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DISSERTATION ASSESSMENT PROFORMA:
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Comments	Section
	Title and Abstract Title to include: A concise indication of the research question/problem. Abstract to include: A concise summary of the empirical study undertaken.
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	Methods and Research Design To include: details of the research design and justification for the methods applied; participant details; comprehensive replicable protocol.
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CARDIFF METROPOLITAN UNIVERSITY
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CARDIFF SCHOOL OF SPORT

DEGREE OF BACHELOR OF SCIENCE (HONOURS)

SPORT AND EXERCISE SCIENCE

ACUTE VASCULAR RESPONSES FOLLOWING A SINGLE BOUT OF HANDGRIP RESISTANCE EXERCISE

(Dissertation submitted under the discipline of PHYSIOLOGY & HEALTH)

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ABSTRACT

Purpose: The aim of the present study was to investigate the acute vascular responses to a single bout of handgrip resistance exercise (RE). **Methods:** 9 physically active male students volunteered who had not previously engaged in chronic weight training. The average physical characteristics were, age (20.4 ± 1.1 years); height (1.78 ± 0.1 cm); weight (79.3 ± 9.2 kg) and body mass index (BMI) (24.9 ± 2.5). Each subject completed a handgrip RE for 5 mins at 10% of their 1 repetition maximum (1RM) with flow mediated dilation (FMD) being carried out pre and post handgrip RE intervention. Vascular measures of base mean diameter (BMD), response mean diameter (RMD), delta diameter (DD), delta % (DP), time to peak (TP), base mean envelope velocity (BMEV), response mean envelope velocity (RMEV), base mean shear rate (BMSR) and response mean shear rate (RMSR) were recorded using ultrasound at baseline and post handgrip RE intervention. Blood pressure (BP) and heart rate (HR) measures were recorded by use of a finometer throughout the entire study. **Results:** Paired t-tests revealed no statistical significant difference between pre and post intervention measures for variables ($P>0.05$), except for BMEV (baseline 7.3 ± 2.2; post intervention 11.1 ± 3.4 = 52% increase) and BMSR (baseline 145.6 ± 41.8; post intervention 214.8 ± 73.1 = 48% increase) ($P<0.05$). **Conclusions:** It was concluded that even though the handgrip RE caused a significant increase in the variables BMEV and BMSR, the exercise was not physically demanding enough to cause enough of an increase in those variables to result in a vasodilatory or arterial stiffening response.

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CHAPTER I

INTRODUCTION

Exercise is considered highly beneficial for the health of the vascular system (Phillips et al., 2011) and extensive research has been conducted in the area (Green et al., 2010; Green et al., 2012; Thijssen et al., 2012). According to Thomas et al. (2011), exercise is an effective strategy to reduce the risk of cardiovascular disease and Kaupuzs (2012) states it can reduce some cancers, type-2 diabetes, obesity and osteoporosis. Numerous scientific statements and health guidelines from around the globe state that regular exercise reduces sickness and death, decreases the markers of cardiovascular disease risk, increases ability to function, and improves quality of life and psychological status by reducing stress or anxiety and enhancing relaxation and mood states. “There is now a wealth of sophisticated epidemiological evidence to demonstrate that physical activity is associated with reduced risk of coronary heart disease…” (O’Donovan et al., 2010).

Consequently, research has now evolved proposing that regular aerobic exercise is currently the exercise modality of preference due to its enhanced and amplified benefits upon health (Collier et al., 2010). According to Franklin & Billecke (2012) regular aerobic training provides cardio-protection resulting in an improvement in blood lipid profiles and enhancements in the intrinsic pump capacity of the myocardium (Chaudhary et al., 2010). Additionally, Okamoto et al. (2007) proposes that regular aerobic training aids the prevention and treatment of cardiovascular disease and can also prevent and reverse arterial stiffening. This is considered highly beneficial as reductions in arterial compliance or increases in arterial stiffness impair the buffering function of the vessels (Miyachi et al., 2004). It is therefore evident that exercise, and more importantly, aerobic exercise confers favourable effects upon the health of the vascular system.

However, RE is a different form of exercise that is becoming increasingly popular amongst the population. It has been highlighted that very little is known of the effects this type of exercise presents upon the vasculature (Kleiner et al., 1996). A relatively small collection of research has been carried out in this area and the resulting theories and hypotheses are very contradicting.

According to Anton et al. (2006), resistance training (RT) has become a critical component in exercise prescription programmes for healthy adults. It has been stated that it is widely recommended for the prevention of sarcopenia and osteoporosis and can also prevent or treat lifestyle related diseases (Okamoto et al., 2010). In addition, RT has been advocated to combat cardiovascular disease and diabetes (Kawano et al., 2010), and can promote favourable effects such as increasing muscle mass, strength and preventing weight gain (Tanimoto et al., 2009).

Although, in contrast it has been suggested that RT increases arterial stiffness and reduces venous compliance (Okamoto et al., 2009). This needs to be carefully considered as arterial stiffness is a marker of the risk of future cardiovascular complications and is associated with increased morbidity and mortality. In addition, arterial stiffness has been reported to contribute to elevations in systolic blood pressure (SBP), left ventricular hypertrophy, coronary ischemic disease and reductions in arterial baroreflex sensitivity (Miyachi et al., 2004). Furthermore, others state that RE confers negative effects on vascular function, as resistance trained men have been shown to possess higher pulse pressures and increased peripheral resistance (Collier et al., 2010). Therefore, it can be seen that the current experimental findings on the topic are very contradicting highlighting the need for further investigation to clarify this area of exercise.

Furthermore, the majority of studies have incorporated the use of exercises that recruit a large amount of muscle mass leaving the effects of lower forms such as handgrip relatively unknown. Significantly less literature has been conducted in this area, which is an issue as a few study's (Shoemaker et al., 1997; Green et al., 2010; Green et al., 2012) have highlighted their importance stating that smaller forms of RE are beneficial as they provide insight into localized effects of exercise that are less dependent upon central regulatory or neural changes (Green et al., 2010).

Therefore as a result, the present study implemented a single bout of handgrip RE to measure the acute vascular responses, and to examine the effect these responses had upon the vasculature. Acute responses were monitored as the majority of studies have investigated primarily the chronic adaptations that occurred in the cardiovascular system as a result of repeated bouts of RE, very few have looked at the acute effects (Kleiner et al., 1996). This was done to address the gap in the literature concerning a lack of research available on the effects of lower forms of RE, and to add to the body of knowledge concerning the vascular effects experienced from engaging in resistance activity.

1.0 Aims and Objectives

The aim of the present study was to determine how the vasculature responds acutely to a single bout of handgrip RE. It was hypothesised that subjects would display an increase in vascular measures, however will not experience arterial stiffening or a reduction in venous compliance. Instead, participants will experience vasodilation of the artery due to the release of nitric oxide (NO) brought on by the shear stress stimulus.

CHAPTER II

LITERATURE REVIEW

2.0 Vascular Responses to Exercise

Extensive research has been conducted in the area of the vascular responses following exercise, with the majority of literature favouring the effects. According to Phillips et al. (2011), exercise is beneficial to the health of the cardiovascular system as it protects against and reduces the development of cardiovascular disease. It is stated that exercise has been shown to enhance peripheral vascular endothelial function in patients with cardiovascular disease, and that chronically, exercise can improve cardiovascular health. Most literature agree with these claims, and authors such as Tanaka et al. (2002) expand that regular exercise is also associated with a decreased risk of stroke. Therefore, it is evident that regular exercise possesses beneficial effects on the cardiovascular system.

However, research has evolved and become more specific stating that these effects are amplified through the use of regular aerobic exercise. Currently there is a greater body of literature conducted in this area, with its content gaining the wide-spread attention of health and medical professionals worldwide. Collier et al. (2010) suggests that aerobic exercise is currently recommended as the exercise modality of preference for decreasing cardiovascular risk, and authors such as Miyachi et al. (2004) have demonstrated that regular aerobic exercise is efficacious in preventing and reversing arterial stiffening in healthy adults.

Coinciding with the above, according to Thijssen et al. (2012), aerobic exercise and higher fitness levels confer cardio-protection and can combat the effects of atherosclerosis. It is stated that exercise training can decrease arterial wall thickness in healthy asymptomatic subjects, as well as in subjects with cardiovascular disease and subjects with risk factors that possess increased arterial wall thickening (Thijssen et al., 2012). However, it is mentioned that more intense or prolonged exercise exposure is required to experience these changes in vascular wall remodelling.

Consequently, due to its beneficial effects, exercise has been recommended and subscribed as treatment by health professionals to aid the health of special populations such as the older generation. Seals et al. (2008) states that regular physical activity, particularly large-muscle aerobic exercise, is an effective strategy for combating several adverse physiological changes associated with arterial

ageing, especially increases in large-elastic artery stiffness and vascular endothelial dysfunction. It is expanded that this aspect accounts for the increased cardiovascular functional capacity and lower prevalence of cardiovascular disease displayed in physically active and aerobically fit middle-aged and older adults, in comparison to sedentary individuals of the same age.

2.0.1 Acute Responses

Studies have shown that there are many acute responses associated with exercise. These include increases in HR and both SBP and diastolic blood pressure (DBP) (Hamer et al., 2012), an increase in arterial diameter along with wall-to-lumen ratio (Green et al., 2012), and also increases in velocity of blood flow and shear rate (Phillips et al., 2011). Literature highlights that the most accurate, non-invasive method to measure these responses is through the use of an FMD (Thijssen et al., 2010).

2.0.1.1 FMD

FMD has shown to be a successful non-invasive method for measuring the acute vascular response to various forms of exercise. According to Thijssen et al. (2010), FMD can describe any vasodilatation of an artery following an increase in luminal blood flow and internal-wall shear stress (Figure 1). In response to flow, the endothelium of an artery releases a substance that possesses the characteristics of a relaxation factor. Blood flow-associated shear stress, which is the tractive force the increased blood flow exerts upon the vessel walls (Ene-lordache et al., 2003), is the stimulus that is sensed by deformation of mechanosensitive structures at the cell membrane. These may include the primary cilia, mechanosensitive ion channels or the glycocalyx. The shear-stress mechanotransduction activates a signalling cascade that results in vasodilator production of NO, with the amount produced dependent upon the nature of the shear-stress stimulus. Vasodilators then diffuse from the endothelial cell into the smooth muscle. Here, the vasodilators trigger a signalling cascade that results in a lowering of calcium concentration and vasorelaxation, therefore causing dilation of the artery. It is suggested that the extent of diameter change that results from a given degree of smooth muscle relaxation is influenced by vessel wall structural factors, such as relative proportions of collagen and elastin or wall-to-lumen ratios.

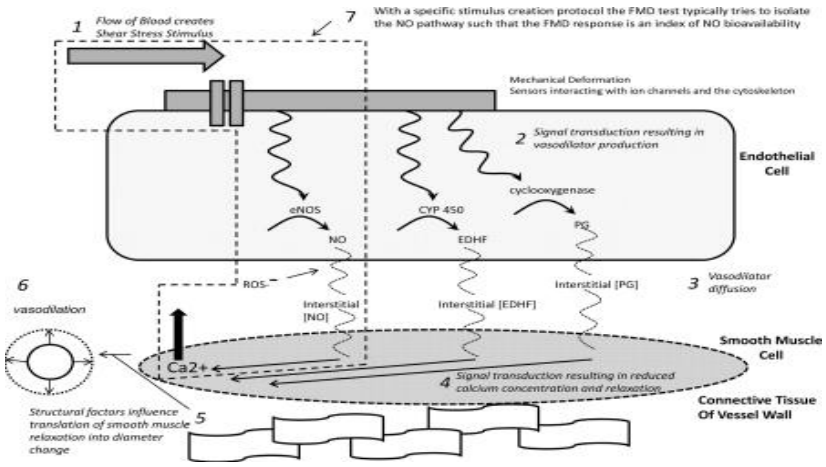

Figure 1. The role of NO in flow-induced dilation (Thijssen et al., 2010).

Therefore, it can be said that FMD is quantified as the change in vessel diameter from baseline dimensions before the application of the shear-stress stimulus. However, Thijssen et al. (2010) elaborates that due to the established vasoprotective properties of NO, FMD is often intended as an index of NO bioavailability.

In addition, according to authors such as Pyke & Tschakovsky (2005), NO, which is one of many vasoactive substances released by the endothelium in response to shear stress, is of particular interest to researchers as it is an antiatherogenic molecule. A reduction in its bioavailability may play a role in the pathogenesis of vascular disease. Furthermore, these authors also highlight an important factor that needs to be considered. They state that within vessels with different diameters, the same flow may represent a very different shear stress stimulus (Figure 2). This is due to narrower vessels placing an increased peripheral resistance upon blood flow, thereby increasing the shear rate and BP (Green et al., 2010; Green et al., 2012; Thijssen et al., 2012). This is important when investigating how athletes respond to exercise, as according to Green et al. (2012) athletes possess larger arteries with a larger wall-to-lumen ratio than sedentary individuals. Therefore, it can be hypothesised that due to Pyke & Tschakovsky (2005) findings, athletes may experience less shear stress due to their larger lumen diameters placing less resistance upon blood flow. This coincides with Jurva et al. (2006) research where it is stated that conditioned individuals display

less or no vascular response to RE in comparison to sedentary individuals. This may be the case as athletes may experience less of a shear stress stimulus, resulting in less NO signalling, leading to less of a vasodilatory response. Or on the other hand, due to less tractive force being exerted on the vessel walls, athletes may be at less risk of experiencing endothelial impairment. This can be said as Jurva et al. (2006) states that it is the effects of elevations in SBP that lead to arterial stiffening. Therefore, due to acquiring larger lumen diameters, they experience less resistance from blood flow, leading to a lower BP and resulting in less chance of endothelial impairment being experienced.

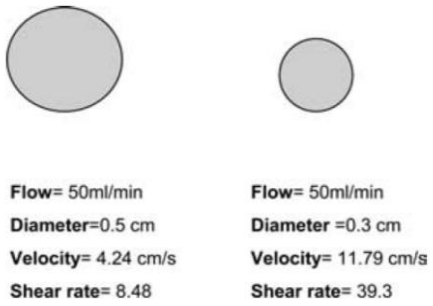

Figure 2. Vessels with different diameters present a different shear rate with the same blood flow (Pyke & Tschakovsky 2005).

2.0.2 Chronic Responses

Chronically, according to Green et al. (2012), endurance athletes possess enlarged arteries which also exhibit decreased wall thickness. Within his study he compared an athlete's artery with the artery of a healthy non-athletic control subject (Figure 3). The findings convey that firstly, the athlete's artery is larger in size, secondly, that it contains a larger lumen diameter and thirdly, that it possesses a thinner muscular wall. It is elaborated that the reasons for the occurrence of these changes are that haemodynamic signals may contribute to arterial remodelling. Shear stress implicates the localized effects of repeated exercise bouts on conduit and resistance artery enlargement, whereas changes in wall thickness may be systemic rather than localised. It is suggested that one such factor known to affect the artery wall is transmural pressure. This is modulated

during exercise as a result of generalized changes in BP. However, it is added that the mechanisms responsible are not completely well defined.

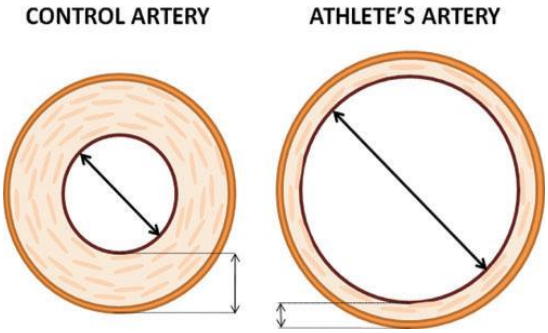

Figure 3. The difference in structure between an athlete's artery and a control subject's artery (Green et al., 2012).

Furthermore, strong evidence exists that the decreases in cardiovascular risk associated with exercise training are due to up-regulation of endothelium-mediated vasodilator function and improvements in arterial compliance (Green et al., 2010). It is reported that enhanced vessel function is observed in both trained and untrained limbs (Green et al., 2010). Improvements in these factors correspond with Green et al. (2012) research suggesting that exercise has a beneficial systemic effect, which may relate to distinctive patterns of blood flow and shear stress. It is mentioned that such changes in shear and upregulation of the vasodilator pathways are also related to changes in arterial vasodilator capacity and wall-to-lumen ratios.

In addition, studies show that chronic aerobic training reduces both SBP and DBP of an individual at rest. Chronic aerobic exercise may prevent or delay the development of hypertension (SBP > 140 mmHg and DBP > 90 mmHg) which is associated with cardiovascular disease (Liu et al., 2012). Due to this, regular physical activity has been broadly recommended by current American and European hypertension guidelines. It states that hypertensive individuals are encouraged to engage in aerobic exercise on a regular basis in activities such as walking, jogging or swimming for 30 to 40 mins daily (Dimeo et al., 2012).

Furthermore, according to Naylor et al. (2008), chronic aerobic exercise training exhibits adaptations such as cardiac enlargement of the myocardium to allow for increased maximal stroke volume (SV) and cardiac output (CO). This is coupled with eccentric left ventricular hypertrophy due to being subjected to increases in cardiac preload (Naylor et al., 2008). It is elaborated that this is manifested as an increase in left ventricular cavity dimensions and a proportional increase in left ventricle wall thickness to normalise myocardial strain. Due to this it can be said that trained individuals possess a lower resting HR than sedentary as the above adaptation allows for an increase in SV. This increase in SV then leads to an increase in CO, therefore the heart does not have to beat as much to achieve the same CO resulting in a lower resting HR (Green et al., 2010; McArdle et al., 2010; Green et al., 2012; Thijssen et al., 2012).

Coinciding with the above, Green et al. (2012) proposed that chronic aerobic training increases maximal CO and decreases mean arterial pressure. It is explained that training reduces total peripheral resistance at maximal exercise and that the capacity for vasodilation in skeletal muscle after training exceeds that of CO to maintain BP. Other vascular adaptations which endurance trained individuals possess are enhanced peak vasodilator capacity, enlargement of conduit arteries, and an enhancement in basal arterial tone (Green et al., 2012). It is evidenced that athletes' arteries undergo increases in total cross-sectional area as peak limb blood flow responses have shown to be enhanced (Green et al., 2012). In correspondence, arterial enlargement is associated with repetitive episodic increases in arterial shear stress which evokes endothelium-mediated remodelling (Figure 4) (Green et al., 2012). It is theorised that this structural remodelling may play a role in accommodating the increase in CO apparent in endurance athletes.

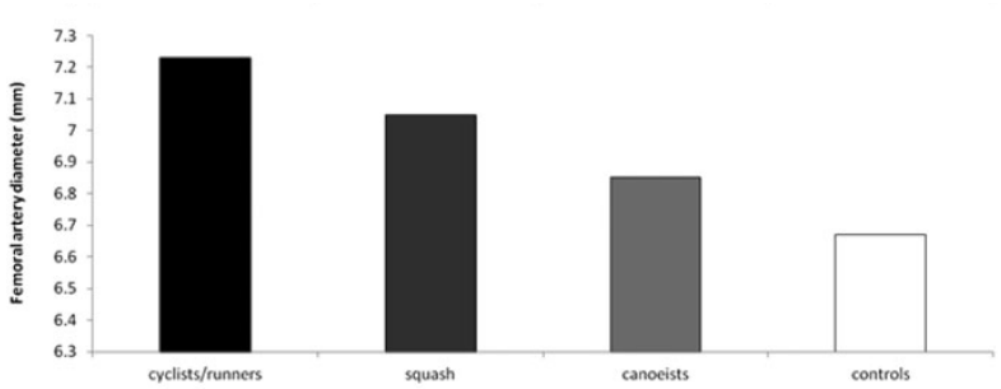

Figure 4. Shows that athlete's arteries have undergone remodelling displaying larger artery diameters in comparison to control subjects. Also displays that greater endurance training leads to greater arterial diameter adaptation (Green et al., 2012).

Furthermore, endurance-trained men have been found to possess a reduction in venous compliance that is preserved to a greater extent than in their sedentary peers (Kawano et al., 2010). This suggests that habitual endurance training may reduce the tolerance response to orthostatic challenge and decrease risk of venous diseases (Kawano et al., 2010).

However critically, although research suggests there are vascular adaptations associated with chronic aerobic training. Most fail to conclude whether these effects are experienced locally within the vessels surrounding the muscles that are used most frequently, or can they be observed systemically in all vessels throughout the body. Consequently, Thijssen et al. (2012) has shown that although an elite squash player possesses vascular adaptation within the peripheral arteries, the adaptation is not to the same extent within the carotid artery (Figure 5). This suggests that the beneficial effects of exercise, especially within the vasculature may be greater on a local scale, in comparison to systemic.

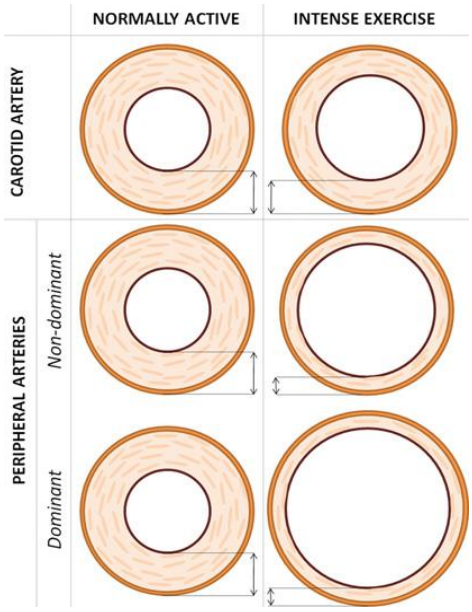

Figure 5. Displays that vascular adaptation is not to the same extent between peripheral arteries and the carotid (Thijssen et al., 2012).

Therefore, it is evident there is a substantial amount of literature conducted on the vascular responses to exercise, and that aerobic exercise is considered highly beneficial for cardiovascular health. However, other forms of exercise exist such as RE which is becoming increasingly popular amongst the population. Although there is great academic debate over whether the effects it possesses upon vasculature are positive or negative.

2.1 Vascular Responses to Resistance Exercise

According to Miyachi et al. (2004), RE has gained widespread acceptance in exercise prescription and has become an integral component in the comprehensive health programme endorsed by the major health organisations. These recommendations are based on the attenuation of osteoporosis and sarcopenia and related risks including falling and functional disability. In addition, others have mentioned that when appropriately prescribed and supervised, RE has favourable effects on different aspects of health such as muscular strength, functional capacity, psychosocial well-being as well as the positive impact on cardiovascular risk factors (Umpierre et al., 2007). Furthermore, it can also be stated that RE can help prevent diabetes mellitus, cardiovascular diseases and

weight gain (Kawano et al., 2010), and promotes muscle hypertrophy and strength gain, improvements in insulin sensitivity and increases in basal femoral blood flow (Tanimoto et al., 2009).

However, the current literature is very contradicting as in contrast it is also stated that acute and chronic RE leads to arterial distensibility (Collier et al., 2010). It is mentioned that non-distensible arteries contribute to increased peripheral resistance, higher pulse pressures and increased ventricular afterload (Collier et al., 2010). According to Miyachi et al. (2004), this leads to elevations in SBP, left ventricular hypertrophy, coronary ischemic disease and reductions in arterial baroreflex sensitivity. This is of clinical importance as increases in the stiffness of central elastic arteries, such as the carotid artery and aorta, has been associated with increased mortality and morbidity and is also now recognized as an independent risk factor for cardiovascular disease (Collier et al., 2010).

2.1.1 Acute Responses

Acutely, according to Jurva et al. (2006), both conditioned weight lifters and non-weight lifters experience an increase in HR, an increase in peak change in flow velocity and an increase in SBP during RE. However, it is displayed that there was an increased change in FMD after exercise for the conditioned weight lifting group, but a reduction for the non-weight lifting group. But there was no significant change in artery diameter for both groups (Figure 6). However, critically the author suggests that some subjects were taking dietary supplements that may have independent effects on endothelial function, and thus highlighting a weakness in the study. This could be a contributing factor for why there was no significant response in artery diameter and why there was a reduction in FMD experienced after exercise by the non-weight lifting group. Furthermore, excluding the latter suggestion, the lack of response in change of artery diameter warrants further investigation into how physically demanding the RE was for the participants. Perhaps if the RE was made more physically demanding a greater response may have been observed.

	Weight Lifters	Non-Weight Lifters
Maximum weight lifted (lbs)*	439 ± 58	213 ± 29
Maximum SBP (mm Hg)*	213 ± 8	196 ± 4
Increase in SBP during exercise (mm Hg)	95 ± 9	78 ± 5
Resting HR after exercise (beats/min)	68 ± 4	61 ± 3
Baseline artery diameter (mm)	4.3 ± 0.2	4.0 ± 0.2
Post-exercise artery diameter (mm)	4.2 ± 0.2	4.0 ± 0.2
Change in FMD after exercise (%)*	1.4 ± 1.1	−4.7 ± 1.1
Pre-exercise peak change in flow velocity (%)	71 ± 8	65 ± 10
Post-exercise peak change in flow velocity (%)	83 ± 12	64 ± 8

Figure 6. Hemodynamic and vascular responses to RT (Jurva et al., 2006).

Furthermore, MacDougall et al. (1985) states that when subjects perform RE, the mechanical compression of blood vessels with each contraction combines with a potent pressor response and a valsalva response. This will produce extreme elevations in SBP and DBP. It is suggested that these elevations are extreme even when exercise is performed with a relatively small muscle mass.

In addition, Jurva et al., (2006) reports that weightlifting elevates SBP up to 400mmHg, possibly impairing endothelial function. It has been demonstrated that <5 mins of moderate elevations in perfusion pressure reduces conduit coronary arterial endothelial function for up to 2.5 hrs. Therefore, agreeing with reports by Kawano et al. (2010) who suggests that RT possesses negative effects on vascular function causing arterial stiffening and high BP.

Although, in contrast study's by O'Connor et al. (1993) and Roltsch et al. (2001) reported that BP does not change after a period of acute RE, and DeVan et al., (2005) also concludes similar findings. The author reported that acutely after RE, brachial artery BP displays no significant difference compared to baseline. However, DeVan et al., (2005) also found that in contrast to no change observed in brachial BP, carotid BP displayed an increased response. The author expands stating that the results obtained suggests that the measurements of central BP may reveal the vascular effects of RE that may not be unmasked by routine brachial BP assessments, and therefore provides a platform for further research. This aspect may have an effect upon how BP assessments are carried out in a clinical setting.

Furthermore, DeVan et al. (2005) concluded that measures of arterial compliance decrease after acute RE, but these measures returned to baseline levels 60-150 min post RE. Thus, it can be said that the acute effects of RE on the vasculature appear to be transient in nature, lasting no longer than 1 hr. DeVan et al. (2005) states that it is not clear why arterial stiffness is elevated acutely after RE, however it is possible that the increase in central arterial stiffness and decrease in central arterial compliance with one bout of RE may be due to an epiphenomenon of corresponding BP changes.

2.1.2 Chronic Responses

Chronically, Jurva et al. (2006) stated that acute RE associated with hypertension impairs endothelial function in unconditioned subjects and that chronic RT protects against this vascular dysfunction. Possible explanations for the observed results could be that exercise increases CO and therefore shear stress, stimulating NO production (Thijssen et al., 2010). Alternatively, NO-independent mechanisms of dilation resistant to acute hypertension may prevail in the conditioned weight lifting subjects (Jurva et al., 2006). Conversely, it is mentioned that it may be due to an improved vascular antioxidant capacity as a result of chronic endurance exercise training (Jurva et al., 2006).

In addition, it has been discovered that chronically resistance trained men possess greater forearm venous compliance than sedentary individuals of the same age (Kawano et al., 2010). This may be explained by the resistance trained men acquiring greater forearm venous capacitance as a result of vascular adaptation due to chronic training (Kawano et al., 2010). It has been outlined by Green et al. (2010) that the repeated exposures to high levels of shear stress and BP experienced with chronic RT causes the signalling for vascular adaptation. Therefore, the latter suggestion may account for the differences in venous compliance observed.

Furthermore, according to Umpierre et al. (2007) recent evidence does not support the ancient dogma that RT elevates resting BP. It is explained that this originated from the idea that greater pressure gains associated with RE, due to the greater isometric component, would lead to a chronic elevation of BP. However, it is now believed that RE demonstrates a beneficial effect on resting systolic and diastolic arterial pressure (Umpierre et al., 2007).

Although in contrast, according to Kawano et al. (2010), increased arterial stiffness is a result of marked pressor responses during RE via ischemia of capillaries caused by muscle contraction. It is explained that venous pressure may be increased during RE leading to an enhancement of mechanical stress. In addition, it is reported that resistance trained men show tolerance of cardiovascular responses to lower body negative pressure, therefore it is possible that habitual RT may decrease venous compliance (Kawano et al., 2010).

However, contradicting with the above, due to the increased number of capillaries with RT, muscle hypertrophy caused by chronic RT may induce greater volume and density in veins, leading to greater venous capacitance (Kawano et al., 2010). Therefore, as greater venous compliance is associated with the larger venous volume induced by functional muscular hypertrophy, habitual RT may increase venous compliance. Greater venous compliance may then be associated with higher venous capacitance and muscle mass (Kawano et al., 2010).

Although, despite the above Miyachi et al. (2004) concluded that firstly, several months of RT significantly reduces central arterial compliance in healthy men and secondly, the reduced arterial compliance returned to the baseline levels a few months after the cessation of RT. This confirms that the change in central arterial compliance was an effect of RE and thereby proposes an argument against Kawano et al. 2010 suggestions.

Therefore, it is evident there is great academic debate on the area and it can be seen that the literature is relatively contradicting. However, perhaps research needs to evolve and follow the route taken by aerobic exercise by experimenting with different types of RE. On the whole, the majority of these studies implemented exercises that recruited large amounts of muscle mass. However,

there is not much research available on the effects of smaller forms providing a gap for investigation.

2.1.3 The Use of Handgrip as a Resistance Exercise

Significantly less research has been conducted on the effects of lower forms of RE such as handgrip upon vasculature. This may be due to handgrip being viewed as an exercise that is not adequately demanding to cause enough of a physiological response that can be measured, especially when larger forms such as barbell squats and bench press exist. These exercises recruit a larger amount of muscle mass which therefore provides a greater observable response in comparison. However, according to Green et al. (2012), a few recent studies have used partial cuff occlusion for unilateral manipulation of blood flow during bilateral handgrip exercise training bouts. They resulted in changes in hyperaemic flows and conduit artery dilation that were shear stress dependent. These studies implicate changes in shear as a principle physiological stimulus to adaptation and vascular remodelling in response to exercise training. To expand, other handgrip studies have indicated that localized resistance vessel remodelling occurs, largely independent of skeletal muscle hypertrophy, sympathetic or circulatory influences. (Green et al., 2012).

Furthermore, a study carried out by Shoemaker et al. (1997) used handgrip as RE to see whether the small muscle mass recruiting exercise had any systemic effects. It was concluded that beat-by-beat measures of both mean blood velocity and diameter of the brachial artery respond in a complex dynamic manner during rhythmic exercise. It was stated that conduit artery dilation may attenuate the pressure drop across the vascular bed, thereby facilitating flow distribution and capillary perfusion.

Consequently, authors such as Green et al. (2010), state that studies which incorporate small muscle group exercises such as handgrip are beneficial. This is the case as they provide insight into localized effects of exercise that are less dependent upon central regulatory or neural changes. Green et al. (2010) study proposes further evidence for the need for greater investigation into the use of handgrip. As can be seen in Figure 7, handgrip training displays that vascular

adaptation occurs as FMD, which is an index of endothelial function, adapts rapidly to training and then returns to baseline levels. This study suggests that a repeated increase in shear stress is obligatory for adaptation of conduit arterial function in response to exercise training.

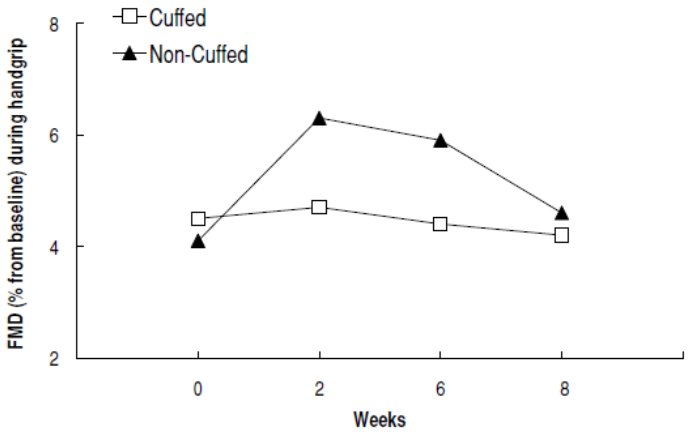

Figure 7. Time course of adaptation in arterial function in response to exercise training and the impact of shear stress modulation on this response (Green et al., 2010).

2.2 Summary

It is evident there is great academic debate over the vascular responses to RE. However, due to the conflicting theories and in order to evaluate these contrasting hypotheses, further research needs to be carried out in this area. There is also limited literature available on the acute vascular effects of lower forms of RE such as handgrip, and a few studies (Shoemaker et al., 1997; Green et al., 2010; Green et al., 2012) have suggested their importance providing a need for further investigation. This therefore highlights a gap within the literature, giving preference and providing justification for this study.

2.3 Aims and Objectives

The aim of the present study was to examine the acute vascular responses following a single bout of handgrip RE. The working hypothesis was that subjects will display an increase in HR, both SBP and DBP, and in all vascular measures of BMD, RMD, DD, DP, TP, BMEV, RMEV, BMSR and RMSR. It is hypothesised that acutely subjects will not experience vascular stiffness or a reduction in venous compliance, but instead will witness vasodilation of the artery due to the release of NO brought on by the shear stress stimulus.

CHAPTER III

METHODOLOGY

3.0 Participants

Participants were 9 physically active (according to ACSM guidelines; appendix A), male students that volunteered from Cardiff Metropolitan University. The average physical characteristics (± standard error of the mean) were, age (20.4 ± 1.1 years); height (1.78 ± 0.1 cm); weight (79.3 ± 9.2 kg) and BMI (24.9 ± 2.5). Males were chosen as females would have to be tested, ideally, within days 1-7 of the menstrual cycle. This is when concentrations of circulating female sex hormones are lowest and will have less of an effect upon vascular endothelial function. Therefore, finding female participants that were willing to be tested during this period would increase the difficulty of testing (Thijssen et al., 2010). Subjects were non-smokers, normotensive and non-obese thereby less likely to have any known cardiovascular diseases or risk factors that could affect the results (Okamoto et al., 2009; Tanimoto et al., 2009). This aspect was confirmed through the use of a carefully constructed Par-Q questionnaire, carried out by subjects prior to testing (Appendix B). Participants also had to satisfy the criteria of being not weight trained to reduce the chances of the response being influenced by adaptations due to training (Jurva et al., 2006). 9 subjects were selected providing an adequate amount of data to analyse. This allowed for a thorough analysis, increasing the reliability and validity of the results and therefore the study.

3.1 Procedure

The study was separated into two testing days. For the duration of the testing day, prior to testing, subjects were asked to refrain from caffeine and alcohol ingestion or any physical activity. This reduced the chances of external factors influencing the results (Shoemaker et al., 1997; Okamoto et al., 2009; Thijssen et al., 2010).

3.1.1 Testing Day 1

Prior to testing, subjects were presented with a participant information sheet (Appendix C), briefly providing an overview of the study as well as highlighting their rights and requirements. Also, they were asked to answer a general Par-Q questionnaire to assess their level of health, and those that possessed characteristics of cardiovascular risk factors were excluded. Subjects were informed of the experimental risks and were asked to sign an informed consent document (Appendix D).

Testing day 1 was used as a familiarisation session and to collect pre-testing data. Participants were introduced to the lab environment to allow them to become familiar with the equipment. This was done to ensure comfort and to give an insight into what was required. After completion of the paper work, participant's height was recorded for profiling (Appendix D) using a wall mounted stadiometer (Holtain Fixed Stadiometer, Holtain LTD, Crosswell, Crymych, Pembs, UK). They were then asked to lie down on the bed with their right arm out to the side resting upon a pillow. A handgrip dynamometer (Grip-A Model 5001, Takei Scientific Instruments Co Ltd, 1-6-18, Hatanodai, Shinagawa-ku, Tokyo, Japan) was placed in their right hand and they were shown how to carry out a 1RM test. This was done so that they understood the technique required to carry out the exercise successfully. Subjects were asked to carry out 3 1RM tests with 1 min rest between each attempt. Rest periods were timed using a stop watch (Fastime 5 Dual Display Stopwatch, The Old Vicarage, Station Road, Ashby-de-la-Zouch, Leicestershire, England). The mean of the three scores was then calculated to give the average. 10% of the average was then calculated in order to work out the amount of weight (kg) to be used during the RE (Shoemaker et al., 1997). A set weight was not used as each subject possessed different levels of strength and

therefore required the correct amount of weight in relation to their degree of strength, otherwise the responses observed may not be accurate and thereby deeming it an unfair test. The weight was then placed upon the modified handgrip dynamometer in preparation for the RE taking place on testing day 2. To further increase awareness, subjects were also given use of the modified handgrip dynamometer with the correct weight added. They were asked to exercise for 10 sec controlled by a metronome (Seiko SQ50, Seiko UK Ltd, SC House, Varnwall Road, Maidenhead, Berks, UK) that would ‘beep’ in 1 sec intervals. Subjects were told that they were to contract on the first ‘beep’ and relax on the next. It was outlined that this was the contraction to relaxation pattern that they must follow for 5 mins on the testing day. This was done to acquire a sense of the degree of stress it would place upon them.

In addition, participants had the occlusion cuff (Hokanson, D.E Hokanson Inc, Bellevue, WA, USA) fitted to their right forearm just below the elbow distal from the measured artery. It was then inflated to 220 mmHg for 10 secs to allow them to become accustomed to the amount of pressure that would be placed upon them during testing, and to witness the effects of how it would feel. Participants also had 3 electrocardiogram (ECG) cables (Finometer Pro, Finapres Measurement Systems, Amhem, Netherlands) connected to patches placed in 3 areas of their torso. Two placed below the clavicle near the right and left shoulder, and one at the bottom of the left side rib cage. It was explained that the ECG would measure HR throughout the study. This along with a BP cuff attached to their left arm and a finger cuff attached to their left hands middle finger were attached to a finometer (Finometer Pro, Finapres Measurement Systems, Amhem, Netherlands). It was explained that this would measure BP throughout the study. The finometer was turned on for a short period of time to allow the subject to experience the BP cuff inflating and the finger cuff pulsating. It was explained that both cuffs would do this throughout the testing and why.

Testing day 1 was considered important as it provided first-hand experience of what was required of the participants, and to reassure that they were not going to be placed in any harm. It also gave subjects the chance to pull out if they were not comfortable with any part. During the session participants were encouraged to ask any questions and mention any doubts or worries they had regarding the procedure. The sole purpose was to ensure participants were fully comfortable and aware of what was involved, their requirements, and to highlight that they were not going to experience any harm or be placed in any danger. This was done in attempt to reduce the effects of white coat syndrome as anxiety of testing can lead to elevations in HR and BP that can have an effect upon the study's results (Mancia et al., 2009).

3.1.2 Testing Day 2

Firstly, Subjects' weight was measured using digital weighing scales (SECA-Model 770, Vogel & Halke, Hamburg, Germany) and calculation of BMI (weight (kg)/height (m) = answer/height (m) = BMI) was carried out for participant profiling (Appendix D). Subjects were then asked to lie down on a bed in the supine position as this would reduce the contributions of changes in mean arterial pressure and cardiac filling pressure during exercise (Shoemaker et al., 1997; Thijssen et al., 2010). A cushion was placed beneath their head for comfort and subjects placed their right arm out to the side, resting it on another cushion placed upon a table (neither higher nor lower than their heart) (Thijssen et al., 2010). 3 electrocardiogram (ECG) cables were connected to 3 patches fitted to their torso, and a BP cuff was fitted to their left arm and a finger cuff attached to their left hands middle finger (as on testing day 1). The ECG cables, BP cuff and finger cuff were all attached to a finometer (Shoemaker et al., 1997). A pressure occlusion cuff was also fitted to their right arm distal from the measured artery (Thijssen et al., 2010). Participants were then left to rest quietly in the supine position for 10 mins. This was done as acute sympathetic nervous system activation can alter FMD (Thijssen et al., 2010).

Following the resting period, ultrasound gel was placed upon an ultrasound probe (Ultrasound, Vi Vidq, GE Healthcare, Amersham, UK) which was then used to scan the brachial artery of the right arm (Thijssen et al., 2010). The imaging probe was hand held so that the probe could be manipulated to track the artery (Shoemaker et al., 1997). Baseline measures of HR via ECG, SBP and DBP via the finometer and vascular measures such of BMD, RMD, DD, DP, TP, BMEV, RMEV, BMSR and RMSR via the ultrasound machine, were recorded and saved onto an attached laptop for 1 min (Thijssen et al., 2010). The pressure cuff was then inflated to 220 mmHg and remained inflated for 5 mins, creating an occlusion (Thijssen et al., 2010). The cuff was then deflated and HR, BP and vascular measures were collected for 3 mins. This was the case as according to Thijssen et al. (2010), measurements should be performed for ≥3 mins in upper limb arteries.

The modified handgrip dynamometer containing 10% of the subjects 1RM in kg (previously calculated on testing day 1) was then clamped onto the table. It was positioned so that the subject's dominant hand reached the station without having to stretch uncomfortably. This was important as if the participant was to move to acquire comfort, they could affect the accuracy of vascular scanning and could cause an increase in HR and BP that was not related to the study. Participants were asked to carry out a single bout of handgrip RE with their right hand using the modified handgrip dynamometer. This involved squeezing a hand lever which lifted and lowered a 10% maximal voluntary isometric contraction weight a distance of 5 cm (Shoemaker et al., 1997). They exercised for one set of 5 mins where they were told to contract for 1 sec and relax for 1 sec (Shoemaker et al., 1997). This ratio was controlled using a metronome including an attached speaker to amplify the sound. The metronome 'beeped' in 1 sec intervals and so the subjects were asked to pick their 'beep' and contract to start the exercise, at which point the stop watch was started. Participants were briefed at 1 min intervals on how much time was remaining until the completion of the exercise.

Ultrasound gel was then once again placed upon the ultrasound probe which was placed on the subject's right arm's brachial artery for vascular scanning (Thijssen et al., 2010). Participants were allowed 45 secs to rest post RE allowing for vascular responses to take effect (Shoemaker et al., 1997). Measures of HR, BP and vascular measures were then taken for 1 min as before (Thijssen et al., 2010). The pressure cuff was then inflated again to 220 mmHg for 5 mins causing an occlusion (Thijssen et al., 2010). It was then deflated and HR, BP and vascular measures were measured for 3 mins once again via the ECG, finometer and the ultrasound machine (Thijssen et al., 2010).

After completion of the testing, it was important to explain to the subject that they were to gradually sit up and then stand up, otherwise they may become light headed, dizzy or faint as they had been lying down for an extended period of time. The data collected by the ultrasound was recorded and saved to an attached laptop. This data was then input into Blood Flow Analysis Software and analysed. The ultrasonography data along with HR recordings from the ECG and BP recordings that were saved on a laptop attached to the finometer, will be gathered and presented in Microsoft Excel. The post intervention measures collected were then compared to the baseline measures to analyse the change in vascular responses as a result of the handgrip RE intervention.

To enhance the accuracy of all measures taken within the study, the experimenters practised and piloted the exercise protocol on subjects prior to data collection (Shoemaker et al., 1997). In addition, tests were performed at a room temperature of 21-23°C (Shoemaker et al., 1997) and testing days were separated by at least 24hr to fully allow for vascular responses to return to resting levels. All tests were also carried out in the same laboratory and by the same testers.

3.2 Statistical Analysis

All data from Microsoft Excel was input into and analysed using a specialist statistical analysis computer programme (SPSS, Version 20, Chicago, IL). Paired t-tests were conducted on the variables in order to compare baseline measures with the measures taken post RE handgrip intervention to determine the significance of the response. Measures were considered statistically significant if a ($P<0.05$) was obtained. This was done to observe what effect the RE intervention had upon the vasculature in comparison to without (baseline). Therefore, to determine what the acute vascular response to a single bout of handgrip RE was.

CHAPTER IV

RESULTS

4.0 Ultrasonography Data

Paired t-tests revealed no significant difference between baseline and post handgrip RE intervention within the following variables, BMD, RMD, DD, DP, TP, RMEV and RMSR (P>0.05; Table 1).

Table 1. Mean ± SD values for vascular data collected by ultrasound and finometer at baseline and post handgrip RE intervention.

Variables	Baseline	Post Handgrip Intervention
BMD (cm)	0.20 ± 0.04	0.21 ± 0.04
RMD (cm)	0.20 ± 0.03	0.21 ± 0.04
DD (cm)	0.02 ± 0.01	0.02 ± 0.01
DP (%)	10.8 ± 5	10.7 ± 3.8
TP (sec)	81 ± 55.1	75.8 ± 31.7
*BMEV (cm/s)	7.3 ± 2.2	11.1 ± 3.4
RMEV (cm/s)	13.6 ± 5.7	17.6 ± 4.2
*BMSR (1/s)	145.6 ± 41.8	214.8 ± 73.1
RMSR (1/s)	275.3 ± 116.6	342.8 ± 94
Base Mean SBP (mmHg)	127.82 ± 6.92	130.34 ± 10.53
Base Mean DBP (mmHg)	67.85 ± 9.18	69.51 ± 11.47
Base Mean HR (BPM)	63.11 ± 7.80	62.22 ± 7.98
FMD Mean SBP (mmHg)	130.96 ± 11.18	132.19 ± 9.15
FMD Mean DBP (mmHg)	71.96 ± 11.03	72.16 ± 9.37
FMD Mean HR (BPM)	63.78 ± 7.31	63.44 ± 6.35

*Indicating variables considered having a statistical significant difference between baseline and post handgrip RE intervention.

However, paired t-tests did reveal BMEV (Figure 8) and BMSR (Figure 9) displayed a significant difference between baseline and post handgrip RE intervention (P<0.05).

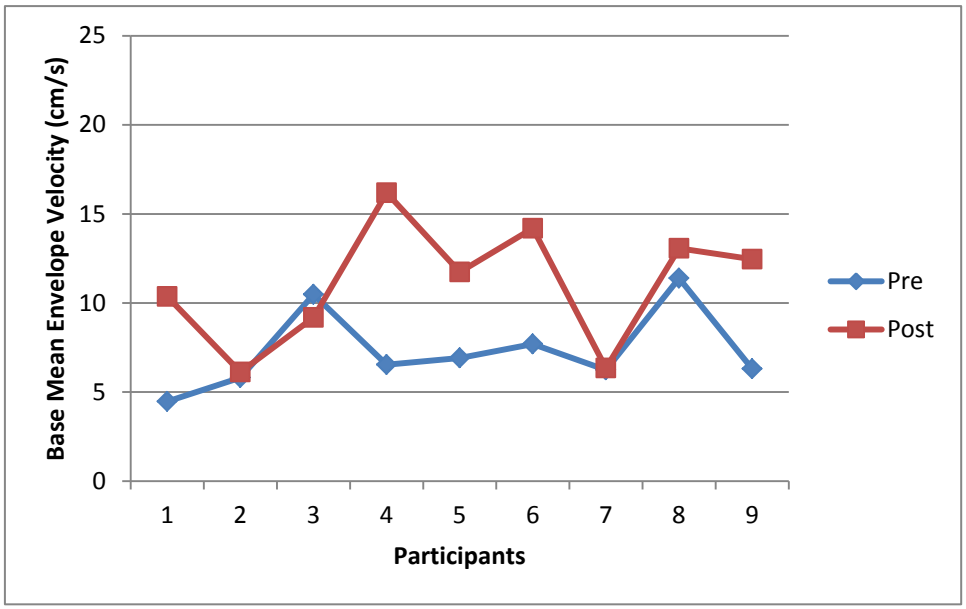

Figure 8. Line graph displaying the general increase in BMEV for participants between pre and post intervention measures.

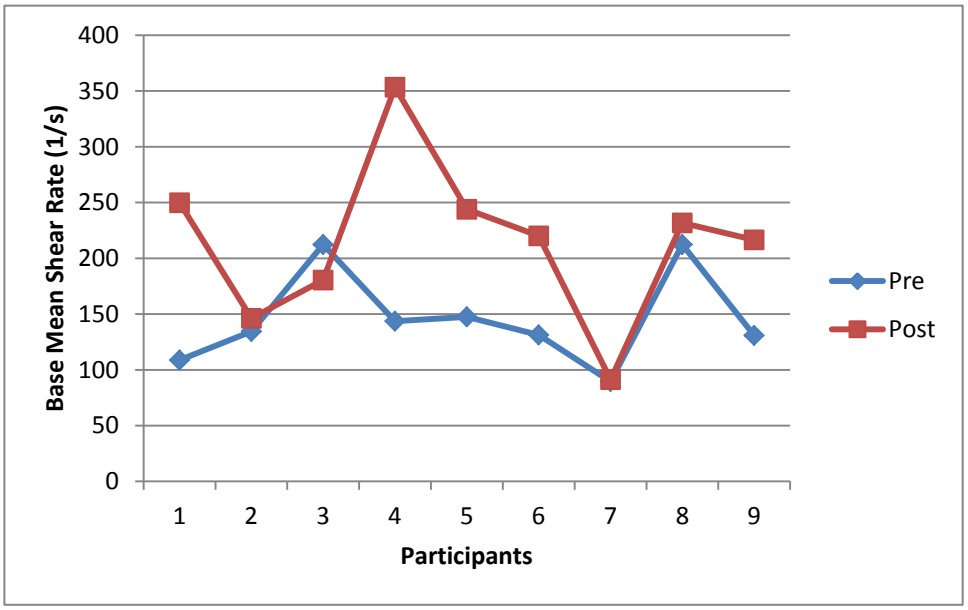

Figure 9. Line graph displaying the general increase in BMSR for participants between pre and post intervention measures.

4.1 Blood Pressure and Heart Rate Data

Paired t-tests revealed no significant difference between baseline and post handgrip RE intervention within the following variables, base mean SBP, base mean DBP, base mean HR, FMD mean SBP, FMD mean DBP and FMD mean HR ($P>0.05$; Table 1).

CHAPTER V

DISCUSSION

5.0 Experimental Findings

The aim of the investigation was to establish how the vasculature responds acutely to a single bout of handgrip RE, with the hypothesis that all vascular measures would display an increase in their response post exercise intervention, resulting in a vasodilatory reaction. However, only two variables (Figures 8 & 9) presented a response that was considered statistically significant ($P<0.05$). Although, from the variables considered to have no significant change, the following did present an increased response to the intervention, BMD = 5%, RMD = 5%, RMEV = 29.4%, RMSR = 25%, base mean SBP = 2%, base mean DBP = 2.4%, FMD mean SBP = 1%, FMD mean DBP = 0.3%. However, the handgrip RE was not physically demanding enough to cause an increase in these variables great enough for them to be considered statistically significant. As a result, these responses were not great enough to cause a vasodilatory response and therefore did not relate to the study's initial hypothesis.

5.1 Ultrasonography Data

The most novel findings of the study were the significant increases reported in BMEV and BMSR ($P<0.05$; Table 1). The increase in BMEV (increased velocity of blood flow) suggests that there is an increase in blood flow through the artery post RE intervention in comparison to baseline. This relates to research by Collier et al. (2010) where it was reported that an increase in blood flow after RE was ($P<0.05$, 0.44). Collier et al. (2010) concluded that this significant increase in blood flow suggests that there is an increase in vasodilator capacity. Consequently, the increase in BMSR suggests that the increased blood flow is causing an increase in the amount of resistance the blood flow is exerting upon the vessel walls (Ene-lordachne et al., 2003) and relates to current literature (Green et al., 2010; Thijssen et al., 2012) where shear stress is considered the main stimulus that signals vasodilation of an artery. Possible explanations for the responses observed can be attributed to the effects caused by the RE. Exercise places a demand upon the body and therefore increases HR (Appendix F) to cope with that demand (Hamer et al., 2012). As a result, there is an increase in SV leading to an increase in CO causing an increase in blood flow through the vessels of the body (McArdle et al., 2010). This increased blood flow through the vessels then forms a

tractive force which acts on the vessel walls, known as shear stress (Ene-lordachne et al., 2003). This increase in shear stress stimulates NO production which is then released from the endothelial cells within the artery walls into the smooth muscle cells causing them to experience a relaxation effect, leading to vasodilation of the artery (Thijssen et al., 2010). Therefore, as shear stress during the exercise mediates NO signalling and release, which has been outlined as a flow-induced dilator (Thijssen et al., 2010), it can be said that up-regulation of this endothelium-dependent dilator causes an increase in reactive hyperaemia induced flow (Collier et al., 2010), similar to what was observed in this study. Thus, the increase in shear rate is a result of the increase in blood flow through the artery and therefore correlates with the other previously mentioned significant finding of an increase in BMEV. According to Ene-lordachne et al. (2003); Green et al. (2010); Thijssen et al. (2012) this mechanism is what influences endothelial cell function to cause vasodilation of the artery. Therefore, It can be said that the increased blood flow brought on by RE, leads to vasodilation of the brachial artery and therefore supports the notion of an increase in vasodilator capacity proposed by Collier et al. (2010).

Although, in contrast BMD and RMD did not display a significant response within this study ($P>0.05$; Table 1), and thereby proposes an argument against the latter suggestion. However, possible explanations could be attributed to the RE not being physically demanding enough to bring about the required increase in diameter response. Other studies such as (Miyachi et al., 2004; Okamoto et al., 2007; Tanimoto et al., 2009; Okamoto et al., 2010; Collier et al., 2010) involved a larger RE such as bench press, leg press or squats that required recruitment of larger amounts of muscle mass. These studies reported significant changes in artery diameter resulting in an increase in arterial stiffness and a reduction in arterial dispensability. This may be due to their greater muscular recruiting RE causing more stress upon the participants resulting in a greater response. As the larger RE is more challenging, the participants HR would show a greater increase causing a greater increase in CO, which has been outlined by Jurva et al., (2006) to result in an increase in shear stress. This greater increase in CO would result in a greater increase in shear stress which may then cause a greater response of NO signalling resulting in a greater vasodilatory response. However, alternatively

in closer correlation with the current literature (Miyachi et al., 2004; DeVan et al., 2005; Jurva et al., 2006; Okamoto et al., 2009; Collier et al., 2010; Kawano et al., 2010; Phillips et al., 2011), the greater response caused by a larger RE may have caused SBP to rise high enough to cause endothelial impairment (Jurva et al., 2006), leading to a reduction in venous compliance and an increase in arterial stiffness.

The current study's BP and HR data support this notion as their response was also considered statistically non-significant ($P>0.05$; Table 1), thereby suggesting that the participants found the RE relatively non-strenuous. Exercise increases HR which increases blood flow through the vessels due to an increased CO. This increased blood flow then exerts greater pressure upon the vessel walls leading to a rise in BP (McArdle et al., 2010). The participants within this study did not display a significant increase in BP or HR following the RE, thereby suggesting the RE was not difficult enough to cause this effect. Therefore, as outlined above, if the present study would have adopted a more physically demanding RE such as a bench press, leg press or squats, perhaps a greater response in closer correlation with the literature may have been recorded.

Furthermore, Jurva et al. (2006) stated that arterial stiffness is caused by impairment of endothelial function due to weight training causing elevations in SBP up to 400 mmHg. The present study did not display measures of SBP anywhere near this value (Table 1) and therefore warrants further suggestion that the handgrip RE was not adequately demanding to cause a significant response. Although, it is important to note that non-invasive measures of BP during RE underestimate direct intra-arterial measurements, therefore the estimations of stimulus intensity may be low (Jurva et al., 2006). However, other explanations may be that even though the participants in the present study were not weight trained, perhaps some participants may be slightly more endurance trained than anticipated. This can account for the lack of response as vascular antioxidant capacity may be improved by chronic endurance exercise training, and shear stress up-regulates superoxide dismutase expression and activity in vascular endothelium (Jurva et al., 2006).

In support of the previous point, Participant 8 displayed characteristics of chronic adaptation within his artery. When analysing the ultrasonography data using the Blood Flow Analysis Software, it could clearly be seen that participant 8's brachial artery was much larger in size (in terms of lumen diameter and wall thickness) in comparison with the rest of the group. Although participant 8 may not engage in weight training, he was a competing gymnast and therefore his vascular system has been exposed to an increased amount of regular stress in comparison to the rest of the group, and thus has undergone vascular adaptation. This relates to findings by Green et al. (2012) where he reported that athletes' arteries are larger in size in comparison to healthy non-athletic control subjects. According to Green et al. (2012) the repeated exposures to high levels of shear stress and BP experienced with chronic exercise training cause the signalling for vascular adaptation. This explanation correlates with Participant 8's training background and may account for the larger size of his brachial artery in comparison to the other participants. Therefore, this aspect may have affected his response to the RE as outlined by Jurva et al. (2006), endothelium-mediated vasodilation is impaired by acute exposure to RE in healthy subjects, however no impairment exists in conditioned individuals. Thus, according to Jurva et al. (2006) participant 8 would not experience any change, or as much of a change in artery diameter in comparison to the rest of the group. This is the case as although he is not weight trained, gymnastics involves a lot of body weight training which can be considered as a form of conditioning. Therefore, the results for participants 8 may have had an effect upon the group mean and can be considered a contributing factor for why there was no significant change in artery diameter post RE.

In addition, participant 3's results also warrant the same argument. It can be seen from figures 8 & 9 that participant 3 displays a different response in BMEV and BMSR in comparison to the rest of the group. In both variables participant 3's pre response is slightly greater than his post intervention response and therefore does not follow the same trend as the other group members. Although participant 3 does not engage in weight training, his sport is rugby, which is a sport that requires a high level of physical strength and fitness, and plays regularly throughout the week. According to Naylor et al. (2008); Green et al. (2010); McArdle et al. (2010); Green et al. (2012); Thijssen et al. (2012), as participant 3

has experienced chronic exercise training, his vascular system may have adapted through mechanisms such as enlargement of the left ventricle cavity and enhancement of the myocardial muscle surrounding the left ventricle leading to an increase in SV which results in a lowering of his HR. This is the case as due to an increased SV his heart does not have to beat as much to achieve the same CO. Therefore, when carrying out the RE within this study his heart did not have to beat as much as the other participants to cope with the demand resulting in a lower blood flow velocity which leads to a reduced shear rate and reduced SBP due to less blood flow placing less stress upon the vessel walls, which may be why his response was much lower than the other participants. Additionally, due to vascular adaptation (Green et al., 2012; Thijssen et al., 2012) his blood vessels may be larger containing a larger lumen diameter and therefore allows more blood to flow through with less resistance, resulting in less shear stress being exerted upon the vessel walls causing less of an NO signalling stimulus leading to less of a vasodilatory response. Therefore, as with participant 8, participant 3 possesses greater vascular adaptation in comparison with the rest of the group, which is an accounting factor for why his response differs.

As a result of the above, it can therefore be said that although there was significant increases reported in vascular measures of BMEV and BMSR, these responses were not great enough to stimulate a significant vasodilation within the participants and therefore did not relate to the study's hypothesis. Possible explanations for this could be that, according to Collier et al. (2010) subjects should experience a significant increase in HR and therefore CO following acute RE, and according to Jurva et al., (2006) this increase in cardiac output leads to an increase in shear stress and an increase in NO signalling leading to vasodilation of the artery. However, the HR data from the present study (Table 1) did not display a significant increase, as although there was an increase during the RE (Appendix F), this increase was very brief and HR swiftly returned to near baseline levels immediately post, explaining why the response was considered to have no significant increase between baseline and post intervention. Due to this, the participants did not experience as much of an increase in CO in comparison to the literature and so therefore did not experience the increased amount of shear

rate to the same extent, resulting in less NO signalling and less of a vasodilatory response.

Furthermore, excluding the significant increases in BMEV and BMSR, the remainder of vascular data collected presented no statistically significant response ($P>0.05$; Table 1). As stated above, the main explanation for this can be attributed to the RE not placing enough of a stress upon the participants, and individual variances within the results that may be a result of independent participant characteristics that have had an effect upon the group mean. Although, the lack of a response in BMD and RMD, and the slight reduction observed in DP (Table 1) relates to findings by Phillips et al. (2011) and warrants the speculation that subjects may have displayed signs of possibly experiencing the onset of endothelial impairment on a small scale caused by the RE. Phillips et al. (2011) reported that brachial artery diameter remained unchanged between baseline (4.6 ± 0.2) and post RE intervention (4.6 ± 0.2), however DP reduced significantly ($P<0.05$) form baseline (8.0 ± 1.0) to post (5.7 ± 0.4) and therefore is similar to the present study's findings. However, although the present study did report no significant changes in BMD and RMD and a slight reduction in DP was observed in correlation with Phillips et al. (2011), the present study's reduction in DP was very small and therefore not considered statistically significant and so disproves the latter suggestion. However, it arises the hypothesis that if a larger, more physically demanding RE was used, it can be speculated that the DP may well have reduced further, making the above suggestion more plausible.

Consequently, it is important to note that from the variables considered to have no statistically significant change, the following did present an increased response to the intervention, BMD = 5%, RMD = 5%, RMEV = 29.4%, RMSR = 25%, base mean SBP = 2%, base mean DBP = 2.4%, FMD mean SBP = 1%, FMD mean DBP = 0.3%. Therefore, all though the response was small, most variables did display a response to the RE. As a result, it can be said to a certain extent that generally vascular measures did appear to relate to the study's hypothesis. However, in contrast these increases were not considered significant, and as a result, disproves the study's hypothesis as they were not great enough to cause

the occurrence of a significant vasodilation as a result of an increase in the shear stress stimulus.

5.2 Blood Pressure and Heart Rate Data

The heart rate and blood pressure data displayed no significant difference ($P>0.05$; Table 1) between baseline and post handgrip intervention and is similar to findings by (O'Connor et al., 1993; Roltsch et al., 2001; DeVan et al., 2005). The results for BP relate with DeVan et al. (2005) findings as it was reported that both brachial artery SBP and DBP show no significant increase post RE. However, DeVan et al. (2005) noted that although there was no significant increase in brachial SBP and DBP, carotid BP within their study displayed a significant difference. It was concluded that the results obtained suggested that the measurements of central blood pressure may reveal the vascular effects of RE that may not be unmasked by routine brachial BP assessments, and therefore warrants further investigation into methods of BP measurement. This suggests that the present study may have observed a greater response in post measures if the measures were recorded from the carotid artery instead of the brachial. However, as with the other non-significant variables, the lack of response can largely be attributed to the RE not being physically demanding enough and thereby placing too little stress upon the participants, resulting in a lack of response being recorded.

However, it should be noted that during the RE itself, all participants did show an increase in HR (Appendix F) which is why participants experienced the increase in BMEV and BMSR. However, this can simply be attributed to the acute effect of any exercise and is to be expected (Phillips et al., 2011; Green et al., 2012; Hamer et al., 2012; Thijssen et al., 2012). As the RE was not stressful enough, post RE HR swiftly reduced nearer resting levels as the RE did not place enough of a demand to require the HR to remain elevated. According to McArdle et al. (2010) post exercise HR remains elevated to aid the removal of bi-products and flush the muscles with oxygen and nutrients. This response did not happen within the present study as the RE did not place enough physical stress upon the participants to require the latter process. Therefore, this is why the HR response between baseline and post RE was considered statistically not significant. The

latter process also explains why it was BMEV and BMSR that increased significantly and RMEV and RMSR did not. As the HR was only elevated for a brief amount of time, it was only the base measures that had the reaction, after this HR swiftly reduced and therefore was not elevated during the time response measures were taken. This provides further evidence that the RE was not physically demanding enough to require HR to remain elevated long enough to allow for the majority of the other vascular measures to react to give a significant response.

Lastly, white coat syndrome is difficult to overcome and can have an effect upon all participants due to the nature of the lab environment (Mancia et al., 2009). This could be considered an accounting factor to why the HR and BP results did not display a significant change, as perhaps participants HR and BP were slightly elevated above normal resting levels to begin with due to anxiety of testing. This may therefore account for why pre and post intervention measures did not differ greatly, thus not allowing for a true comparison between pre and post handgrip intervention measures. This can also be linked to why participant 3's pre BMEV (Figure 8) and BMSR (Figure 9) was recorded as being greater than his post intervention response.

5.3 Limitations of the Study

The key limitations within the present study were that, although two key variables were considered to display a statistically significant response (Figures 8 & 9), the handgrip RE was not a physically demanding enough exercise to cause a significant response in the other measures (Table 1). If the present study was to have adopted a larger RE that required recruitment of a larger amount of muscle mass such as a bench press exercise, leg press or squats, then the participants would have been placed under a greater amount of physiological stress that would have caused a greater response in correlation with the literature.

In addition, it was found that participant 3's BMEV (Figure 8) and BMSR (Figure 9) values did not follow the same trend as the rest of the group as his pre intervention responses were greater than his post. Also when analysing the data, participant 8's artery displayed signs of vascular adaptation as it was much larger

than the other group members. After further inquiry, although they did not engage in weight training, both these participants were regularly competing athletes that carried out physical activity levels much greater than the other participants, and thereby possessed greater vascular adaptation in comparison to the rest of the group. Therefore, in order to overcome this, VO2 max tests could have been carried out on all participants prior to testing to ensure all subjects were of a similar trained level.

Furthermore, only 9 participants were investigated within this study and therefore the results may not represent a wider population. If more participants were incorporated then a larger data set would have been collected which may have led to greater significance readings between variables as there would be more data to draw conclusions from.

In addition, an important factor to note is that due to the difficulties of timetabling and lab availability, not all participants were tested at the same time of day and therefore diurnal factors may have come into effect regarding the reliability of testing. According to Chtourou et al. (2011) diurnal increases in central body temperature may exert a beneficial passive warm-up that may enhance metabolic reactions, increase the extensibility of connective tissue, reduce muscle viscosity and increase conduction velocity of action potentials. It is stated that there have been reports of increases in force production and speed of contraction in the evening compared to the morning. Therefore, this aspect could have an effect upon the results.

Furthermore, participants' diet was not monitored during the study and according to Jurva et al. (2006) dietary supplements can have an effect upon endothelial function and therefore can lead to having an effect upon the present study's results. This could have been overcome by simply asking participants to keep a food diary, therefore monitoring what they ate prior to testing.

Finally, other limitations could extend to the measures that were not incorporated within the present study. Mean arterial pressure and β-stiffness index are variables that are widely used within the literature (Miyachi et al., 2004; DeVan et al., 2005; Collier et al., 2010) that could have been included. According to DeVan

et al. (2005) mean arterial pressure is used to display the pressure-compliance relation and β-stiffness provides an index of arterial compliance adjusted for distending pressure. If these measures were adopted the findings of the present study may have differed and may have displayed a response that relates more closely to the current literature.

5.4 Practical Implications

The adoption of regular RE within lifestyle can promote many favourable benefits to individuals of all ages such as prevention of disease, promoting weight loss, improving muscular strength and enhancing bone health (Collier et al., 2010; Kawano et al., 2010; Phillips et al., 2011). As a result, RE should therefore be considered as part of a daily exercise training programme (Seals et al., 2008; Collier et al., 2010; Kawano et al., 2010). Furthermore, it has been outlined from the present study that RE offers greater benefits in increases in blood flow to active muscles (BMEV, $P<0.05$) and can therefore be used alongside aerobic training by clinical end users (Collier et al., 2010).

5.5 Further Research

The present study only investigated the responses observed in males. Therefore possible expansions of the research can extend to the investigation of the acute vascular responses to a single bout of handgrip RE within females. This combined with the responses observed in males will cover a wider population and may give rise to a greater understanding of the effects acute RE has upon human vasculature.

In addition, to further investigate what acute vascular responses a single bout of handgrip RE causes, future research should look to increase the percentage of 1RM placed upon the RE. As only 10% of 1RM was used within the present study, perhaps a greater percentage may cause a greater physiological stress that is adequate enough to bring about a greater significant response, correlating better with the current literature.

Lastly, what participants ate prior to testing was not monitored in the present study. An investigation into the affects pre exercise meals have upon vascular endothelial function may provide further insight into the endothelium's functional properties.

CHAPTER VI

CONCLUSION

To conclude, a single bout of handgrip RE carried out for 5 mins at 10% of 1RM is not a physically demanding enough exercise to cause a significant vasodilatory response or cause an increase in arterial stiffness. Therefore, the results did not relate to the study's initial hypothesis. Although the RE did cause a significant response in the variables BMEV and BMSR ($P<0.05$), this can simply be attributed to the acute effects caused by exercise. The RE did not cause enough of an increase in the latter responses to cause a significant response in the other variables ($P>0.05$; Table 1). This explains why no vasodilatory response or arterial stiffening was observed. In addition, the study's BP and HR data support this notion as they too did not report a significant response between baseline and post handgrip RE intervention ($P>0.05$; Table 1). This highlights the fact that the RE did not place a great deal of stress upon the participants and was therefore not physically demanding enough to cause a vasodilatory or arterial stiffening response.

However, other possible explanations could account for the lack of response observed within the data. It can be seen from Figures 8 & 9 that participant 3's BMEV and BMSR response differed to the rest of the group. Also when analysing the data participant 8's artery possessed characteristics of chronic adaptation as it was much larger in size in comparison to the other participants. Although both these participants did not engage in weight training, they were both part of sports teams that competed on a regular basis. Therefore, in comparison to the rest of the group, both these participants carry out higher levels of physical activity which has caused adaptation within their arteries. These adaptations may then have had an effect upon how their arteries responded to the RE. Therefore, this aspect could have affected the group means and could have attributed to the lack of significance observed from the other variables. Although, the latter suggestion may only account for a small part of the results as it was only 2 participants out of the entire group ($n=9$). Therefore their results should not have had a great deal of an effect upon the group mean, suggesting that the latter explanation of the RE not being physically demanding enough is more plausible.

Furthermore, in order to more accurately investigate the acute effects of RE upon human vasculature, future research needs to either increase the percentage of 1RM used or incorporate a larger RE that recruits a greater amount of muscle mass. As both suggestions relate to placing greater physiological stress upon the participants, a greater response is more likely to be observed which may have correlated more closely with the current literature and the present study's initial hypothesis.

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APPENDICES

APPENDIX A

AMERICAN COLLEGE OF SPORTS MEDICINE (ACSM) GUIDELINES FOR RECOMMENDATIONS OF QUANTITY AND QUALITY OF EXERCIS

American College of Sports Medicine (ACSM) Guidelines for Recommendations of Quantity and Quality of Exercise

Cardiorespiratory Exercise

- Adults should get at least 150 mins of moderate-intensity exercise per week.
- Exercise recommendations can be met through 30-60 mins of moderate-intensity exercise (5 days per week) or 20-60 mins of vigorous-intensity exercise (3 days per week).
- One continuous session and multiple shorter sessions (of at least 10 mins) are both acceptable to accumulate desired amount of daily exercise.
- Gradual progression of exercise time, frequency and intensity is recommended for best adherence and least injury risk.
- People unable to meet these minimums can still benefit from some activity.

APPENDIX B

PAR-Q QUESTIONNAIRE

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PAR-Q Questionnaire

Please circle either Yes or No to each question.

1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?

   Yes / No

2. Do you feel pain in your chest when you do physical activity?

   Yes / No

3. In the past month, have you had chest pain when you were not doing physical activity?

   Yes / No

4. Do you lose your balance because of dizziness or do you ever lose consciousness?

   Yes / No

5. Do you have a bone or joint problem (for example, back, knee or hip) that could be made worse by a change in your physical activity?

   Yes / No

6. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?

   Yes / No

7. Do you know of any other reason why you should not do physical activity?

   Yes / No

Name: **Signature**: **Date**:

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APPENDIX C

PARTICIPANT INFORMATION SHEET

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Participant Information Sheet

Project Title: Acute Vascular Responses Following a Single Bout of Handgrip Resistance Exercise.

This document provides a run through of:

1. The background and aim of the research
2. My role as the researcher
3. Your role as a participant
4. Benefits of taking part
5. How data will be collected
6. How the data / research will be used

The purpose of this document is to assist you in making an *informed* decision about whether you wish to be included in the project, and to promote transparency in the research process.

1) Background and aims of the research:

An extensive amount of research has been conducted on the vascular responses following aerobic exercise (Green et al., 2012; Thijssen et al., 2012). However, there is limited literature concerned with the effects of other forms upon the vasculature such as resistance exercise (Miyachi et al., 2004). Resistance exercise has been recommended for the attenuation of osteoporosis, sarcopenia and possesses beneficial effects to the muscular and cardiovascular system (Miyachi et al., 2004). However, in contrast it has been stated that acute and chronic resistance exercise leads to arterial distensibility and arterial stiffness (Collier et al., 2010), and therefore possesses the characteristics of cardiovascular disease. This form of exercise is becoming increasingly popular amongst the population, although health professionals are undecided over whether the effects it possesses upon the vasculature are positive or negative. It is evident that there is a lack of research available in the area, however there is even fewer available on the effects of lower forms of resistance exercise such as handgrip. This is an important issue as it has been highlighted that its use is beneficial as Green et al., (2010) states that smaller forms of resistance exercise are important as they can provide insight into localized effects of exercise that are less dependent upon central regulatory or neural changes. Therefore, due to the conflicting theories and in order to evaluate these contrasting hypotheses, by conducting this study it will address the gap in the literature concerning a lack of research available on the effects of lower forms of resistance exercise, and will add to the body of knowledge concerning the vascular effects experienced from engaging in resistance activity. The aim of this study is to determine how the vasculature responds acutely to a single bout of handgrip resistance exercise.

2.) My role as the researcher:

The study involves me (Liam Capener-Jones) the researcher, and my supervisor (Dr Joseph Esformes) collecting data such as:

- Height
- Weight
- Heart Rate
- Blood pressure
- Vascular Measures (base mean diameter, response mean diameter, delta diameter, delta %, base mean envelope velocity, response mean envelope velocity, base mean shear rate and response mean shear rate)

3.) Your role as a participant:

Two visits to the laboratory are required. The first visit will be a familiarisation session lasting 20 mins. Here you will be given the chance to have a go with the equipment to allow you to develop the correct exercise technique, to allow you to become comfortable with the measures taken, the occlusion cuff, and generally to gain an understanding of how the procedure is going to be carried out and what is expected of you.

Visit 2 will be the testing day. Here measures such as height, weight, heart rate, blood pressure and vascular measures of artery dilation, change in blood flow, velocity of blood flow and shear rate will be collected. A one rep max test using the handgrip dynamometer will be required in order for 10% of the 1RM to be calculated (required for resistance exercise within this study). You will be given 3 attempts at a 1 RM test with 30 secs rest between each attempt. An occlusion cuff will be fitted and inflated to 220 mmHg for 5 mins. You will then be required to carry out a resistance exercise using the modified handgrip dynamometer, containing 10% of the 1RM in kg, which will last for 5 mins. Following this the occlusion cuff will be inflated once again for 5 mins and then post exercise measures will be collected.

4.) Benefits of taking part:

The information we obtain from this study will allow better insight into the acute vascular responses following a single bout of handgrip resistance exercise. We will be happy to share this information to any of the participants of this study. On request, we can also provide you with your own personal results, and discuss this with you in relation to your own sporting performance.

5.) How data will be collected:

A stadiometer and digital weighing scales will be needed for profiling and to work out BMI. An ECG will be used to collect hear rate, and blood pressure will be recorded through the use of a finometer. An ultrasound machine will be used to measure vascular data as well as variables such as artery dilation, change in blood flow, velocity of blood flow and shear stress. A pressure cuff will be used and inflated to 220 mmHg for 5 mins to cause an occlusion, and a modified handgrip dynamometer (containing 10% of subjects 1RM in kg) will act as the resistance exercise. A metronome and attached speaker will be used to control a contraction-relaxation ratio of 1 sec contraction and 1 sec

relaxation and a stop watch will be used to time cuff inflation, duration of FMD measures and resistance exercise time periods.

6.) How the data / research will be used:

In agreeing to become a *voluntary* participant, you will be allowing me to use your data and include them within a larger data set that includes the data of other participants. Your personal data will be anonymous and will not be reported alone, but within the total sample of participants.

Your rights

Your right as a *voluntary* participant is that you are free to enter or withdraw from the study at any time. This simply means that you are in full control of the part you play in informing the research, and what *anonymous* information is used in its final reporting.

Protection to privacy

Concerted efforts will be made to hide your identity in any written transcripts, notes, and associated documentation that inform the research and its findings. Furthermore, any personal information about you will remain *confidential* according to the guidelines of the Data Protection Act (1998).

Contact

If you require any further details, or have any outstanding queries, feel free to contact me on the details printed below.

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E-mail: st10001374@outlook.uwic.ac.uk

APPENDIX D

INFORMED CONSENT FORM

Liam Capener-Jones
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CARDIFF METROPLOLITAN
INFORMED CONSENT FORM

CSS Reference No:
Title of Project: Acute Vascular Responses Following a Single Bout of Handgrip Resistance Exercise.
Name of Researcher: Liam Capener-Jones

Participant to complete this section: Please initial each box.

1. I confirm that I have read and understand the information sheet dated ……. for this evaluation study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily. ☐
2. I understand that my participation is voluntary and that it is possible to stop taking part at any time, without giving a reason. ☐
3. I also understand that if this happens, our relationships with the Cardiff Metropolitan University, or our legal rights will not be affected. ☐
4. I understand that information from the study may be used for reporting purposes, but I will not be identified. ☐
5. I agree to take part in this study on: Acute Vascular Responses Following a Single Bout of Handgrip Resistance Exercise. ☐

Name of Participant:

Signature of Participant:

Date:

Name of Person Taking Consent:

Signature of Person Taking Consent:

Date:

APPENDIX E

PARTICIPANT PROFILE DATA

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Table 2. Participant Profile Data.

Participants	**Age**	**Height**	**Weight**	**BMI (weight (kg)/height (m) = answer/height (m) = BMI)**	**1 Rep Max**				
					Attempt 1	**Attempt 2**	**Attempt 3**	**Mean 1RM**	**10% of 1RM**
Participant 1	20 yrs	1.70m	69.7kg	24.1	32kg	32.5kg	31kg	31.8kg	3kg
Participant 2	19 yrs	1.74m	65.9kg	21.8	41kg	46kg	43kg	43.3kg	4kg
Participant 3	20 yrs	1.82m	87.3kg	26.4	55kg	56kg	55kg	55.3kg	5.5kg
Participant 4	20 yrs	1.77m	82.8kg	26.4	46kg	45kg	49kg	46.6kg	4.5kg
Participant 5	21 yrs	1.83m	74.3kg	22.2	34kg	46kg	44.5kg	41.5kg	4kg
Participant 6	20 yrs	1.78m	94.4kg	29.8	44kg	46kg	51kg	47kg	4.5kg
Participant 7	21 yrs	1.73m	77.1kg	25.8	47kg	49kg	47kg	48kg	4.5kg
Participant 8	21 yrs	1.76m	75.6kg	24.4	50kg	48kg	56kg	51.3kg	5kg
Participant 9	23 yrs	1.91m	86.4kg	23.7	59kg	56kg	60kg	58.3kg	5.5kg

APPENDIX F

HEART RATE DURING INTERVENTION

Table 3. Heart Rate During Intervention.

Participants	Baseline Mean Heart Rate (BPM)	During Intervention Mean Heart Rate (BPM)	Post Mean Heart Rate (BPM)
Participant 1	64	78	67
Participant 2	64	75	63
Participant 3	57	71	63
Participant 4	60	69	62
Participant 5	73	76	59
Participant 6	54	87	46
Participant 7	66	83	69
Participant 8	76	76	74
Participant 9	54	64	57