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< The impact of constant load *vs* submaximal interval exercise on post exercise hypotension, using arm crank ergometry >

(Dissertation submitted under the discipline of Physiology & Health)

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TITLE PAGE

DECLARATION

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LIST OF ABBREVIATIONS AND ACRONYMS

~ – About

≈ – Approximately equal to

% – Percentage

> – Greater than

≥ – Greater or equal to

< – Less than

≤ – Less or equal to

± – Plus-minus

α – alpha

Δ – Change in

5-HT – Serotonin

5-HTP – 5- Hydroxytryptophan - *serotonin precursor*

A – Adrenaline

ABPM – Ambulatory Blood Pressure Monitoring

ACh – Acetylcholine

ADH – Anti Diuretic Hormone

ANG II – Angiotensin II

ANOVA – Analysis of variance

ANP – Atrial Natriuretic Peptide

ANS – Autonomic Nervous System

ARM – Arm-crank ergometry

ATP – Adenosine Triphosphate

BMI – Body Mass Index ($kg.m^{-2}$) – *mass/height*^{2}

BP – Blood Pressure (mmHg)

$b \cdot min^{-1}$ – beats per minute

CMT – Continuous Moderate-Intensity Training

CO – Cardiac Output – *(stroke volume x heart rate)*

CO_2 – Carbon dioxide

CONT – Continuous Protocol – *maintaining exercise at the same intensity*

CVD – Cardiovascular disease(s)

CVS – Cardiovascular System

DBP – Diastolic Blood Pressure – *recorded as the 4th Korotkoff sound*

EDV – End Diastolic Volume

EF – Ejection Fraction – *(stroke volume/end diastolic volume) x 100%*

ESV – End Systolic Volume

ET-1 – Endothelial-1 – *vasoconstrictor peptide produced by vascular endothelium*

FH+ – Normotensive offspring of hypertensive parents - *high familial risk of HTN*

GXT – Graded Exercise Test

H_1/H_2 – Histamine Receptors

HIIT – High-Intensity Interval Training

HR – Heart Rate

HR_{max} – Maximum Heart Rate

HRR – Heart Rate Reserve

HTN – Hypertension

INT – Interval Protocol

K^+ – Potassium

LEG – Cycle Ergometry

MAP – Mean Arterial Pressure (mmHg) – [(SBP + 2.DBP) ÷ 3]

mmHg – millimeters of mercury

NA – Noradrenaline

Na^+ - Sodium

NO – Nitric Oxide

NTS – Nucleus Tractus Solitarii

O_2 – Oxygen

PAR-Q – Physical Activity Readiness Questionnaire

PEH – Post Exercise Hypotension

PO – Power Output

pPCA - Para-Chlorophenylalanine - *specific serotonin depletor*

PNS – Parasympathetic Nervous System

PSH – Post Stimulatory Hypotension

RAA – Renin-Aldosterone-Angiotensin (system)

RPE – Rating of Perceived Exertion

$rev \cdot min^{-1}$ – revolutions per minute

$reps \cdot min^{-1}$ – repetitions per minute

SBP – Systolic Blood Pressure (mmHg) - *recorded as the 1st Korotkoff sound*

SD – Standard Deviation

SE – Standard Error

SHR – Spontaneously Hypertensive Rats

SM – Smooth Muscle

SNS – Sympathetic Nervous System

SNA – Sympathetic Nerve Activity

SSPS – Statistical Package for the Social Sciences

SSRI – Selective Serotonin Re-uptake Inhibitor – *such as fluoxetine*

SV – Stroke Volume – *(end diastolic volume – end systolic volume)*

TPR – Total Peripheral Resistance

VO_2 – Oxygen Consumption

VO_{2peak} – Peak Oxygen Consumption

VO_{2max} – Maximal Oxygen Consumption

V_T – Ventilatory Threshold

W_{peak} – Peak Aerobic Power (W)

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Abstract

Aims: Post exercise hypotension is the transient drop in systolic and/or diastolic blood pressure that follows an acute bout of exercise.

represents a transient drop in SBP and/or DBP below resting baseline values in the minutes to hours after an acute bout of exercise

Eight normotensive recreationaly active particpiants eged

CHAPTER 1

INTRODUCTION

Essential arterial hypertension (HTN) is a chronic disease of elevated blood pressure (BP) and is considered a major risk factor for cardiovascular disease, morbidity and mortality (Whelton, 1994). HTN is defined as systolic blood pressure (SBP) ≥ 140 mmHg and diastolic blood pressure (DBP) ≥ 90 mmHg; whilst pre-hypertension (pre-HTN) refers to SBP of 120-139 and DBP of 80-89 mmHg (European Society of Hypertension, 2003). In terms of managing this prevalent disease, aerobic exercise is a key non-pharmacological, non-nutritional lifestyle intervention prescribed to enhance cardiorespiratory fitness (Haskwell *et al.*, 2007), and promote improved overall health (Chase *et al.*, 2009). Amongst a normotensive population, regular physical activity has been proven to be preventative against the development of high BP (Hayashi *et al.*, 1999). Whilst in borderline and hypertensive individuals, such exercise training minimises excessive elevations in BP (Ciolac *et al.*, 2009) and improves a number of factors in the pathophysiology of HTN (Cornelissen & Fagard, 2005). Two popular mechanisms for this pattern of chronically lowered BP following exercise training are 1) a sustained decrease in heart rate (HR) and 2) a lower concentration of circulating catecholamines (Tipton, 1984); both of these acute responses have been associated with decreased sympathetic nerve activation (SNA).

Post exercise hypotension (PEH) represents a transient drop in SBP and/or DBP below resting baseline values in the minutes to hours after an acute bout of exercise (Kenney & Seals, 1993; Taylor *et al.*, 2010). Hill (1898) was the first to document this phenomenon. However, it was not put into a clinical perspective until nearly a century later (Halliwill *et al.,* 2013), following an anecdotal study by Fitzgerald (1981) into the blood pressure lowering effects of 25-min jogging (70% HR_{max}) on his own labile HTN. The prospect that recurrent transient hypotensive reponses induce sustained BP decline overtime (Lui *et al.*, 2012) gives PEH potential clinical significance in terms of BP management. In the last few decades a greater understanding of the physiology of PEH has emerged; thought to result from a continuous post exercise decline in systemic

vascular resistance (SVR) that cannot be totally offset by an increase in cardiac output (CO) (Halliwill, 2001). At present PEH has been extensively reported in response to both aerobic and resistance exercise; in hypertensive and normotensive individuals of all ages and gender (Rueckert *et al*., 1966; Raglin *et al.*, 1993).

Most research in this field has employed submaximal, endurance protocols using leg ergometry, treadmill walking or running. Few studies have implemented upper body modes of aerobic exercise, such as arm crank ergometry (MacDonald *et al.,* 2000a; Almeida *et al.*, 2010). Consequently, our understanding of the acute blood pressure response to exercise that requires relatively small volumes of skeletal muscle is limited. Arm crank ergometry is valuable for developing aerobic fitness in individuals that struggle with lower body exercise, such as paraplegics, or those with spinal cord injuries or diabetic feet (Almeida *et al.,* 2010). Although it is currently recommended as an alternative aerobic exercise mode in such populations, relatively little is known of its acute and chronic effect on BP.

In a similar manner, research that compares interval (INT) and continuous (CON) exercise protocols that vary in work pattern, but are matched for total work done, is lacking. Recently INT exercise has become increasingly popular for improving cardiorespiratory fitness and other health-related outcomes across a range of different populations (Whyte *et al.,* 2010; Hood *et al.,* 2011). It has been suggested INT training that typically incorporates intermittent, short, high intensity exercise bouts, is more effective than traditional CON exercise in terms of reducing ambulatory BP (Ciolac *et al.,* 2010). Elsewhere it appears both protocols are equally effective (Guimaraes *et al*., 2010). It remains unclear whether the acute blood pressure response (PEH) is greater with INT patterns of exercise (Cunha *et al.,* 2006; Ciolac *et al.,* 2009).

Furthermore, most studies of PEH have recruited clinical, hypertensive human participants. Although PEH has been well documented amongst this diseased population, its occurrence in individuals with normal resting BP (<140/90 mmHg) (European Society of Hypertension, 2003) has been less consistent, and its reported magnitude and duration smaller (MacDonald *et al.*, 1999a). To the author's knowledge, the proposed study is unique in comparing the PEH responses of a normotensive population to INT and CON exercise, using arm crank ergometry.

With the above in mind, the main objectives of this study were to establish whether young, healthy and reasonably fit normotensive men 1) experienced a period of sustained PEH following submaximal arm crank ergometry and 2) whether the magnitude and duration of PEH varied following a period of either CON or INT exercise of equivalent duration, intensity and volume of work performed.

CHAPTER 2

REVIEW OF LITERATURE

2.1 Physiological Mechanisms of Post Exercise Hypotension

Mean arterial pressure (MAP) is the product of total peripheral resistance (TPR) and cardiac output (CO); the former determined by vasoactivity and blood viscosity, the later by stroke volume (SV) and heart rate (HR). Most studies have stated that decreased MAP in the immediate post exercise period is mediated predominantly by reduced TPR rather than by reduced CO (Cléroux *et al.*, 1992; Pescatello *et al.*, 2004a); although there are exceptions (Hagberg *et al.*, 1987; Brandão Randon *et al.,* 2002). CO usually remains elevated from a pre-exercise value for the duration of PEH (Halliwell, 2001), but gradually declines secondary to reductions in venous return, end diastolic volume (EDV) and SV. Accompanying these changes there is a progressive decline in HR, whilst BP often begins to increase towards the end of recovery (Halliwell *et al.*, 2013). According to Poiseuille's Law, the resistance of a blood vessel is directly proportional to the 4^{th} power of its radius, but inversely proportional to its length and blood viscosity (Davis *et al.*, 2001). As exercise does not affect the latter variables, reduced TPR and hence MAP can be attributed to increased vessel radius (Pescatello *et al.*, 2004b). Accordingly, a small post exercise increase in vessel diameter will significantly reduce vascular resistance and hence MAP.

Following acute exercise, a number of physiological mechanisms reduce vascular resistance and sustain postexercise vasodilation of the previously working muscle (Halliwill *et al.,* 2013). It is impossible to isolate a specific physiological mechanism for PEH from the literature concerning systemic and regional haemodynamics post exercise (Pescatello *et al.*, 2004b). Instead it is currently believed PEH arises from the interaction of several factors, in varying time scales and sequences, which can be grouped broadly as follows. 1) There is a centrally mediated decrease in sympathetic nervous activity. 2) A decrease in signal transduction and vascular responsiveness to such sympathetic stimulation, results in reduced vasoconstriction. 3) An increase in local vasodilatory mechanisms and substances that serves to further enlarge the lumen diameter and increases blood flow in skeletal muscle vasculature.

2.1.1 Sympathetic Inhibition

2.1.1.1 Central Sympathoinhibition

Efferent sympathetic nerve activity (SNA) promotes vasoconstriction and increased TPR. It is characteristically elevated in Hypertension (HTN) (Abboud, 1982); whilst is decreased in both normotensive and hypertensive individuals postexercise (Floras *et al.,* 1989). The atrial baroreflex mechanism is key in the acute control of BP, and reciprocal control of HR and sympathetic nerve activity (Ichinose *et al.,* 2008). During the recovery from acute exercise, baroreceptors are reset to a lower BP than pre-exercise in humans (Halliwell *et al.,* 2006) and rats (Kajekar *et al.,* 2002). This is accompanied by reduced efferent SNA; evident as reduced sympathetic skeletal muscle activity in humans (Halliwell *et al.,* 2006) and reduced lumbar/renal nerve activity in rats (Miki *et al.,* 2003).

Building on the work of Potts (2006), Chen & Bonham (2010) confirmed recently that skeletal muscle afferents, and their input into the nucleus tractus solitarii (NTS), are key in the mechanism of postexercise baroreflex resetting and sympathoinhibition. There is also evidence to suggest a role of cardiac afferent sympathetic activity in PEH from studies by Collins & DiCarlo (1993) and Chandler & DiCarlo (1997); showing the hypotensive response is significantly attenuated by cardiac muscle afferent blockade, and prevented by sinoaortic denervation respectively. Furthermore the mechanism for baroreceptor resetting by cardiac afferents could incorporate stimulation of NTS opioid receptors.

Significant evidence from rat studies exists to support an exercise-mediated activation of central opioid and serotonergic systems, which in tern reduces sympathetic outflow and MAP. Hoffmann *et al.* (1990) observed PEH when β-endorphins bound opioid receptors; whilst Yao *et al.* (1982) found post stimulatory hypotension (PSH) was augmented by the selective serotonin reuptake inhibitor (SSRI) zimelidine and abolished by pCPA (serotonin depletor). However human studies have yielded contradictory findings that question the mechanistic importance of these systems (Hara & Floras, 1992; MacDonald *et al.,* 2002a). This may imply species variation in the mechanism of PEH/PSH.

2.1.1.2 Blunted Transduction

In addition to central sympathoinhibition via baroreflex resetting, the peripheral transduction of efferent sympathetic activity into vasoconstriction is also blunted. Halliwill *et al.,* (2003) found responsiveness to α1- or α2-adrenergic stimulation was unchanged by acute exercise. This implies reduced vascular sensitivity was due instead to changes (reduced release or increased reuptake) in pre-synaptic noradrenaline (NA).

2.1.2 Vascular Adaptations

The post-exercise vasodilation that occurs in previously active skeletal muscle generally follows the pattern of an initial hyperaemia that lasts seconds or minutes (≤ 20-mins), followed by a sustained vasodilation that persists typically for several hours (≥ 2-hr) (Laughlin *et al.,* 2012). A number of tissue-level metabolic factors and hormones are likely to influence this sustained vasodilation and hence mediate the associated PEH (Lockwood *et al.,* 2005b). In addition to those discussed below; adenosine (Duffy *et al.,* 1999) and prostaglandins (Boushel *et al.,* 2002) have both been implicated, although Halliwill *et al.* (2000) and Lockwood *et al.* (2005a) both observed PEH despite inhibition of prostaglandin synthesis.

2.1.2.1 Nitric Oxide and Endothelial-1

Nitric Oxide (NO) and Endothelial-1 (ET-1) are endothelium-derived growth factors thought to be produced by counter-inhibitory pathways. NO is an important vasodilator and relaxant of smooth muscle (SM) (Powers & Howley, 2009), whilst ET-1 promotes vasoconstriction and SM proliferation. Following 3-months of leg ergometry training (30-min at 80%V_T, 5-days/wk), Maeda *et al.,* (2003) found that the pre-exercise ET-1 and SBP/DBP of normotensive women were significantly reduced, by 2.90 to 2.22 $pg.ml^{-1}$ and 127/79 to 112/65 mmHg respectively. They inferred the ET-1 suppression was associated a reciprocal increase in NO (not measured), which together enhanced vasodilation, reduced resistance and hence lowered BP. Whilst this is a worthy explanation, it must be noted the exact physiological interactions of such endothelium-derived factors remain unclear.

From early animal work, Patil *et al.,* (1993) stated that NO mediated sustained vasodilation postexercise, by reducing α-adrenergic sensitivity. Maeda *et al.,* (2001) noted that training improved NO production; inferring exercise leads to improved endothelial-dependent vasodilation. This may be particularly beneficial to hypertensive individuals who classically have impaired endothelial function. However, later human work of Halliwill and colleagues (2000; 2003) disputed the supposed BP lowering benefits of NO, claiming it did not significantly contribute to vascular tone.

There is disagreement amongst the literature as to the importance and influence of NO and other endothelial-derived factors (including Adenosine, K^+, ANP etc.) in mediating PEH. In response to muscle contraction and augmented muscle blood flow, the release of such vasodilatory factors certainly seems to contribute to BP reduction at a tissue level via promotion of vasodilation. But, amongst other reasons, as PEH usually persists for a considerable length of time after the concentrations of such factors have normalized; it unlikely they are responsible for PEH *per se*.

2.1.2.2 Histamine H_1 and H_2 Receptors

More recently, histamine receptor activation has emerged as a key mechanism for sustained post exercise vasodilation and PEH. When H_1 and H_2 receptors were simultaneously antagonized prior to 60-min moderate intensity leg ergometry, recovery vasodilation was reduced by ~80% and hypotension by ~65% (Lockwood *et al.,* 2005b; McCord & Halliwill, 2006; McCord *et al.,* 2006). For resistance exercise of equivalent intensity (60-min unilateral knee-extensions), such H_1 and H_2 antagonism abolished vasodilation (100%) and PEH (Barrett-O'Keefe *et al.,* 2012). The histamine-independent vasodilation (~20%) that remained following the aerobic exercise was likely to be from baroreflex resetting and sympathoinhibition; triggered by the comparatively greater volume of exercising muscle. Whilst these findings highlight the vasodilatory importance of histamine receptor activation, establishing the cascade of events that leads up to this increase in intramuscular histamine warrants further research.

2.1.3 Structural Adaptations

Musculoskeletal (muscle tissue) and cardiovascular (surrounding vasculature) structures adapt to training. There is widespread evidence for training-induced vascular remodeling and angiogenesis in muscle (Laughlin *et al.,* 1996), and for the development of conduit arteries with greater lumen diameter (Ishikawa-Takata, 2003) and increased arterial compliance (Cameron, 1994). Remodeling of existing arteries allows for increased length, cross sectional area and diameter (Laughlin *et al.,* 1996), whilst new vessel growth (angiogenesis) allows for capillary bed expansion (Halliwell *et al.,* 2013). Such changes develop after repeated exposure to acute stressors (regular exercise bouts) and are likely to contribute to long-term decreases in TPR. The lumen diameter of conduit arteries was found to be greater in endurance-trained athletes than control (Huonker *et al.,* 1996), whilst the wall thickness of peripheral arteries was reduced (Moreau *et al.,* 2002). Such adaptations reduce vascular tone and encourage lower MAP longer-term (Dinenno *et al.,* 2001).

2.2 Post Exercise Hypotension in Normotensives and Hypertensives

2.2.1 Post Exercise Hypotension in Normotensive Individuals

Whilst PEH is well documented in those with borderline and diagnosed HTN (Cléroux *et al.,* 1992; Hara & Floras, 1994; Brownley *et al.,* 1996), its occurrence and extent in individuals with normal resting BP (< 120/80 mmHg) is less consistent (MacDonald 2002b; Cornelissen & Fagard, 2004). Studies into the MAP response of normotensive subjects to single bouts of dynamic aerobic exercise have yielded contradictory findings (Floras & Wesche, 1992). When looking into hypotension post submaximal leg ergometry, Pescatello and colleagues (1991) found that, unlike in the mildly hypertensive group, MAP did not appear to drop significantly prior to postexercise, or between exercise and control (non-exercise) days in the normotensive controls. This is likely to be because individuals with lower pre-exercise MAP experience a reduced absolute drop during recovery, which is less likely to reach statistical significance or produce consistent hypotensive trend (Tipton, 1983). In other words the literature implies that lack of consistency across studies occurs because PEH is of a lessor magnitude in normotensives and consequently harder to detect (Pescatello *et al.,* 1991; Pescatello *et al.,* 2004a). A thorough review study

(Kenney & Seals, 1993) supports this, quantifying the average reductions in SBP/DBP to be 9/4 mmHg in normotensives, compared with 19/8 mmHg in hypertensives.

Amongst studies that have reported PEH in normotensives (Somers *et al.,* 1991; Franklin *et al.,* 1993; Halliwill *et al.,* 2000; Raine *et al.,* 2001) there is indecision whether higher exercise intensities elicit greater hypotension (Piepoli *et al.,* 1994; Forjaz *et al.,* 2004) or not (Forjaz *et al.,* 1998; MacDonald *et al.,* 1999b). As the BP response to stress can be exaggerated by measurement of concomitant haemodynamic variables, or blunted by greater prior exercise exposure; such factors could account for greater and lessor PEH reported. Of the aforementioned studies, Forjaz *et al.* (2004) had the largest sample size (n=23). Although their findings were limited to young (age 24±1), healthy, normotensive (MAP 84±2) subjects, there was a clear dose-response relationship between exercise intensity (30/50/75% VO_{2peak}) and magnitude of decline in SBP/DBP/MAP in the 90-min post intervention.

2.2.2 Post Exercise Hypotension in Hypertensive Individuals

Most insights into the mechanisms of PEH are based on normotensive humans or spontaneously hypertensive rats (SHR); hence caution is needed in extrapolating these results to hypotensive populations. That said, it is generally thought any differences in PEH between normo- and hypertensives are quantitative not qualitative in nature. It has become clear that whilst the exact mechanism for PEH is unknown, there is a compensatory relationship between TPR and CO, in so far as at least one will be reduced post exercise to ensure a decline in MAP. In *elderly* hypertensive individuals, Hagberg *et al.* (1987) suggested the mechanism for PEH is more reliant on reduced CO, as the additional age-related arterial stiffness causes decreased vasodilatory capacity and increase TPR.

A review article by Anunciac & Polito (2011) assimilated the findings of 32 studies of post exercise (27 aerobic, 5 resistance) hypotension in hypertensives; generally greater PEH was observed following aerobic than resistance modes. Consensus regarding whether higher exercise intensities induced greater (Hagberg *et al.,* 1987; Quinn, 2000) or equal (Blanchard *et al.,* 2006; Guidry *et al.,* 2006; Syme *et al.,* 2006, Pescatello *et al.*, 2007) PEH responses than milder intensities (75% vs. 50% VO_{2peak}) was inconsistent. Studies by Ciolac *et al.,* (2009) and Cunha *et al.,* (2006), both suggest continuous (CON)

patterns are more beneficial than interval (INT) exercise; although there is growing evidence on the contrary as discussed in a later section (*2.5. Type of Exercise*). The current recommendations (Pescatello *et al.,* 2004a) of continuous, low to moderate (40% - 60% VO_{2peak}) intensity exercise, reduces risk of unsafe BP elevations during exercise. Indeed most studies have prescribed 30 to 45-mins of 50-60% VO_{2peak} although it must be emphasized there is still significant conflict surrounding what combinations of modality, intensity, pattern and duration are most effective.

Greater sensitivity of skeletal muscle afferents along with exaggerated central baroreflex resetting could trigger the exaggerated exercise pressor response characteristic of HTN; as well as account for the heightened drop in BP often observed after exercise. Hecksteden *et al.* (2012) and Lui *et al.* (2012) have postulated more recently (using pre-hypertensive individuals) that greater PEH reponses can be used as a positive predictor of greater long-term BP decrements with aerobic training. Clearly, an attractive implication of PEH in hypertensives is that it might translate directly into the chronic training-mediated reductions in BP (Thompson *et al.,* 2001); although it remains unclear which acute mechanisms would prompt this.

2.3 Exercise Modality

2.3.1 Arm-Crank Ergometry

Dempster & Graghram (1967) outline that the upper limbs account for 7.6% of total body mass, compared to 32% for the lower limbs. It is reasonable therefore to hypothesize differences in the characteristics of PEH between upper and lower body exercise modalities, due to obvious differences in the volume of exercising muscle mass and hence absolute metabolic rate. In addition, the absolute skeletal muscle production of vasodilatory substances such as adenosine/K^+ increases with muscle mass. To this extent, an increased hypotensive response to leg ergometry might suggest PEH is mediated peripherally (MacDonald *et al.,* 2000a).

To the author's knowledge, only MacDonald *et al.* (2000a) and Almeida *et al.* (2010) have assessed PEH following arm-crank ergometry (ARM), both of whom reported similar BP reductions to post cycle ergometry (LEG). MacDonald *et al.* (2000a) implemented 30-min submaximal ARM (65% VO_{2peak}) and LEG (70% VO_{2peak}) protocols in borderline

hypertensives (n=9). Whilst Almeida *et al.* (2010) employed incremental tests starting at 6/30W (ARM/LEG) at 60 rev•min^{-1} and increasing by 6/30W every 3-min, using normotensive men (n=11). Interestingly in the latter study SBP and MAP were reduced by both exercise modalities, but LEG failed to significantly reduce DBP, in contrast with the significant PEH of DBP with ARM (mean reduction of 9 ± 1 mmHg, $p<0.05$). This reduction in DBP represents higher vasodilation of upper limb musculature, which can potentially be explained by closer proximity of the active skeletal muscle to the heart; by different predominant fibre typology of arms vs. legs; and/or by greater stress induced by unconventional exercise using a smaller and less trained muscle mass (Volianitis *et al.,* 2004; Lyons *et al.,* 2007). Data from this group (unpublished) also showed immediate (<1-min) post-ARM DBP to be elevated more (+17 mmHg) than post-LEG (+10 mmHg). This suggests an association between greater end-exercise DBP and greater subsequent post-exercise drop in MAP (Almeida *et al.,* 2010).

Although few studies, with relatively small sample sizes, have used arm-crank exercise, their results have encouragingly suggested it is as effective as leg cycling at inducing PEH. Hence, work so far supports the use of arm ergometry to elicit PEH, and potentially aid BP control/HTN prevention in both normo- and hypertensives. Evidence suggests it can be advised as an equally effective alternative mode, in those where lower limb exercise is contraindicated.

Lastly, the finding that PEH was similar following exercise that employed notably different volumes of exercising skeletal muscle lends support to two previously mentioned theories. Firstly, that the hypotensive response is controlled centrally; potentially involving the opioid/serotonergic systems. Secondly, that PEH is mediated by an exercise-induced reduction in vascular sensitivity.

2.4 Exercise Factors that Affect Post Exercise Hypotension

2.4.1 Exercise Intensity

It is generally accepted that PEH is greater following higher (70 – 75% VO_{2peak}) than lower (50% VO_{2peak}) intensity exercise (Quinn, 2000; Kenny *et al.,* 2003; Forjaz *et al.,* 2004). Although conversely Pescatello *et al.* (2004b) and Blanchard *et al.* (2006) state PEH is greater with lower (40% VO_{2peak}) than moderate intensities (60% VO_{2peak}); whilst Pescatello *et al.* (2007) and Cornelissen *et al.* (2010) propose the magnitude of PEH is infact unaffected by exercise intensity (40 – 70% VO_{2peak}). Conflicting results can be attributed to relative variation in what is considered low, moderate or high intensity between study designs.

Eicher *et al.* (2010) described a dose-dependent relationship between exercise intensity and PEH: for each relative 10% increase in VO_{2peak}, SBP/DBP decline increased by 1.5/0.6mmHg. Although clinical translatability is somewhat limited by restrictive inclusion criteria (overweight, white, middle-aged, male, mild HTN), the authors suggest higher intensities of aerobic exercise should be prescribed in those able/willing to tolerate it, in order to maximize the blood pressure lowering benefits of exercise. Syme *et al.* (2006) found that in subjects with a high peak SBP (in response to a GXT), light exercise (40% VO_{2peak}) would elicit PEH, whereas moderate intensity exercise (60% VO_{2peak}) was required in those with lower peak SBP. They suggest this is because the vasoconstrictor effect of the sympathetic system is reduced in the former group due to endothelial dysfunction; hence TPR can be overcome more easily. As implied by Eicher *et al.* (2010), these findings also suggest a more individualized prescription of exercise intensity is required, particularly here to establish the minimum stimulus for PEH in a particular population and avoid overtraining. Finally, Liu *et al.* (2012) have confirmed more recently that the magnitude of PEH is greater following high than moderate intensity (80% and 60% VO_{2peak}) prolonged aerobic exercise (120-min treadmill running), in both young (28 years) and middle aged (52 years) endurance athletes.

The majority of literature has found the intensity of exercise to have significant effect on PEH. In addition, as the CV responses of the body *during* exercise (such as sympathetic activity, NA release and haemodynamic changes) are directly related to intensity, it is likely such *post* exercise responses (that includes PEH), are also influenced

by intensity (Rueckert *et al.,* 1996; Forjaz *et al.,* 2004). Exercise intensity is therefore considered an influential component of training programs aimed at reducing BP.

2.4.2 Exercise Duration

Whilst it is generally assumed longer duration exercise will elicit greater PEH, it is hard to evaluate the influence of duration in isolation from concomitant variables, most notably intensity (MacDonald, 2002b). Both the actual duration as well as the total work done during the exercise bouts varies between across study designs. Bennett *et al.* (1984) showed exercise duration influences PEH by mapping BP decline following 5 consecutive intermittent bouts of exercise (10-min bouts, 3-min intervals). In normotensives, SBP/DBP was significantly reduced after 50-min (108/73 mmHg) vs. 10-min (117/78 mmHg) of exercise. In hypertensives significant PEH occurred after all bouts, the duration of which increased progressively with time (17 mmHg at 10-min; 29 mmHg after 50-min).

Similarly, Mach *et al.* (2005) reported significantly greater PEH after longer (40/80-min) than shorter (10/20-min) duration exercise, in mild hypertensives performing continuous (CON) moderate intensity (80% VO_{2peak}) leg ergometry. Yet conversely, in a similar borderline hypertensive population, MacDonald *et al.* (2000b) found no significant differences in magnitude of PEH between 10-min and 30-min duration of cycling at moderate intensity (70%VO_{2peak}). Although the conclusions of MacDonald *et al.* (2000b) contradict earlier work of Bennett *et al.* (1984), we should be cautious to compare BP data directly given the dissimilarity between study designs. Whilst the magnitude remained unaffected, the authors could report that exercise durations as short as 10-min induced PEH, as well as comment that the duration of this response increased with longer exercise bouts (MacDonald *et al.,* 2000d).

Finally, although exercise intensity often varies between study designs as demonstrated, intensity within each study has typically been kept consistent when assessing the effect of duration. In contrast, Guidry *et al.* (2006) altered both duration (15-min vs. 30-min) and intensity (40% and 60% VO_{2peak}), and concluded the magnitude of PEH depended more on intensity than duration. Evidently we cannot conclude from present literature what duration is optimum for eliciting beneficial hypotensive response. It does however highlight the need to consider duration in parallel with intensity, shown previously to be an influential determinant of PEH.

2.4.3 Exercise Volume

The volume of aerobic exercise, otherwise referred to as the *total energy expended*, *total work done*, or the *dose* (Shephard & Balady, 1999); is a combination of the intensity and duration of exercise. To my knowledge, Jones *et al.* (2007) have conducted the only study into the effect of intensity on PEH that controlled total work done; using 7 normotensive males aged 28 ± 6 (mean ± SD).

A high intensity short duration (HS) (70% VO_{2peak}, 30-min) test was matched for total work done to a moderate intensity long duration test (ML) (40% VO_{2peak}, 50-min), which was intensity-matched to a moderate intensity short duration test (MS) (40% VO_{2peak}, 30-min). The MS and HS tests were evidently matched for duration, whilst PEH responses following all 3 tests were compared to a control (30-min seated rest). Their major finding was lower-intensity longer-duration exercise induced equal BP benefits to more rigorous exercise. They concluded that as PEH did not vary for exercise of equal *dose,* the hypotensive response must be dependant on total work done, rather than intensity or duration in isolation.

It must be noted this study only measured the short term BP response, for 20-min post-exercise, in contrast to more longitudinal approaches (Pescatello *et al.,* 2004b) that have assessed BP up to 24-hrs post exercise, into every day living. In addition, their findings were confined to normotensive participants. That said, recommendations of lower-intensity longer-duration exercise are effective likely to be relevant to hypertensives; firstly as PEH magnitude is reportedly greater in this population anyway (Kenney & Seals, 1993; Thompson *et al.,* 2001), and secondly as this would reduce the risk of adverse events associated with high intensity exercise (Lemaitre *et al.,* 1999).

2.5 Type of Exercise

Continuous (CON) moderate intensity aerobic exercise training has been traditionally advised to manage poor cardiorespiratory fitness (Lawes *et al.,* 2008) and essential HTN (Myers *et al.,* 2002), the strongest and most common predictors of CV mortality respectively. However newer evidence suggests interval (INT) exercise, which typically alternates short (30-sec to 4-min) high-intensity bouts with active recovery or rest (Ciolac *et al.,* 2012), brings about greater aerobic and pathophysiological benefits than CON. The

effectiveness of INT training on performance (Laursen *et al.,* 2002), insulin sensitivity (Hood *et al.,* 2011), fat loss (Whyte *et al.,* 2010) and other health-related outcomes has already been well established. It has been speculated that INT has similar benefits on PEH. That said relatively few studies have assessed PEH following INT exercise in comparison to other types of exercise. The varied results so far can be attributed to differences in the duration and intensity of each high intensity/recovery bout, as well of the intervention as a whole.

2.5.1 Acute Response to Interval versus Continuous Exercise

Rossow *et al.* (2010) compared PEH following 4 x 30-sec 'all out' cyling bouts interspersed by 4.5 mins light active recovery (INT) against 60-min at 60% HRR (CON). The decreases in SBP/DBP/MAP were 5.6/5.1/4.4 mmHg (INT) and 5.1/3.9/3.8 mmHg (CON); showing an acute session of only 2-min exercise could provoke PEH of similar magnitude to 60-min steady state exercise. Chan and Burns (2013) implemented the same Wingate test protocol (INT) in 10 healthy males and saw a BP decrement of 8/6 mmHg (SBP/DBP), slightly greater than the 5.6/5.1 mmHg observed by Rossow *et al.* (2010). This study confirms a significant PEH occurs following INT exercise, compared with a control of no exercise.

Cunha *et al.* (2006) assessed if a single bout of INT exercise (alternating between 2-min at 60% and 1-min at 75% HRR) induced an additional hypotensive benefit to CON (60% HRR) amongst hypertensive subjects (n=11) aged 56.8 ± 2.6 years. They found both submaximal, 45-min, treadmill-based protocols induced similar hypotensive responses in terms of SBP. However the PEH responses of MAP, and DBP following INT were less prolonged, and absent respectively. Conclusions were made that the hypotensive effect of INT was no better than CON (Cunha *et al.,* 2006).

Contrastingly, Ciolac *et al.* (2009) suggest INT (2-min 50%, 1-min 80% HRR for 40-min) does have a greater hypotensive effect than equivalent steady state cyling. Again the participants (n=52) of this study were medication-controlled hypertensives of similar age (< 55 years), and similarly had no washout period prior testing. Notably their BP was assessed over 24-hr as opposed to just 2-hr post-acute exercise (Cunha *et al.,* 2006). Ciolac *et al.* (2009) recognize their ability to promote INT training is limited, as their conclusions are based around a single bout of exercise only. Nonetheless, they do present

a number of hypotheses for INT exercise as a more efficient method of lowering BP in HTN. One such theory is that exercise bouts of different intensities cause a pattern of alternating higher and lower shear stress is skeletal muscle vasculature (Niebauer & Cooke, 1996); which stimulates greater improvements in endothelial function than constant flow (CON).

2.5.2 Interval versus Continuous Training-Programmes

The anti-hypertensive benefits of aerobic training sit in the region of 3 - 10.5 for systolic and 2 - 7.6 mmHg for diastolic BP (Whelton *et al.,* 2002; Cornelissen & Fagard, 2005). Typically training of low intensity has been reported to produce the same if not greater BP benefits as higher intensities (Kokkinos *et al.,* 2009). Whilst evidence is growing in support of more vigorous training for HTN (Haskell *et al.,* 2007) the benefits over CON are still challenged (Cornelissen *et al.,* 2009). Most HTN studies have implemented intensities <85% HR_{max} which could explain why a dose-dependant relationship between intensity and BP reduction not always evident (Molmen-Hansen *et al.,* 2011), or indeed statistically significant (Nemoto *et al.,* 2007).

Ciolac *et al.* (2010) compared the effects of two 16-week treadmill training programs match for total work done: INT (2-min at ~55%; 1-min at ~85% VO_{2peak}; total 40-min) versus CON (40-min at ~65 VO_{2peak}). They used young normotensive women at high familial risk (FH+) of HTN, whom typically display exaggerated BP response to exercise. Results showed INT was more effective than CON at improving the BP response to exercise as well as increasing overall cardiorespiratory fitness as shown by greater VO_{2max} (15.8 ± 6.3% vs 8.9 ± 6.1%). Mechanistically, the authors suggested that the lower BP was from the improved vasodilatory markers (reduced NA, reduced ET-1, increased NO).

Tjønna *et al.* (2008) implemented a similar design of two supervised, volume-matched training-programmes; INT alternated between 4-min at 90% HR_{max} and 3-min at 70% HR_{max}, whilst CON maintained 70% HR_{max} for a longer total duration. They used metabolic syndrome patients, whom typically have elevated BP and increased risk of mortality from heart disease. The INT program significantly increased VO_{2max} by 35%, whilst reduced SBP, DBP and MAP by 9, 6 and 6 mmHg. It proved to be more effective than CON at improving aerobic fitness and reversing disease risk factors. This suggests that for a given

‘dose’ of exercise, adopting an INT program will encourage greater physiological adaptations, particularly in regard to cardiorespiratory fitness.

Molmen-Hansen *et al.* (2011) performed the first randomized interventional study that employed intensities in > 90% HR_{peak} (INT); using hypertensive individuals (n=88) aged 52 ± 7.8 years. Significantly greater reductions in SBP/ DBP occurred with INT (12/8 mmHg) over CON (4.5/3.5 mmHg). The magnitude of this BP reduction indicated that INT exercise had effect comparable to a single antihypertensive drug. Nyberg *et al.* (2012) and Eicher *et al.* (2010) also used hypertensives and reported similar BP benefits, which supports that this population can tolerate more strenuous types of exercise. Based on their findings, Molmen-Hansen *et al.* (2011) stated the ACSM guidelines for HTN should be updated to include prescription of more vigorous exercise.

Astorino *et al.* (2012) were the only study that failed to note BP reductions with INT training. Although they used a similar cyling protocol (Wingate test) to other researchers (Whyte *et al.*, 2010; Chan & Burns, 2013), participants had more days resting than training per week. It is likely the interval bouts were effective acutely, but were not performed regularly enough to translate into maintained BP reduction. This highlights the frequency of exercise training must be considered alongside the type.

From the aforementioned studies and other work in this area (Tjønna *et al.,* 2009; Eicher *et al.,* 2010; Nybo *et al.,* 2010; Burns *et al.,* 2012; Nyberg *et al.,* 2012) it can be concluded that; INT exercise stimulates desirable physiological adaptations that are at least comparable to CON exercise, but occur over a reduced time scale.

2.6 Aims and Hypotheses

There were two main objectives of present study. Firstly, to establish whether there was a period of sustained post exercise hypotension (PEH) following submaximal arm crank ergometry in a normotensive population of fit healthy young men. Secondly, to determine if the magnitude and duration of this acute blood pressure response varied following constant load continuous (CON) exercise, or high intensity interval (INT) exercise, of equivalent duration, intensity and volume of work performed.

In accordance with these aims and existing literature, there were two main hypotheses. The first being a sustained post exercise hypotension of systolic and diastolic blood pressure would occur in relation to baseline, for the duration of passive recovery (60-min) from submaximal arm crank ergometry (30-min). The second that the post exercise hypotension would be of greater magnitude and duration following INT (40% and 80% W_{peak}) than CONT (60% W_{peak}).

CHAPTER 3

METHODS

3.1 Participants

Eight physically active, young, Caucasian, non-smoking males, aged between 19 and 22 yrs were recruited into this study, which was approved by the Cardiff School of Sport Ethics and Research Committee (Appendix A). Mean (±SD) values of physical characteristics of the sample are presented in Table 1. Prior to testing each participant completed a PAR-Q (Thomas *et al.,* 1992) (Appendix B). Having had the research design and all procedures explained to them, they provided written informed consent (Appendix C).

Table 1. Mean (±SD) values of physical characteristics of the sample (n=8)

Variables	Mean (±SD)
Age (yrs)	21 (1)
Height (m)	1.83 (0.08)
Mass (kg)	79 (7)
Body Mass Index (BMI) ($kg.m^{-2}$)	23.7 (1.2)
Peak Oxygen Consumption (VO_{2peak}) ($ml.min^{-1}.kg^{-1}$)	34.1 (7.2)
Peak Power (W_{peak}) (W)	160 (33)

None of the participants had any contraindications to exercise, nor were they taking any chronic medications. In addition, they had all been performing regular physical activity over the preceding 12 months; defined as ≥ 60-min moderate to high intensity aerobic exercise, ≥ 4 days/wk. Anthropometric measurements were taken during the initial laboratory visit. Their body mass index (BMI) was calculated from height (m) and body mass (kg) data to be between 20 and 25 $kg.m^2$.

3.2 Study Design

Each participant attended the physiology laboratory on 3 separate occasions, each separated by ≥ 48-hrs but within 7-days. During the first visit they underwent a peak oxygen consumption (VO_{2peak}) test. Data from this preliminary test informed the exercise intensity for the subsequent constant load continuous test (CON) and intermittent high intensity interval test (INT). The order in which the two follow-up tests were completed was counter-balanced. All exercise was performed using an electrically braked arm crank ergometer (Angio ergometer, Lode, Groningen, Netherlands) using an imposed crank rate of 75 rev•min^{-1}.

Participants were at least 2-hr postprandial, and had been abstained from caffeine consumption for at least 12-hr, along with alcohol and strenuous exercise for 24-hr. All data were collected at the same time of day between 13:00 – 16:00, to account for diurnal variation in blood pressure. Each visit took a total of 80-mins, consisting of 15-min of passive rest pre-exercise, a 5-min warm-up, a 30-min protocol and 60-min of passive recovery.

3.3 Measurements

3.3.1 Brachial Artery Blood Pressure

Blood pressure (BP) was measured manually by auscultation using a mercury sphygmomanometer and stethoscope (Littmann, Classic II). This was positioned just superior to the antecubital space, over the brachial artery. The participants remained still and quiet, seated in an upright position at the ergometer with their left arm resting supinated and at approximately heart level. The same investigator and equipment was used consistently to measure all BP readings for each participant, throughout each test. Systolic BP (SBP) was defined as the 1st Korotkoff sound and diastolic BP (DBP) as the 4th Korotkoff sound.

A total of 13 SBP and 13 DBP values were recorded for each test. Baseline values were taken during the 15-min rest prior to exercise, as an average of 3 measurements spaced 5-min apart. Post-exercise 12 further readings were taken, at 5-min intervals from

5 to 60-min inclusive. Each recording represented the average of 2 values spaced 2-min apart; such that the BP at 5-min was averaged from the 4 and 6-min values *etc.*

3.3.2 Heart Rate

Heart Rate (HR) was recorded using a short-range telemetric system (Polar Electro, RS4000, Kemple, Finland). This HR monitor was worn throughout the entire protocol. A total of 13 values were recorded for HR. The same time points were used as with BP but measurements were spaced 1-min apart; such that the BP at 5-min was averaged 4.5 and 5.5-min. This was to minimize the effect of measuring concomitant variables and to allow the same investigator to take all readings.

3.4 Procedures

3.4.1 Measurement of Peak Oxygen Uptake

Peak oxygen uptake (VO_{2peak}) was measured using a progressive and continuous test protocol following a standard 5-min submaximal warm-up. The test commenced at a work rate of 50 W, which was increased by 20 W every 2-min until volitional exhaustion, defined as an inability to maintain the working frequency (75 rev•min^{-1}). Expired gases were collected and measured using a calibrated online gas analysis system (Oxycon Pro, Warwick, UK), and oxygen uptake was plotted against work rate. Peak aerobic power (W_{peak}) was defined as the average power output sustained during the final minute of the exhaustive test. W_{peak} was used to prescribe relative exercise intensities for the CON and INT protocols.

3.4.2 Exercise Protocols

The continuous (CON) and interval (INT) protocols were matched for duration and total volume of work done, although each protocol varied in its pattern of intensity. Both 30-min tests were preceded by a standard 5-min warm-up at 40% W_{peak}, and followed by 60-min seated passive recovery. During the CON test, intensity was equivalent to 60% W_{peak} throughout. For the INT test, the 30-min was split into twelve intervals that alternated between 80% and 40% W_{peak} every 2.5-min. The mean (±SD) values of power output that corresponded to the relative exercise intensity are presented in Table 2. Sixty min of

passive recovery followed each exercise test during which time SBP, DBP and HR data were collected.

Table 2. Mean (±SD) values of prescribed power outputs for each protocol

Test	Intensity (W)	Power Output (W)
Continuous (CON)	60% W_{peak}	96 (20)
Interval (INT)	40% W_{peak}	64 (13)
	80% W_{peak}	128 (27)

3.5 Statistical Analysis

Descriptive statistics were calculated initially in the form of mean (±SD) values. A repeated measures two-way analysis of variance (ANOVA) was performed. The first factor was test, with 2 levels (CON/INT), and the second time, with 13 levels (baseline and 5 to 60-min recovery). Statistical significance was defined as P value ≤ 0.05. Initial checks for sphericity were made and where assumptions of sphericity were not observed, corrections to the degrees of freedom were achieved using the Huynh-Feldt technique. The interaction between test and time was also measured. An example is presented in Appendix F.

CHAPTER 4

RESULTS

4.1 Participants

The mean (±SD) values of haemodynamic characteristics of the sample are presented in Table 3. There were no significant differences in baseline SBP, MAP, DBP or HR between CON and INT tests (P>0.05). Individual physical and haemodynamic characteristics of the 8 participants are shown in Appendix D.

Table 3. Mean (±SD) values of haemodynamic characteristics of the sample (n=8)

Variables	Mean (±SD)
Resting systolic blood pressure (SBP) (mmHg)	129 (4)
Resting mean arterial pressure (MAP) (mmHg)	99 (5)
Resting diastolic blood pressure (DBP) (mmHg)	84 (6)
Resting heart rate (HR) (beats.min^{-1})	66 (7)

4.2 Blood Pressure

Table 4 shows that collectively mean (± SD) values of SBP, MAP and DBP across the entire 60-min of passive recovery were significantly reduced (P≤0.05) compared to values measured at rest. It also highlights the magnitude of the respective PEH responses and confirms there were differences (P<0.05) between tests.

Table 4. Mean values and differences in hypotensive responses between tests

Mean (± SD) mmHg. $^{\$}$Significantly lower than baseline (P≤0.05). *Significant difference between tests (P<0.001). Recovery refers to mean (± SD) value across full 60-min recovery. Difference = (INT) - (CON).

		Baseline	Recovery	PEH
SBP	Mean	129 (4)	123 (4)$^{\$}$	6 (2)
	Difference	1 (1)	2 (1)*	3 (1)
MAP	Mean	99 (5)	94 (5)$^{\$}$	5 (2)
	Difference	0 (1)	2 (1)*	2 (1)
DBP	Mean	84 (6)	80 (5)$^{\$}$	4 (2)
	Difference	0 (1)	2 (1)*	2 (1)

It is evident from Figures 1, 2, 3 and Appendix E that the interval (INT) test induced a greater PEH response associated with all BP variables. Collectively the largest and smallest BP decrements were observed in SBP (6 (2) mmHg) and DBP (4 (2) mmHg) respectively.

4.2.1 Systolic Blood Pressure

Figure 1 shows significant PEH of SBP between 5 and 60-min of passive recovery inclusive (P<0.04), and a similar pattern response during both tests. Mean (± SD) values of SBP were 125 (4), 123 (4) and 125 (4) mmHg at 5, 20 and 60-min respectively during CON; whilst were 123 (4), 120 (4) and 123 (4) during INT. The SBP nadir occurred at the same time point (20-min) during both tests but was significantly lower (P<0.02) during INT compared to CON. Mean post-exercise BP was lower with INT than CON by 3 (1) mmHg (Table 1) for the duration of recovery. However, this time x test interaction was only statistically significant (P<0.03) during the initial 30-min of the passive recovery.

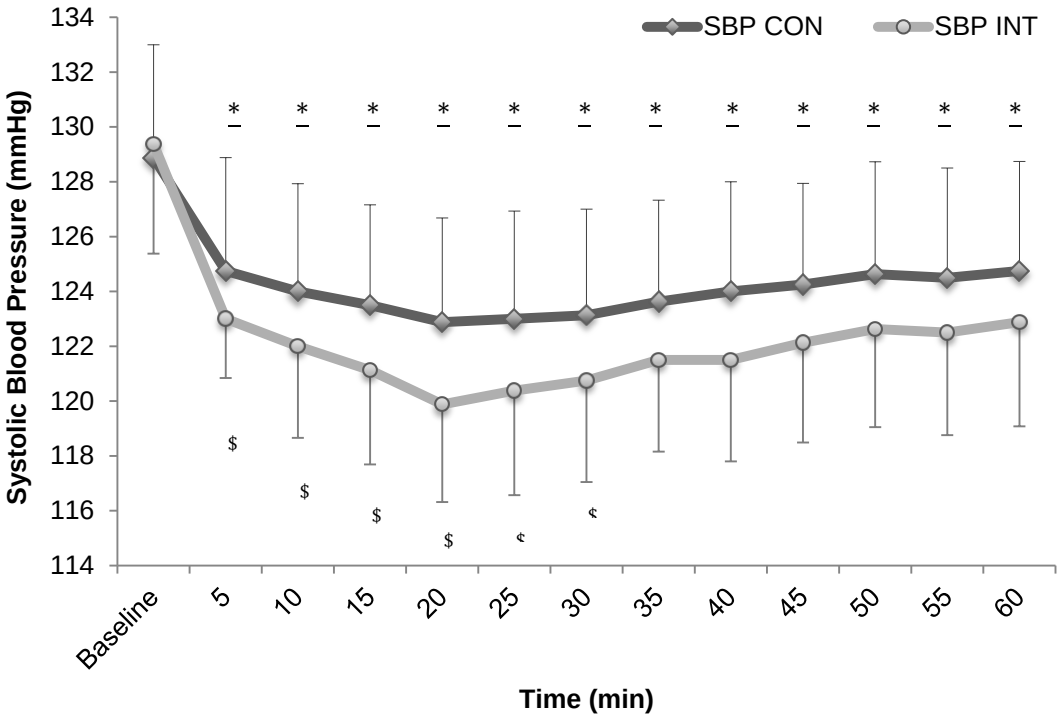


Figure 1. Mean (± SD) values of systolic blood pressure (SBP) measured at rest and during recovery. *Denotes all post exercise SBP significantly lower than baseline (P<0.05). $Denotes INT significantly lower than CON (P<0.05).

4.2.2 Diastolic Blood Pressure

Figure 2 shows DBP remained lower than baseline (84 (6) mmHg) for 60-min post exercise. This PEH was statistically significant (P<0.04) at all time points except 5-min (CON/INT) and 45-min (CON) (P>0.05). The lowest DBP were 80 (6) mmHg at 25-min (CON) and 78 (6) at 30-min (INT). Values increased gradually thereafter but remained ≥3 mmHg below baseline at 60-min. The magnitude of PEH following INT was consistently lower (P<0.03) than CON for the duration of recovery by 2 (1) mmHg (Table 3).

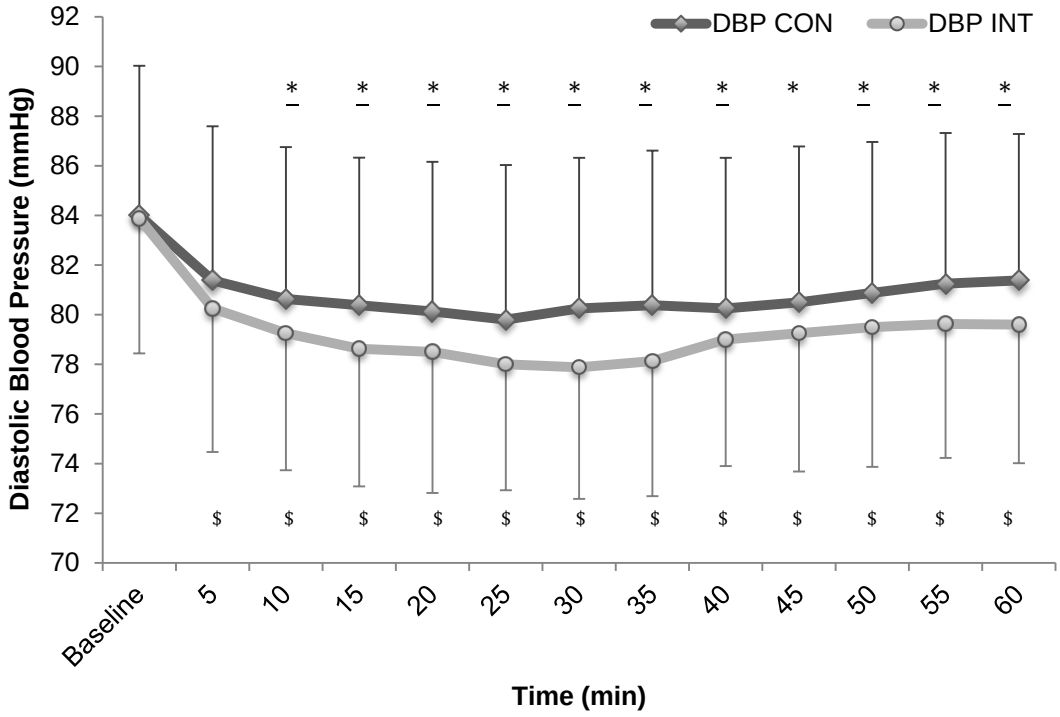


Figure 2. Mean (± SD) values of diastolic blood pressure (DBP) measured at rest and during recovery. *Denotes all DBP significantly lower than baseline (P<0.03). *Denotes only INT DBP significantly lower than baseline (P<0.03). $Denotes INT significantly lower than CON (P<0.05).

4.2.3 Mean Arterial Pressure

As MAP ≈ (SBP + 2•DBP) ÷ 3, the hypotensive response in MAP during passive recovery was between systolic and diastolic in magnitude, and followed a similar pattern. This PEH of MAP was 5 (2) mmHg relative to baseline (99 (5) mmHg) (Table 3). As shown by Figure 3, the drop in MAP occurred from 5 to 60-min inclusive during recovery (P<0.04), and was greater following INT than CON by 2 (1) mmHg (P<0.02) (Table 3).

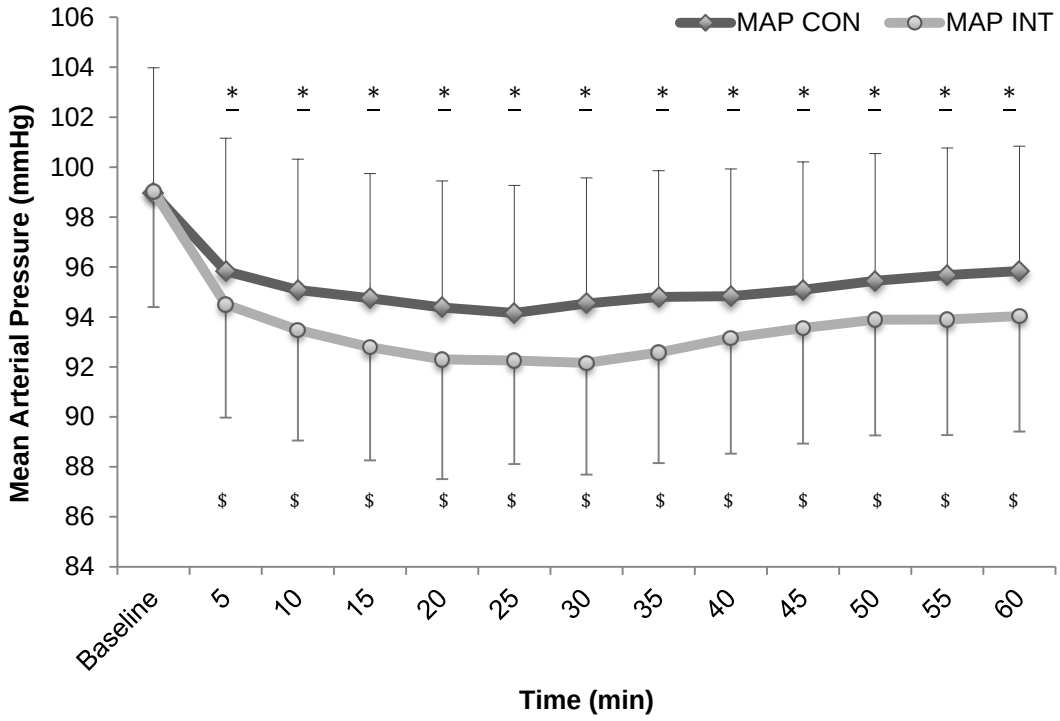


Figure 3. Mean (± SD) values of mean arterial pressure (MAP) measured at rest and during recovery. *Denotes all MAP significantly lower than baseline (P<0.04). $Denotes INT significantly lower than CON (P<0.02).

4.3 Heart Rate

Mean (± SD) heart rate increased by 28 (2) b•min^{-1} from baseline (66 (7) b•min^{-1}), to peak value at 5-min (94 (1) b•min^{-1}). Thereafter HR gradually declined throughout passive recovery. It remained significantly greater than baseline up to 50-min (73 (2) b•min^{-1}) (P<0.03). Figure 4 depicts post-exercise HR to be greater with INT, but statistical analyses revealed no main effect for test (P=0.28).

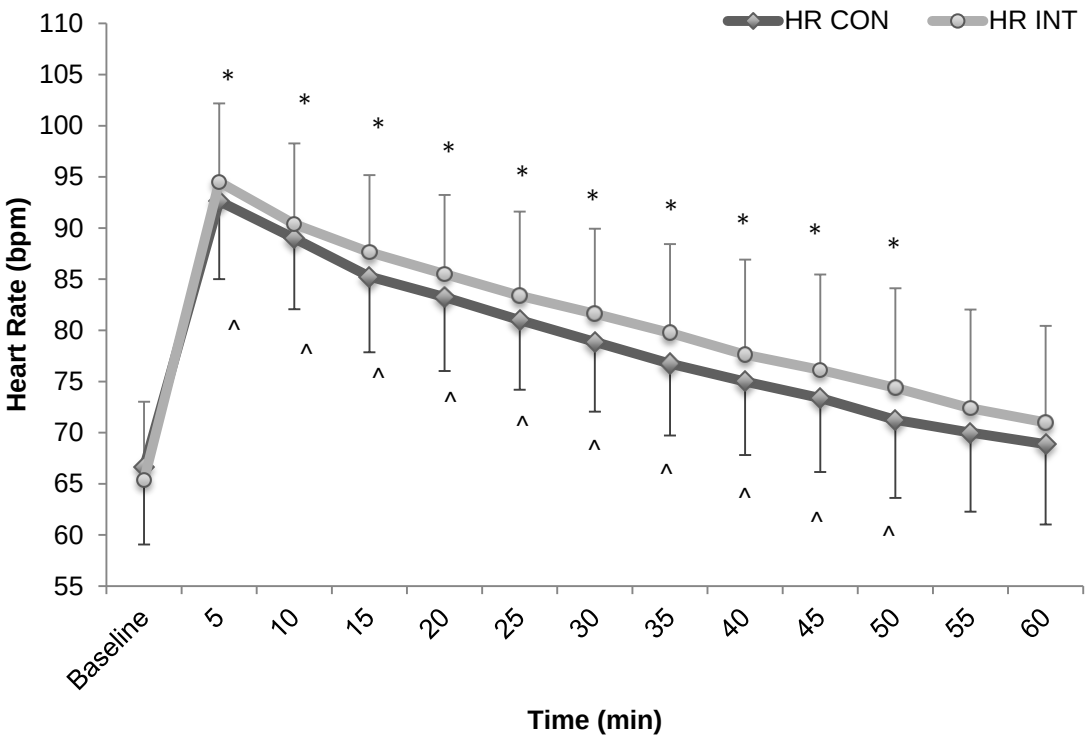


Figure 4. Mean (± SD) values of heart rate (HR) measured at rest and during recovery. *Denotes CON MAP significantly increased from baseline (P<0.03). ^Denotes INT MAP significantly increased from baseline (P<0.02)

CHAPTER 5

DISCUSSION AND CONCLUSIONS

APPENDICES

APPENDIX A

ETHICS STATUS

Name of applicant:	**Alex Coull**
Supervisor (if student project):	**Paul Smith**
School:	School of Sport, *Cardiff Metropolitan*
Student number (if applicable):	st20048764
Programme enrolled on (if applicable):	**iSES** - Intercalated Sports and Exercise
Project Title:	The impact of constant load *vs* submaximal interval exercise on post exercise hypotension, using arm crank ergometry
Expected Start Date:	01/11/13
Approximate Duration:	6 months
Funding Body (if applicable):	N/A
Other researcher(s) working on the	N/A
Will the study involve NHS patients or	No
Will the study involve taking samples of human origin	No

Non technical summary of the project

Fitzgerald (1981) was first to describe the phenomenon of Post Exercise Hypotension (PEH), defined here as a sustained drop in systolic and/or diastolic resting arterial blood pressure (BP) from baseline in the minutes/hours following single acute bouts of physical exertion.

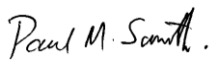
Promotion of aerobic exercise by clinicians to encourage this prolonged drop in resting BP can be utilized as a non-pharmacological technique to lower the risk of developing hypertension (HTN) and cardiovascular disease (CVD).

Using arm ergometry, this study will compare blood pressure patterns following constant load continuous exercise (CON) 60% W_{peak}) versus submaximal interval training (INT) (40 to 80% W_{peak}) during passive recovery in normotensive subjects, with view to establishing whether different exercise prescriptions affect PEH magnitude and hence potential anti-hypertensive benefit.

Most existing PEH research has implicated lower body dynamic exercise such as leg ergometry, with few studies using arm ergometry (MacDonald *et al.,* 2000), a mode that

employs much less exercising muscle mass yet still allows development of aerobic fitness. Rossow *et al.*, (2010) previously used leg ergometry to compare CON vs INT but their intervals were 'all-out' sprints in the form of Wingate tests. However a comparison of short rem responses following CON/INT comparison has not yet been explored during upper body exercise, and hence is the principal behind this study.

DECLARATION	
I confirm this project conforms with the Cardiff Met Research Governance Framework	
Applicant:	Date:
Alex Coull	31st October 2013
FOR STUDENT PROJECTS ONLY	
Name of supervisor:	Date:
Paul Smith	1st November 2013
Signature of supervisor:	



APPENDIX B

PHYSICAL ACTIVITY READINESS QUESTIONNAIRE (PAR-Q)

STRICTLY CONFIDENTIAL

Please answer questions truthfully and completely. The sole purpose of this questionnaire is to ensure that you are in a fit and healthy state to complete the exercise test. Please circle yes/no as appropriate.

Name ..... D.O.B .....

1. Have you had to consult your doctor in the last 6 months?	Yes/No
If yes, please give relevant details to test supervisor	
2. Are you presently taking any form of medication?	Yes/No
If yes, please give relevant details to test supervisor	
3. Do you / have you ever suffered from:	
Asthma (*or other breathing problem)*	Yes/No
Diabetes	Yes/No
Epilepsy	Yes/No
Hypertension (*High blood pressure*)	Yes/No
4. Do you/have you ever suffered from, any heart complaint?	Yes/No
5. Is there a history of heart disease in your family?	Yes/No
6. Do you currently have any form of muscle or joint injury?	Yes/No
7. Do you know anything that may prevent you completing the tests?	Yes/No
8. Have you suffered from palpitations, heart murmur or dizziness?	Yes/No

Do you have any questions before proceeding?

_____

As a participant I certify that I have answered these questions truthfully to the best of my knowledge.

Participant's signature:

Date:

APPENDIX C

INFORMED CONSENT FORM

Title of Project: *******

Name of Researcher: Alex Coull

Participant to complete this section

Please initial each box

1. ☐ I confirm that I have read and understand the information sheet dated for this 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 I am free to withdraw at any time, without giving any reason, without my relationship with Cardiff Met, or my legal rights, being affected.
3. ☐ I understand that relevant sections of any of research notes and data collected during the study may be looked at by responsible individuals from Cardiff Met for monitoring purposes, where it is relevant to my taking part in this research. I give permission for these individuals to have access to my records.
4. ☐ I agree to take part in this study

Name of Participant: **Name** of person taking consent:

Signature of Participant: **Signature** of person taking consent:

Date: **Date**:

When completed, 1 copy for participant and 1 copy for researcher

APPENDIX D

INDIVIDUAL CHARACTERISTICS OF THE SAMPLE

Table of individual physical and haemodynamic characteristics of the sample (n=8)

BMI, Body Mass Index; VO_{2peak}, peak oxygen consumption; SBP, systolic blood pressure; MAP, mean arterial pressure; DBP, diastolic blood pressure; HR, heart rate; W_{peak}, peak power; SD, Standard Deviation.

Subject (n=8)	Age (yrs)	Height (m)	Mass (kg)	BMI ($kg.m^{-2}$)	VO_{2peak} ($ml.min^{-1}.kg^{-1}$)	Resting SBP (mmHg)	Resting MAP (mmHg)	Resting DBP (mmHg)	Resting HR (mmHg)	W_{peak} (W)	60% W_{peak} (W)	40% W_{peak} (W)	80% W_{peak} (W)
1	22	1.8	78	24.1	36.5	128	101	88	75	150	90	60	120
2	20	1.9	78	21.6	41.8	128	102	89	55	170	102	68	136
3	21	1.89	89	24.9	23	132	103	88	63	140	84	56	112
4	21	1.87	87	24.9	35.3	132	97	79	75	170	102	68	136
5	19	1.87	82	23.4	31.8	133	103	88	73	170	102	68	136
6	20	1.67	69	24.7	24.2	125	93	78	64	90	54	36	72
7	21	1.75	70	22.9	40.3	122	90	74	59	180	108	72	144
8	20	1.86	81	23.4	39.7	133	103	88	64	200	120	80	160
Mean	21	1.83	79	23.7	34.1	129	99	84	66	160	96	64	128
(±SD)	1	0.08	7	1.2	7.2	4	5	6	8	33	20	13	27

APPENDIX E

MAGNITUDE OF PEH

Table of mean (± SD) values for blood pressure and post exercise hypotension in response to each test

Mean (± SD) mmHg. SBP, systolic blood pressure; MAP, mean arterial pressure; DBP, diastolic blood pressure; CON, continuous test; INT, interval test; PEH, post exercise hypotension

		Baseline	Recovery	PEH
SBP	CON	129 (4)	124 (4)	5 (2)
	INT	129 (4)	122 (4)	8 (2)
MAP	CON	99 (5)	95 (5)	4 (1)
	INT	99 (5)	93 (4)	6 (2)
DBP	CON	84 (6)	81 (6)	3 (1)
	INT	84 (5)	79 (5)	5 (2)

APPENDIX F

STATISTICAL PACKAGE FOR SOCIAL SCIENCES (SSPS)

Two-way analysis of variance (ANOVA) and post-hoc analyses were run on the mean (± SD) values for each variable, for each time point and test. Systolic blood pressure (SBP) data has been used below as an example.

Table of Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Test	1.000	.000	0	.	1.000	1.000	1.000
Time	.000	.	77	.	.206	.328	.083
Test * Time	.000	.	77	.	.214	.350	.083

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept
Within Subjects Design: Test + Time + Test * Time

b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Table of Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Test	Sphericity Assumed	212.019	1	212.019	30.396	.001
	Greenhouse-Geisser	212.019	1.000	212.019	30.396	.001
	Huynh-Feldt	212.019	1.000	212.019	30.396	.001
	Lower-bound	212.019	1.000	212.019	30.396	.001
Error(Test)	Sphericity Assumed	48.827	7	6.975		
	Greenhouse-Geisser	48.827	7.000	6.975		
	Huynh-Feldt	48.827	7.000	6.975		
	Lower-bound	48.827	7.000	6.975		
Time	Sphericity Assumed	716.067	12	59.672	64.830	.000
	Greenhouse-Geisser	716.067	2.475	289.279	64.830	.000
	Huynh-Feldt	716.067	3.935	181.991	64.830	.000
	Lower-bound	716.067	1.000	716.067	64.830	.000
Error(Time)	Sphericity Assumed	77.317	84	.920		
	Greenhouse-Geisser	77.317	17.327	4.462		
	Huynh-Feldt	77.317	27.542	2.807		
	Lower-bound	77.317	7.000	11.045		
Test * Time	Sphericity Assumed	33.106	12	2.759	4.368	.000
	Greenhouse-Geisser	33.106	2.573	12.864	4.368	.021
	Huynh-Feldt	33.106	4.199	7.884	4.368	.006
	Lower-bound	33.106	1.000	33.106	4.368	.075
Error(Test*Time)	Sphericity Assumed	53.048	84	.632		
	Greenhouse-Geisser	53.048	18.014	2.945		
	Huynh-Feldt	53.048	29.394	1.805		
	Lower-bound	53.048	7.000	7.578		

Table of Pairwise Comparisons (Test * Time)

Time	(I) Test	(J) Test	Mean Difference (I–J)	Std. Error	Sig.[b]	95% Confidence Interval for Difference[b]	
						Lower Bound	Upper Bound
1	1	2	-.500*	.189	.033	-.947	-.053
	2	1	.500*	.189	.033	.053	.947
2	1	2	1.750*	.726	.047	.034	3.466
	2	1	-1.750*	.726	.047	-3.466	-.034
3	1	2	2.000*	.627	.015	.518	3.482
	2	1	-2.000*	.627	.015	-3.482	-.518
4	1	2	2.375*	.375	.000	1.488	3.262
	2	1	-2.375*	.375	.000	-3.262	-1.488
5	1	2	3.000*	.567	.001	1.659	4.341
	2	1	-3.000*	.567	.001	-4.341	-1.659
6	1	2	2.625*	.625	.004	1.147	4.103
	2	1	-2.625*	.625	.004	-4.103	-1.147
7	1	2	2.375*	.498	.002	1.198	3.552
	2	1	-2.375*	.498	.002	-3.552	-1.198
8	1	2	2.125*	.441	.002	1.083	3.167
	2	1	-2.125*	.441	.002	-3.167	-1.083
9	1	2	2.500*	.500	.002	1.318	3.682
	2	1	-2.500*	.500	.002	-3.682	-1.318
10	1	2	2.125*	.350	.001	1.296	2.954
	2	1	-2.125*	.350	.001	-2.954	-1.296
11	1	2	2.000*	.567	.010	.659	3.341
	2	1	-2.000*	.567	.010	-3.341	-.659
12	1	2	2.000*	.598	.012	.587	3.413
	2	1	-2.000*	.598	.012	-3.413	-.587
13	1	2	1.875*	.581	.014	.502	3.248
	2	1	-1.875*	.581	.014	-3.248	-.502

Based on estimated marginal means

*. The mean difference is significant at the

b. Adjustment for multiple comparisons: Bonferroni.

Table of Pairwise Comparisons (Test * Time)

(I) Time	(J) Time	Mean Difference (I–J)	Std. Error	Sig.[b]	95% Confidence Interval for Difference[b]	
					Lower Bound	Upper Bound
1	2	5.250*	.551	.002	2.036	8.464
	3	6.125*	.588	.001	2.694	9.556
	4	6.813*	.574	.001	3.462	10.163
	5	7.750*	.701	.001	3.662	11.838
	6	7.438*	.664	.001	3.562	11.313
	7	7.188*	.661	.001	3.331	11.044
	8	6.563*	.630	.001	2.888	10.237
	9	6.375*	.666	.002	2.487	10.263
	10	5.938*	.710	.005	1.796	10.079
	11	5.500*	.707	.009	1.375	9.625
	12	5.625*	.646	.004	1.856	9.394
	13	5.313*	.619	.005	1.700	8.925
2	1	-5.250*	.551	.002	-8.464	-2.036
	3	.875*	.125	.017	.146	1.604
	4	1.563*	.148	.001	.702	2.423
	5	2.500*	.350	.002	1.043	3.958