure, reducing the risks of all causes of mortality.

Another unhealthy population in which HIIT has been shown to significantly improve heart rate variability was examined in a study by Munk, Butt & Larsen (2010). They investigated the effects of a HIIT programme on heart rate variability in patients following percutaneous coronary intervention with stent implantation for angina pectoris. They used 38 males and females for the study, with 20 being used in the HIIT programme and 18 being used in the control. They conducted a six-month investigation using the HIIT protocol being four minutes at 95% HR_{peak} with three minutes of active recovery between the sets. They

found that resting heart rate over a 24-hour period significantly reduced by 4.9 $b \cdot min^{-1}$ ($p < 0.02$), whereas the control group had no significant change. They also found that both low frequency and high frequency power increased by 0.1 ms^2 and 0.4 ms^2 respectively in the HIIT group, although this was not significant. However, the 24-hour LF/HF ratio did significantly reduce by 0.6% ($P < 0.05$), meaning there was likely to be greater activation of parasympathetic tone and/or a reduction in sympathetic nervous system activity.

Although many studies have shown that HIIT significantly improves cardiac autonomic function, not all studies have found this to be the case. Gamelin, Baquet, Berthoin, Thevenet, Nourry *et al* (2009) investigated the effects of HIIT on prepubescent children to see if HIIT improved their heart rate variability. They used 52 male and female children with n=26 (15 boys and 11 girls) participating in HIIT and n=26 (14 boys and 12 girls) participating in the control group. The intervention took place over seven weeks, with the work to rest ratio varying from 5/15, 10/10, 15/10, 20/20 and 30/30 seconds intermittent runs each week as the children progressed through the programme. The children trained at an intensity of 100% to 190% of their maximal aerobic velocity. After the training programme, they found that both the HIIT and the control group had no significant changes in any heart rate variability parameter. However, the HIIT group did significantly improve $VO_{2\ peak}$ ($P < 0.05$). They suggested that there was no significant change in any heart rate variability parameter because they did not have the participants training at a high enough intensity to evoke significant change. This is supported by Currie, Rosen, Millar, McKelvie & MacDonald (2013), who also found that there was no significant improvement in any heart rate variability parameters after a HIIT programme in patients with coronary artery disease. They used 14 participants, splitting them into a HIIT group n=7 and TAET group n=7. To be included for the study patients had to

have a history of myocardial infarction and stenosis in at least one major coronary artery. The study lasted 12 weeks with two sessions a week. The TAET group did a 30-minute cycle at 60% of peak power output, with the duration increasing 10 minutes after every four weeks. The HIIT group did one minute of HIIT at 90% of peak power output, followed by one minute of active recovery at 10% of peak power output for 10 minutes. After the training programme finished, they found that $VO_{2\ peak}$ significantly improved in both the TAET group, 20.7 ± 6.0 mL · kg^{-1} · min^{-1} to 25.3 ± 5.1 mL · kg^{-1} · min^{-1} ($p < 0.05$), and in the HIIT group, 20.9 ± 3.1 mL · kg^{-1} · min^{-1} to 25.2±4.3 mL · kg^{-1} · min^{-1} ($p < 0.05$). However, they found that neither the HIIT nor the TAET groups elicited any significant changes in any heart rate variability parameter. They suggested that the lack of significant changes in autonomic nervous system activity could have been caused by the length of recovery between the coronary artery disease event and the beginning of training programme (five to six months).

Sloth *et al* (2013) recommend that each high intensity interval of the HIIT programme is 30 seconds all out maximal sprint. This is because it allows for maximal effort to be exerted for the full length of time and allows for maximal glycolytic power to build up (Micklewright, Alkhatib & Beneke, 2006). The periods of active recovery between the bouts allow for the cardiopulmonary system to recover so they are able to complete the next bout at a high intensity. However, these periods of recovery are not long enough for the participants to recover to resting level (Andersen, Schnohr, Schroll & Hein, 2000). It has been suggested that it is the intensity the individuals exercise at and not the duration of the cycle that is the cause for change (Wilson, Ellison & Cable, 2016), so they can exercise at a vigorous intensity to improve cardiac autonomic function, reducing the risk of morbidity and mortality. The

literature has shown that if participants are not training at a high enough intensity, there will not be any significant changes in heart rate variability.

2.5: *Rationale*

From reviewing the literature, there are clear health benefits associated with undertaking an exercise training programme that can reduce the risk of all causes of morbidity and mortality. HIIT has been shown to lead to significant health adaptations on risk factors for cardiovascular disease, with less time commitment than TAET. Autonomic dysfunction has shown to be a good predictor of morbidity and mortality rates, but current research on HIIT and the effect on cardiac autonomic function is limited and equivocal. In addition, there is limited enquiry into what the minimum amount of time is needed to incur significant health adaptation to the autonomic nervous system. There is currently no research on whether a short term HIIT programme over a four week period can significantly improve cardiac autonomic function in young, physically inactive males and females. Therefore, the aim of this study is to investigate the effects of a four-week HIIT programme on physically inactive, but healthy males and females on cardiac autonomic function and heart rate variability.

2.6: *Hypothesis*

From the literature review and rationale, the following hypothesis can be theorised for this investigation:

Experimental hypothesis - There will be a statistically significant difference in cardiac autonomic function following a four-week High Intensity Interval Training intervention compared to the control group.

Null hypothesis - There will be no statistically significant difference in cardiac autonomic function following a four-week High Intensity Interval Training intervention compared to the control group.

3.0: Method

3.1: *Participant information*

Forty-five physically inactive males and females from Canterbury Christ Church University volunteered to participate in the study. The demographic of the participants was age 22.8 ± 2.7 years, height 172.98 ± 9.23 cm and weight 74.15 ± 14.93 Kg. Participants were recruited through posters placed around the university and word of mouth. Participants then completed a short questionnaire, in which they stated how much physical activity they did and whether they had any cardiac or respiratory risk factors. The inclusion criteria for the study were that individuals did not meet the recommended amount of physical activity per week and were non-smokers but were healthy. The exclusion criteria for the study were if any of the subjects had one or more risk factors for cardiovascular disease or had any respiratory or heart conditions. A written information guide, which explained the study and what would happen at each phase, was handed out to all the participants. Before testing began, each participant had to complete a written informed consent and a Physical Readiness Questionnaire (PAR-Q). It was also made clear that participation was completely voluntary and they could withdraw from the study at any time. Ethical clearance for the experimental procedure was sought and granted by the Canterbury Christ Church University Ethics Panel.

3.2 *Equipment*

Before the start of the study, a baseline measure of both mass (Kg) and height (cm) was recorded. Weight was measured using a precision balance scale (model 848, Ravencourt Limited, United Kingdom) and height was measured using a Stadiometer (Seca model 202, Seca Gmbh & co.kg, Hamburg, Germany).

To measure the participant's cardiac autonomic function, the Task Force Monitor® (CNSystems, Medizintechnik AG, 2010, Version 2.2.22.2) was used to non-invasively calculate heart rate variability. This was done by placing electrodes both the clavicles and hips, which provided a continuous beat-to-beat monitoring and real time calculation for the parameters of autonomic function (Sharma, O'Driscoll, Saha, Sritharan, Sutton *et al* 2015). The Task Force Monitor® recorded the data at a frequency of 1000 Hz and 16-bit resolution. R – R intervals of heart rate variability were calculated using a 6-channel ECG (Fortin, Habenbacher, Heller, Hacker, Grüllenberger *et al* 2006). Power spectral analysis was used to calculate heart rate variability and was applied to an autoregressive model. By using the power spectrum, autonomic control of the cardiovascular system could be used to quantify different frequencies of heart rate variability (Fortin, Habenbacher, Gruellenberger, Wach & Skrabal, 1998). There are three frequency fluctuations the Task Force Monitor® can measure: very low frequency fluctuations (< 0.039); low frequency fluctuations (0.04 – 0.15 Hz); and high frequency fluctuations (0.15 – 0.4 Hz). LF/HF ratio can then be calculated, which is an index of sympathovagal balance (Lanfranchi & Somers, 2002; Hillebrand *et al* 2013). These frequencies are produced by the autonomic nervous system, which is jointly mediated through parasympathetic and sympathetic nervous systems. From this, heart rate variability data and LF/HF ratio can be calculated to see if any significant adaptations have occurred. VLF

was not measured for this study as it is unclear what this parameter measures (Usui & Nishida, 2017).

Each HIIT session was performed on a Wattbike cycle ergometer (Wattbike Ltd, Nottingham, UK). The Wattbike cycle ergometer was chosen for the study as it can be programmed using the Polar View Software for the protocol of the study and has been demonstrated be a reliable and valid cycle ergometer (Hopker, Myers, Jobson, Bruce & Passfield, 2010). This means the Wattbike cycle ergometer can calculate the exact level of resistance prescribed for the individual and it can be automatically applied through the fly wheel (Driller, Argus & Shing, 2013). A Wattbike was used as Hopker *et al* (2010) & Driller *et al* (2013) suggest the Wattbike is valid, reliable and reproducible for 30-second maximal sprint for physically inactive individuals as power output and physiological variables can be measured.

3.3: *Study Protocol*

Based on instrument coefficient of variation for Power Spectral Density using the Task Force Monitor, a sample size of 17-participants in each group has 80% power to detect a significant change, with a 2-sided $p < 0.05$. It was estimated that there would be a dropout rate of between 10-30%, leading to an overall sample size of 45 participants. Participants were needed for a total of five weeks to complete testing. Once all the participants were recruited, subjects were randomly allocated, by using stratified randomisation for gender into either the HIIT group (n=23) or the control group (n=22) (Field, 2013). Before each subject came into the

laboratory, they were instructed not to eat or drink for fours beforehand and no caffeinated products or alcohol for 24 hours beforehand as food, caffeine and alcohol are stimulants that could affects the results (Turley, Rivas, Townsend & Morton, 2017); however, water was allowed. Participants were instructed to be euhydrated and be well-rested before each test. This was to minimise any impact of individuals being dehydrated or sleep deprived on the results (Wallen, Gomersall, Keating, Wisløff & Coombes, 2016). This was done by instructing participants to keep hydration levels up and get eight hours of sleep. To minimise other variabilities that may have affected the results, both the HIIT and the control group were encouraged to maintain the same dietary habits and living activities they had before the intervention (Wallen *et al* 2016). The control group was urged to refrain from any physical activity they did not to do before the intervention and also maintain the same dietary habits. This was encouraged to maximise the possibility of only the HIIT intervention affecting the results and was measured through verbal confirmation.

This study used an independent-measures design, meaning each group had different participants. An independent-measures design was selected due to time constraints of the subjects as many subjects were not available for eight weeks of testing due to other commitments. This allowed for more subjects to be tested and to avoid having to have a washout period (Field, 2013).

3.4: *Experimental Procedure*

Before the testing, all subjects were given a designated time and day when they would come into the lab for testing. This was to keep everything consistent and attempting to make sure it just the HIIT that was affecting the results and not getting tested at different times of

the day (Rakobowchuk, Tanguay, Burgomaster, Howarth, Gibala *et al* 2008). Participants' height and weight were entered in to the Task Force Monitor®. The Kleiger, Stein & Bigger (2005) method for recording heart rate variability was carry out for each subject. Subjects were then instructed to lie down in the supine position, four large Ambu Bluesensor T electrodes were positioned just below the subjects' clavicles and just above their hips. The correct coloured wires were then attached to the electrodes. Participants then had fifteen minutes of rest so they could relax and get used to the Task Force Monitor®. The fifteen minutes of rest was followed by five minutes of continuous reading of heart rate variability via the 6-channel ECG. Once the participant was in the supine position, the lights in the room were turned off and there was complete silence to guarantee they were in the maximal rested state.

Post testing occurred 48 hours after the completion of the final HIIT session. This was to ensure that any changed were due to the HIIT intervention and not an acute response to the exercise (Miller, Herniman, Ricard, Cheatham & Michael, 2006). The post testing procedure was identical to the baseline testing. The control participants post testing occurred four weeks after the completion of their baseline testing.

3.5: *Randomisation Procedure*

For the study, it was important the both the HIIT and the control groups had an equal number of males and females. This was to make sure that the HIIT and the control group were representative of the population being tested and so that there was not more of one gender in each group. To achieve this, stratified randomisation for gender was conducted. Two mutually exclusive groups were created, with one being all the males and other being all the

females. Each group was homogenous as there was very little variability between the groups. Each group was made up of 21 individuals. Their names were then drawn out of a hat to see whether the participant would be in the control or the HIIT group. This meant there was a constant ratio of males to females in the HIIT and the control group, stopping one of the conditions having a lot more of one gender and there being high variability amongst each condition.

3.6: *Training Intervention*

From reviewing the literature, the minimum amount of exercise and active recovery needed was chosen to see if this could lead to significant changes in cardiac autonomic function. Four weeks for the HIIT intervention was selected as Laursen (2010) suggests that this is the minimum length of time needed for a training programme to evoke significant physiological adaptations. A low volume HIIT programme was selected as HIIT has been shown to evoke significant physiological remodelling, with lower time commitments and less exercise volume. This shows that quality exercise can be more successful than quantity of exercise (Gibala *et al* 2012). From reviewing the literature, the most common form of HIIT protocol was a 30-second maximal sprint, made up of three to seven bouts, with two to five minutes of active recovery. Sloth *et al* (2013) recommend that there should be two to three HIIT session a week. Cipryan, Laursen & Plews (2016) suggest that the maximal all out cycle sprints should be between 30 and 60 seconds to elicit significant changes in heart rate variability. Therefore, after reviewing the literature, Sloth *et al.'s* (2013) protocol was adapted for this study, with three, 30-second maximal sprints being conducted on a Wattbike, with two minutes of active recovery between bouts.

All HIIT sessions were supervised (Kessler, Sisson & Short, 2012). Before each HIIT session, the participant's weight (Kg) was taken so their resistance could be calculated to 7.5% of the individuals body weight. Each session was made up of three Wingate bouts of 30-seconds. During the 30-seconds maximal sprints, subjects cycled at 95% - 100% of HR_{peak}. Between bouts, two minutes of active recovery took place, when there was no resistance on the bike. Before the subjects started each session, there was a five-minute warm up at 65% of HR_{peak}. The amount of resistance each individual had to cycle at was 7.5% of their body weight (Kg) (Astorino, Allen, Roberson & Jurancich, 2012). The resistance was then automatically applied through the fly wheel and airbrake. After the three Wingates, each session ended with a three-minute cool down at 65% of HR_{peak}. Strong verbal encouragement was given to each subject during their all-out cycle sprints in an attempt to get every participant to exercise at 100% of HR_{peak}. Participants had to complete 80% of the HIIT sessions to be included in the statistical analysis, with 100% of the subjects adhering to this.

3.7: *Data Analysis*

All the data analysis was performed through the Statistical Package for Social Science (IBM SPSS Statistics for Windows, version 22; SPSS Inc., Chicago IL, USA). First, as under 50 participants were used, a Shapiro Wilk Test of Normality was conducted to determine whether the data met parametric assumptions (Field, 2013). This identified the data as being parametric. To evaluate whether a significant difference had occurred pre and post intervention between the HIIT and the control group, an analysis of covariance (ANCOVA) test was used., which calculated the delta score as the baseline score was use as a covariate. The data is expressed as mean ± standard deviation, unless otherwise addressed. A *p* value of

<0.05 was regarded as statistically significant. Normalised units were not used for this study as it can lead to the final results being changed (Field, 2013).

4.0: *Results*

Four participants withdrew from the study for undisclosed reasons, meaning that a total of 41 participants completed the study, with 21 in the HIIT group and 20 in the control group. The age of the HIIT group was 21 ± 1.7 years of age compared to 22 ± 3.5 of the control group. The HIIT group was slightly taller 173.7 ± 9.5 (cm) compared to the control group 172.4 ± 8.8 (cm). The power analysis suggested that a minimum of 17 participants were needed in each group for the study to detect significant change. As there were more than 17 participants in each group, this study had adequate power to detect significant changes

Table 4.1 showing the demographics of the control and HIIT group

	Control (n=20) Mean ± SD		HIIT (n=21) Mean ± SD	
	Pre	Post	Pre	Post
Weight (kg)	74.3 ± 15.9	74.2 ± 15.7	73.9 ± 14.4	74.3 ± 15.0
BMI ($kg \cdot m^2$)	24.9 ± 4.5	24.8 ± 4.4	23.4 ± 3.2	23.5 ± 3.4
BSA (m^2)	1.87 ± 0.22	1.87 ± 0.23	1.84 ± 0.22	1.84 ± 0.23
Resting sBP (mmHg)	120.9 ± 9.6	119.7 ± 10.9	121.2 ± 10.3	114.6 ± 8.8
Resting mBP (mmHg)	88.6 ± 7.6	88.8 ± 9.3	87.8 ± 8.4	85 ± 6.3
Resting dBP (mmHg)	69.9 ± 7.4	69.9 ± 8.8	69.5 ± 10.8	66.1 ± 5.9
Resting PP (mmHg)	51.2 ± 8.6	49.7 ± 7.9	51.7 ± 12.3	48.5 ± 8.34

The results show that there was a significant reduction in resting heart rate (66 ± 10 to 63 ± 13 $b \cdot min^{-1}$) and no significant change (61 ± 8 to 61 ± 7 $b \cdot min^{-1}$) in the control group (P

= 0.013). As illustrated in Figure 3.1, it can be seen that four weeks of HIIT resulted in significant improvements in heart rate variability. R – R total power spectrum density significantly increased in the HIIT group, improving 498.8 ± 239.8 ms^2, compared to -252.6 ± 227.4 ms^2 in the control group (p = 0.03). R – R high frequency power also significantly increased in the HIIT group, improving 389.4 ± 123 ms^2, compared to -163.7 ± 125.5 ms^2 in the control group (p = 0.004). Although R – R low frequency power was not statistically significant, there was a positive trend in the data as in the HIIT group increased 189.7 ± 91.2 ms^2, compared to -42.8 ± 86.5 ms^2 in the control group (p = 0.074).

The ANCOVA also established that there were no significant differences (p > 0.05) between the HIIT group and the control group in R – R very low frequency power (p = 0.292), LF/HF ratio (p = 0.365), R – R normative units of low frequency power (p = 0.364) and R – R normative units of high frequency power (p = 0.363).

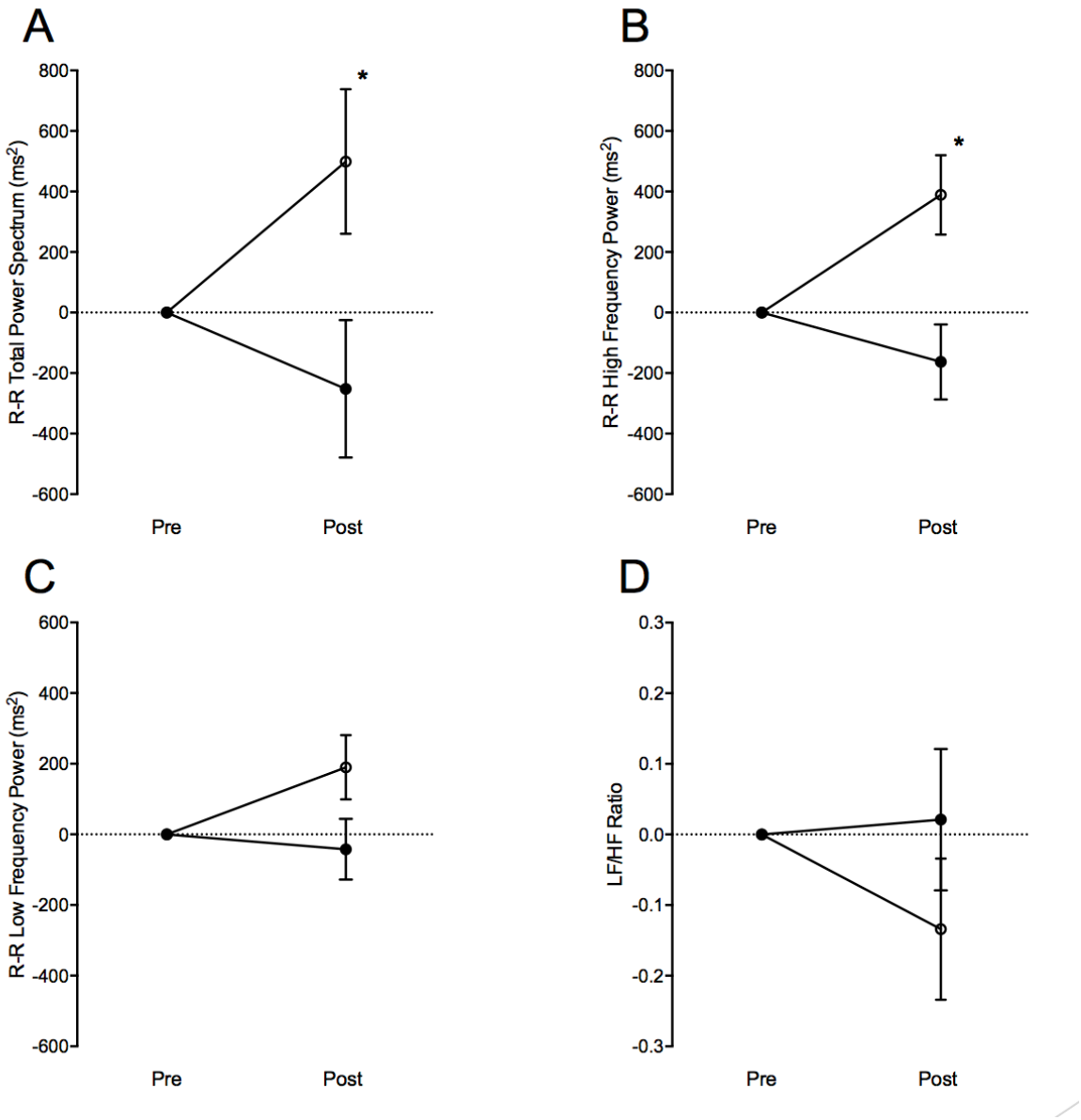

Figure 4.2 mean R – R total power spectrum density (**A)**, R – R high frequency power (**B**). Mean R – R low frequency power (**C**) and LF/HF ratio (**D**) change values for the intervention (open circles) and control (closed circles) condition. Error bars show the standardised error of the mean. * significant ($P < 0.05$) difference between the control and the intervention change values.

5.0: *Discussion*

The aim of the study was to examine the changes in cardiac autonomic function following four weeks of HIIT in physically inactive, but healthy young males and females. As each group had more than 17 participants, the study was adequately powered to find significant results, meaning the data could be confidently interpreted. The results show that HIIT significantly reduces resting heart rate and significantly increases R – R total power spectrum density and R – R high frequency power compared to the control group. Therefore, the null hypothesis can be rejected and the experimental hypothesis can be accepted as heart rate variability significantly improved. Previous research has shown that physical activity reduces the risk of cardiovascular diseases in individuals who previously lived unhealthy lifestyles. The results found in this study support previous research, showing that HIIT can improve autonomic function, but offers new research as it shows that HIIT can improve autonomic function in physically inactive, but healthy young males and females in four weeks. This demonstrates that HIIT can reduce the risk of autonomic dysfunction as heart rate variability was improved. These results show that HIIT can cause significant adaptations to occur in less time than the current physical activity guidelines recommend.

5.1: *Adaptations to Resting Heart Rate in response to HIIT programme*

The results from the study show that resting heart rate significantly reduced (2.5 $b \cdot min^{-1}$; $p < 0.05$) after the HIIT programme. These findings are supported by Kiviniemi *et al* (2014), who found that resting heart rate significantly reduced by 2 $b \cdot min^{-1}$. This is further supported by Matsuo, Saotome, Seino, Shimojo, Matsushita *et al* (2014), who found that resting heart rate significantly reduced by 5.4 $b \cdot min^{-1}$ following a HIIT programme. Reduced resting heart rate is a sign of improved cardiovascular health. Reduction in resting heart rate

following a HIIT programme is prevalent amongst the literature. However, the mechanisms causing these reductions are complicated and not yet fully understood (Heydari *et al* 2013).

A potential mechanism that could have significantly reduced resting heart rate is improvements in the autonomic nervous system. This study has shown that HIIT improves heart rate variability as high frequency power significantly increased. High frequency power has shown to be an indicator of parasympathetic activity. A rise in parasympathetic activity at rest has shown to inhibit sympathetic activity, due to the increase of acetylcholine that leads to the polarisation of the sinus node cells (Olshansky *et al* 2008). HIIT may lead to a rise in angiotensin II and nitric oxide, thus inhibiting cardiac vagal activity. Nitric oxide has been shown to inhibit sympathetic activity, thus increasing control over vagal modulation and reducing myocardial stress, therefore reducing heart rate. However, the effects angiotensin II and nitric oxide have on the autonomic nervous system are not fully understood and need to be researched further (Routledge *et al* 2010).

Another potential mechanism that could have caused resting heart rate to significantly reduce is cardiac remodelling. The HIIT programme could have led to hypotrophy of the left ventricle. This means the heart would have been able to pump out more blood to the working muscles as stroke volume would have increased, resulting from a greater end diastolic volume. Resting heart rate will thus reduce to a higher systolic volume. This could have increased due to greater contractility or an increase in blood volume (La Rovere, Bersano, Gnemmi, Specchia, & Schwartz, 2002). This was suggested by Heydari *et al* (2013), who found a significant reduction in resting heart rate and attributed this to a significant increase in

stroke volume, going from 77.2 ± 24.9 to 90.4 ± 26.3 ml; $p<0.05$. However, as cardiac remodelling was not examined in this study, this may not have occurred.

A drop of 2.5 beats per minute in resting heart rate is meaningful change. Lowering resting heart rate means the heart is more efficient at delivering oxygenated blood and nutrients around the heart and the body. This reduces the risk of cardiovascular disease as there is less stress on the myocardium (Smith, Benjamin, Bonow, Braun, Creager *et al* 2011). Therefore, showing that HIIT has led to a significant health adaptation for the participants.

5.2: *Adaptations to Cardiac Autonomic Function in response to HIIT programme*

Detailed mechanisms that could have caused the adaptations in the present study were not explored and cannot be confirmed by the data. The effects HIIT has on cardiac autonomic function to cause positive health adaptations have still not been fully explored. Nevertheless, Bond, Cockcroft, Williams, Harris, Gates *et al* (2015) suggest that HIIT increases parasympathetic nervous system activity and decreases sympathetic nervous system activity to the myocardium at rest. The results found in the present study following four weeks of HIIT are supported by Kiviniemi *et al* (2014), who also found that heart rate variability significantly improved following an HIIT programme. Heart rate variability has been seen to increase at rest when the myocardium is predominately controlled by the parasympathetic nervous system (Sztajzel, 2004).

The present study documented that R – R total power spectrum density significantly improved by 498.8 ± 239.8 ms^2 ($p = 0.03$) and R – R high frequency power significantly

improved by 389.4 ± 123 ms^2 ($p = 0.004$) when compared to the control group. There was a non-significant increase in R – R low frequency power. However, there was a trend in the data, improving by 189.7 ± 91.2 ms^2 ($p = 0.074$). In comparison to these findings, Kiviniemi *et al* (2014) reported that total power spectrum density and low frequency power significantly improved ($p < 0.05$) whilst high frequency power had a non-significant trend in the data ($p = 0.068$). They found that there was no significant difference in LF/HF ratio, which the present study also found. Both low and high frequency are predictors of cardiac morbidities. The results from this study suggest there has been an increase in parasympathetic activity and/or a decrease in sympathetic activity, which is beneficial for cardiovascular health as cardiac autonomic function has improved, thus suggesting the participants will have a reduced risk of all causes of morbidity and mortality. This means that HITT may be an efficient strategy to improve cardiac autonomic function (Kiviniemi *et al* 2014).

Following the four week HIIT programme, high frequency power significantly improved, which can also be found in Heydari *et al* (2013), as they found high frequency power significantly improved after 12 weeks of HIIT compared to the control group. This is supported by Rakobowchuk, Harris, Taylor, Cubbon, & Birch (2013), who also found that high frequency power improved following six weeks of HIIT compared to the control group. This indicates an improvement in cardiovascular health as there has been an increase in parasympathetic activity and/or a decrease in sympathetic activity, thus improving cardiovascular health (Laing *et al* 2011). The results also show that there was a positive trend in low frequency power; however, these results were not significant. Boutcher *et al* (2013) also found an increase in low frequency power that was not significant following a HIIT programme. In addition, Rakobowchuk et al (2013) found that in normalised units there was

an improvement in low frequency power; however, this was not significant. Low frequency power has been noted to represent both sympathetic and parasympathetic activity, which means the increase in low frequency power could be due to an increase in parasympathetic activation (Ruediger *et al* 2004). However, both the present study and Kiviniemi *et al* (2014) found that there was no significant decrease in LF/HF ratio. Reason why LF power and HF/LF ration did not significantly increase)

Both the present study and the literature demonstrate that HIIT has a positive effect on cardiac autonomic function, as HIIT leads to significant adaptations occurring as parasympathetic activation increases and/or sympathetic activation decreases. R – R high frequency power has been well documented as a marker for parasympathetic activity, which indicates cardiac vagal activity (Pomeranz, Macaulay, Caudill, Kutz, Adam *et al* 1985). R – R low frequency power has been noted to include both parasympathetic and sympathetic neural outflows, which is a marker for cardiac vagal activity and sympathetic effects (Hillebrand *et al* 2013). However, much of this is determined by vagal modulation and baroreflex function (Kiviniemi *et al* 2014). Therefore, the increase in high frequency power and low frequency power could be due to an increase in vagal modulation or baroreflex mediation of the sinoatrial node. However, no changes occurred in sympathovagal balance as LF/HF ratio did not decrease, which was also found by Rakobowchuk et al (2013). These adaptations can reduce the risk of autonomic dysfunction as heart rate variability has been improved, and potentially reducing the risk of cardiovascular disease, meaning these individuals are at a lower risk of morbidity and mortality.

A potential mechanism that has been highlighted by previous research to explain the improvements following a HIIT programme in autonomic function is improved baroreflex sensitivity (Kiviniemi *et al* 2014). Improvement in baroreflex sensitivity could be caused by an increase in blood volume, which would increase baroreflex sensitivity. This reduces blood pressure as blood vessels vasodilate due to an increase in baroreceptor impulse frequency, meaning that the blood vessels can vasoconstrict. The improvements in baroreflex sensitivity could be due to the improvements in heart rate variability and parasympathetic activity, as R – R total power spectrum density and R – R high frequency power significantly improved. Following HIIT, cardiovascular health has been shown to improve as the enhancement of baroreflex sensitivity reduces the risk of ventricle fibrillation (La Rovere, *et al* 2002). However, if baroreflex sensitivity was enhanced, it could have increased parasympathetic activity. As parasympathetic activity can be detected in both low frequency power and high frequency power, it could explain why HIIT increased both low and high frequency power. Therefore, it could be interpreted that no detectable change in LF/HF ratio could be due to no changes in sympathetic activity (Figueroa *et al* 2007). Improvements in baroreflex sensitivity following a HIIT programme has led to a reduction in ventricular fibrillation and improvements of the baroreflex function in the peripheral sympathetic nervous system, showing improved cardiovascular health as it reduces the risk of mortality (La Rovere *et al* 2002). Improvements of baroreflex function in the peripheral nervous system has been demonstrated by the endothelium releasing an increased amount of nitric oxide synthase. This helps the non-working muscles to vasodilate, causing them to relax. This can cause the inhibition of the sympathetic nervous system, leading to great cardiovascular protection (Shephard & Balady, 1999). This supports the findings as resting heart rate reduced by 2.5 $b \cdot min^{-1}$ as the

parasympathetic nervous system predominantly works when the body is at rest (Olshansky *et al* 2008).

These adaptations caused by the HIIT programme can lead to positive health benefits, which can reduce the risk of cardiovascular disease, meaning there is a reduced risk of morbidity and mortality (Parpa *et al* 2009). As baroreflex sensitivity and blood pressure were not measured in this study, they may not be the mechanisms that led to the significant adaptations. However, improvements in baroreflex sensitivity leading to improvements in cardiac autonomic function are prevalent within the literature, so it is plausible this may have led to the improvements in heart rate variability.

Some components of this study have shown that HIIT can lead to significant health adaptations by improving heart rate variability and reducing the risk of cardiovascular disease. The amount of time spent exercising was less than the current physical activity guidelines recommended. Therefore, individuals who lack motivation and who do not have the time to meet the guidelines for physical activity would benefit from participating in a similar HIIT programme and would likely see many health benefits. Four weeks of HIIT has shown that cardiac autonomic function can be significantly improved compared to the control group. As autonomic dysfunction is a predictor for cardiovascular disease and poor health, the present study has provided evidence that a low volume HIIT programme, can lead to significant improvements in cardiac autonomic function in four weeks.

This study shows that short term HIIT can lead to significant improvements in cardiac autonomic function, whilst other literature has shown that HIIT can improve other health

parameters, meaning HIIT can be effective method at improving health. As HIIT takes up less time and is seen as more enjoyable than TAET, it has a higher adherence rate as less people drop out (Heydari *et al* 2013). This study shows that partaking in three, 30 second, all out maximal cycle sprints three times a week, with two minutes of active recovery between bouts, for four weeks can lead to significant adaptations in young physically inactive males and females. Therefore, the current physical activity guidelines should perhaps be amended, as significant adaptations can occur in less time, with an increase in adherence with HIIT.

5.3: *Limitations*

The present study has some limitations that merit discussion. As there is limited literature on the effects of HIIT on cardiac autonomic function in physically inactive, but healthy males and females, it can make it difficult to make generalisations about the results. There are some factors that were not controlled throughout the study, which could have impeded upon the results. There was no dietary restriction placed upon the participants outside of the laboratory, with the exemptions that they were requested to refrain from consuming food, caffeinated products and alcohol before baseline and post testing. It was recommended that participants did not change their diets; however, some may have used the HIIT programme as a method to start getting fit and changed their diet accordingly. Furthermore, the consumption of alcohol was not measured during the study, which could have impacted upon the results. Participants were also advised to refrain from any physical activity that they participated in before the study began. However, how much physical activity participants were undertaking away from the intervention was not recorded, providing a further limitation to the findings. Although the present study had four participants drop out, the HIIT programme only took place over a four week period. It is unknown if the adherence

would have been as high, or if there would have been more drop outs if the intervention was conducted over a longer period of time

The autonomic nervous system is complex, so may not be the only explanation for the changes seen in the study. The mechanisms that could have caused the changes were not measured in the study, so the explanation given in the discussion may not be the only reasons for the adaptations that occurred following the HIIT programme. Therefore, the use of non-invasive equipment, such as an echocardiogram, could be used to measure other potential adaptations. The echocardiogram was not used for this study as there was no one available who had the expertise or qualifications to use this piece of equipment. This could have provided further insight into the changes that occurred and mechanisms that caused the changes. Furthermore, the present study has shown that significant adaptations can occur in four weeks to improve individuals' health. However, four weeks may not be a long enough programme to make generalisations about long term health benefits.

However, another potential mechanism which could have resulted in reduced resting heart is by intrinsic electrophysiological changes in the sinus node. This causes the pacemaker ion channels to remodel forcing the downregulation of the channel HCN4 (D'Souza, Bucchi, Johnsen, Logantha, Monfredi *et al* 2014). This research throws in the question that the autonomic nervous system may not have been altered by the four weeks of HIIT and that it could be due to the remodelling of the pacemaker ion channels and thus changes in the sinus node.

5.4: *Future Research*

From the limitations of the study, future research may want to investigate the mechanisms for changes in autonomic function. Whilst conducting research into autonomic function, it may also be beneficial to investigate haemodynamic parameters, cardiorespiratory parameters and left ventricular hypotrophy, as these could play a role in the improvements in heart rate variability. This is because they impact on baroreceptor impulse which could be responsible for changes in autonomic function (Gibala *et al* 2012). It may also be beneficial to investigate the longer term effects of a HIIT programme versus a TAET programme. This is because HIIT has shown to lead to superior adaptations in the cardiovascular system compared to TAET in short term studies. However, more longitudinal studies will need to be conducted over different time lengths to examine and compare the long term effects of HIIT compared to a TAET programme. This would be worthwhile to see if the benefits from a HIIT programme plateau compared to the benefits from a TAET programme. Finally, future research may want to look at 24 hour ambulatory heart rate variability. This could examine the impact HIIT would have on the autonomic nervous system in individuals through the day, and not just in a laboratory at rest. This could give additional understanding into any adaptations that occur.

5.5: *Conclusion*

The findings from the present study show that a short term HIIT programme can lead to significant adaptations in cardiac autonomic function in resting heart rate and heart rate variability compared to the control group. This study demonstrates that there is a higher

parasympathetic dominance and/or a decrease in sympathetic dominance at rest. This study has shown that a low volume HIIT programme can lead to improvements in autonomic function, meaning individuals are less likely to suffer from autonomic dysfunction. This means they have a reduced risk of developing cardiovascular diseases, leading to a reduced risk of morbidity and mortality. This study has also shown that short volume HIIT programme has improved health parameters in less time than the current physical activity guidelines recommend. Therefore, these physical activity guidelines should be examined further as HITT may be good alternative for individuals who lack time and find TAET tedious. It should also be noted that HITT is not for everyone and in particular may not be suitable for older individuals with pre-existing Cardiovascular diseases, but also for younger individuals with non-ischaemic cardiovascular disease. The results demonstrate that the experimental hypothesis can be accepted as four weeks of high intensity interval training led to significant adaptations in cardiac autonomic function.

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7.0: *Appendix*

7.1: *Informed consent*

INFORMED CONSENT

The full details of the tests have been explained to me. I am clear about what will be involved and I am aware of the purpose of the tests.

I know that I am not obliged to complete the tests. I am free to stop the test at any point and for any reason.

The test results are confidential and will only be communicated to others such as my coach if agreed in advance.

As far as I am aware, there is nothing that might prevent me from successfully completing the tests that have been outlined to me.

Signature of Participant: ……..

Signature of Sport Scientist: ……..

Date: ……

7.1: *PAR-Q*

Department of Sport Science, Tourism and Leisure

Sport Science Informed Consent & Health and Fitness Questionnaire

Name: …….

Date of Birth: …… **Age: ……** **Sex: ……..**

Please answer the following questions by ***circling*** the appropriate response and if necessary providing extra information in the spaces provided.

ANY INFORMATION CONTAINED HEREIN WILL BE TREATED AS CONFIDENTIAL

1. How would you describe your present level of fitness?

Untrained / Moderately trained / Trained / Highly trained

2. Average number of hours spent exercising **…….……..per wk**

3. How would you describe your present bodyweight?

Underweight / Ideal / Slightly overweight / Very overweight

4. How would you describe your smoking habits?

Non smoker / Previous smoker / Currently smoking

5. How would you describe your alcohol intake?

Never Drink / An occasional drink / A drink every day / More than one drink a day

(Note 1 drink = 1 unit)

6. Have you had to consult your doctor within the last six months? **Yes / No**

If you have answered **yes**, please give

details:**…….**

7. Are you presently taking any form of medication? **Yes / No**

If you have answered **yes**, please give details:

……

8. Do you suffer or have you ever suffered from any of the following?

a. Diabetes **Yes / No** b. Asthma **Yes / No**

c. Epilepsy **Yes / No** d. Bronchitis **Yes / No**

e. Any form of heart complaint **Yes / No** f. Serious Back or Neck Injury **Yes / No**

g. High blood pressure **Yes / No** h. Aneurysm [1] or Embolism[2] **Yes / No**

1: Arterial wall weakness causing dilation. 2: Obstruction in the Artery.

9. Is there a history of heart complaint in your family?

Yes / No

If you have answered **yes**, please give details:

……

10. Do you have any allergies?

Yes / No

If you have answered **yes**, please give details:

……

11. Do you currently have any form of muscle or joint injury?

Yes / No

If you have answered **yes**, please give details:

……

12. Have you had to suspend your normal training/physical activity in the last two weeks?

Yes / No

If you have answered **yes**, please give details:

……