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The Effects of Hydration Status on Post-exercise Hypotension in Healthy Normotensive Recreationally Active Adults

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Riley Dixon

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Abstract

Post-exercise hypotension (PEH), the acute reduction in blood pressure (BP) following a single exercise bout, contributes to long-term BP reductions associated with regular exercise. The magnitude and duration of PEH are modified by individual and exercise characteristics, yet the effect of hydration status has rarely been examined. Hypohydration may amplify PEH by reducing plasma volume and venous return. This study determined the effect of hydration status on PEH following moderate-intensity cycling in healthy, normotensive, recreationally active adults. Nine participants completed two 30-minute cycling bouts at 80% of functional threshold power under euhydration and hypohydration conditions. Hypohydration was induced through 18 hours of fluid restriction, with no subsequent rehydration. Heart rate (HR) was recorded every two minutes and rating of perceived exertion (RPE) every five minutes during exercise, and BP was measured every five minutes during a 30-minute standing recovery. Urine specific gravity was greater in the hypohydration vs. euhydration condition (1.023 ± 0.002 vs. 1.009 ± 0.007, t-test – $P = 0.004$). Despite matched power output between conditions, a main effect of hydration was present for HR during exercise ($P = 0.014$) and recovery ($P = 0.019$). Post-hoc comparisons identified higher RPE with hypohydration at minute 25 (7 ± 1 vs 6 ± 2, $P = 0.009$) and 30 (8 ± 1 vs 6 ± 2, $P < 0.001$). The maximum reduction from baseline in systolic BP (SBP) was greater in the hypohydrated condition (-12 ± 3 vs -6 ± 5 mm Hg, t-test – $P = 0.002$), with no difference for diastolic BP (-8 ± 6 vs -8 ± 6 mm Hg, t-test – $P = 0.965$) or mean arterial pressure (MAP) (-8 ± 3 vs -5 ± 5 mm Hg, t-test – $P = 0.138$). Analysed as the change from baseline at each recovery time point, a main effect of hydration was present for SBP ($P < 0.001$) and MAP ($P = 0.050$) with greater reductions following hypohydration. Hypohydration produced PEH sufficient, in some cases, to induce orthostatic intolerance and syncope. As hypohydration is common in recreationally active adults and athletes, these findings carry practical relevance for safe management of post-exercise recovery.

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Table of Contents

Abstract ..... *ii*

Acknowledgements ..... *iii*

Table of Contents ..... *iv*

List of Tables ..... *vi*

List of Figures ..... *vi*

Abbreviations ..... *vii*

Chapter One : Introduction *1*

1.1 Background **1**

1.2 Study aims **3**

1.3 Study hypothesis **3**

Chapter Two : Literature Review *4*

2.1 Overview **4**

2.2 Normal Regulation of BP **4**

2.2.1 Arterial baroreflex control of BP 5

2.2.2 Blood volume and hormonal control of BP at rest 6

2.2.3 Blood pressure control during endurance exercise 7

2.2.4 Hydration and BP responses during endurance exercise 8

2.3 Post-exercise hypotension **9**

2.3.1 Intensity 10

2.3.2 Duration 14

2.3.3 Modality 15

2.3.4 Training status 17

2.4 Mechanisms of PEH **19**

2.4.1 Autonomic and haemodynamic mechanisms 19

2.4.2 Local vascular mechanisms 20

2.4.3 Sex differences in PEH mechanisms 21

2.5 Hypohydration and PEH **22**

Chapter Three : Methods *29*

3.1 Ethics and informed consent **29**

3.2 Participants **30**

3.3 Study design **31**

3.4 Experimental design **32**

3.5 Hypohydration visit **33**

3.6 Euhydration visit **33**

3.7 Exercise protocol **34**

3.8 Measurements **35**

3.8.1 Cardiovascular 35

3.8.2 Core temperature 36

3.8.3 Anthropometric 36

3.8.4 USG 36
3.8.5 Rating of perceived exertion 37
3.9 Data acquisition 37
3.10 Calculations 37
3.11 Statistical analysis 37
Chapter Four : Results 39
4.1 Pre-exercise measures 39
4.2 Power 40
4.3 Core temperature 40
4.4 Rating of perceived exertion 41
4.5 Heart rate 42
4.6 Blood pressure 43
4.7 Safety/Adverse events 48
Chapter Five : Discussion 49
5.1 Hydration status 49
5.2 Physiological responses during exercise 50
5.3 Physiological responses post-exercise 52
5.4 Orthostatic intolerance and safety 54
5.5 Implications 56
5.6 Limitations 58
5.7 Future directions 60
5.8 Conclusions 61
Chapter Six : References 63
Appendices 75

List of Tables

Table 1. Participant physical characteristics, resting cardiovascular variables, and cycling power outputs 39

List of Figures

Figure 1. Participant recruitment overview. Consort diagram based off framework by Hupalo et al. (2023). 30
Figure 2. Experimental protocol overview for visits 2 and 3. Pre- and post-exercise measures include HR, BP and core temperature. Warm-up was 50% of FTP for 5 minutes and cool-down was 40% of FTP for 3 minutes. Image created with BioRender.com. 34
Figure 3. Rating of perceived exertion (RPE) during exercise in the euhydration and hypohydration conditions (n = 9). Data are mean ± SD. * indicates a difference between conditions at minutes 25 and 30 (P ≤ 0.009). 41
Figure 4. Heart rate during exercise (n = 9). Data are mean ± SD. * indicates a difference between conditions (P ≤ 0.05)..... 42
Figure 5. Heart rate during recovery (n = 7). Data are mean ± SD. * indicates a difference between conditions (P < 0.05). End of exercise denotes HR at the final minute of the 30-minute exercise bout. 43
Figure 6. Systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) during recovery from cycling exercise (n =7). Data are mean ± SD. * indicates a difference between conditions (P < 0.05). 45
Figure 7. Change from baseline to nadir for systolic blood pressure (ΔSBP), diastolic blood pressure (ΔDBP) and mean arterial pressure (ΔMAP) (n = 9). * indicates a significant difference between conditions (P < 0.05). 46
Figure 8. Absolute change from baseline at each recovery time point for systolic blood pressure (ΔSBP), diastolic blood pressure (ΔDBP) and mean arterial pressure (ΔMAP) (n = 7). Data are mean ± SD. 47

Abbreviations

ADH	Antidiuretic hormone
ANOVA	Analysis of variance
ANS	Autonomic nervous system
BP	Blood pressure
CO	Cardiac output
CP	Critical power
DBP	Diastolic blood pressure
ECG	Electrocardiogram
FTP	Functional threshold power
GPAQ	Global Physical Activity Questionnaire
HIIE	High-intensity interval exercise
HR	Heart rate
HR_{max}	Maximum heart rate
HRR	Heart rate reserve
MAP	Mean arterial blood pressure
MIIE	Moderate-intensity interval exercise
NO	Nitric oxide
PEH	Post-exercise hypotension
PV	Plasma volume
RAAS	Renin-angiotensin-aldosterone system
RPE	Rating of perceived exertion
SBP	Systolic blood pressure
SD	Standard deviation
SV	Stroke volume
SVR	Systemic vascular resistance
T_{core}	Core Temperature
USG	Urine specific gravity
$\dot{V}O_{2max}$	Maximal oxygen uptake
$\dot{V}O_{2peak}$	Peak oxygen uptake

Chapter One: Introduction

1.1 Background

Hypertension, defined as a sustained elevation in arterial blood pressure (BP) (≥140 mm Hg systolic or ≥90 mm Hg diastolic), is the most common preventable risk factor for cardiovascular disease (World Health Organisation, 2023; Zhou et al., 2021). Worldwide, 30-40% of adults aged 30-79 years are affected by hypertension, with prevalence in those aged ≥ 60 years exceeding 60%, largely due to arterial stiffening and increased vascular resistance (Alpsoy, 2020). In Aotearoa New Zealand, 31% of adults aged 30-79 years were estimated to have hypertension in 2019, with 63% diagnosed, 50% treated, and just 27% controlled (World Health Organisation, 2023). This highlights the need for ongoing screening and effective treatment strategies within primary care and community settings (McLean et al., 2013). Lifestyle factors, including physical inactivity, obesity, high sodium intake, harmful alcohol use and poor diet quality are established, modifiable risk factors for hypertension (Samadian et al., 2016). Within this, structured exercise is a key therapy for preventing and managing hypertension and cardiovascular disease non-pharmacologically.

Physical inactivity is an established contributor to cardiovascular disease burden, with an estimated 7.6% of cardiovascular disease deaths globally attributed to inactivity (Katzmarzyk et al., 2022) and 1.6-13% attributed to hypertension specifically (Börjesson et al., 2016; Katzmarzyk et al., 2022). Exercise offers numerous health benefits, including the prevention of several chronic diseases and improvements in musculoskeletal health (Warburton et al., 2006). Furthermore, it enhances mood and stress regulation, supporting mental well-being

(Sharma, 2006). In the context of BP management, a single exercise session can lower BP below pre-exercise levels for several hours, a response termed post-exercise hypotension (PEH – as described below in section 2.3). When exercise is performed regularly, these acute reductions likely contribute to long-term reductions in BP (Cornelissen et al., 2013; Cornelissen & Fagard, 2005). Importantly, the magnitude and duration of PEH are not fixed; they vary with individual characteristics and manipulation of exercise variables (Brito et al., 2014). These variables include exercise intensity, duration and modality, training status, recovery posture, and hydration status (Trammel, 2023). These factors that shape the PEH and blood pressure adaptations to exercise are highly relevant to the Aotearoa New Zealand context, where practical and achievable exercise prescriptions in community and clinical settings may be used to improve BP control and combat the high prevalence of cardiovascular disease. However, what remains unclear is the extent to which hydration status modifies the magnitude and duration of PEH.

Hydration status is a particularly relevant factor to consider, as hypohydration, defined as a deficit in total body water, is common among both athletic and recreationally active populations (Burke, 1997). For example, hypohydration has been reported in 66% of collegiate athletes based on pre-practice urine testing (Volpe et al., 2009), while a systematic review of soccer players reported hypohydration prevalence of 55% among recreational players and over 60% in elite players (Chapelle et al., 2020). While data specific to recreationally active adults are limited, these findings indicate hypohydration is common in both elite and recreational athlete populations. However, hydration status is not consistently measured or reported in studies investigating PEH (Forjaz et al., 2004; Forjaz et al., 1998; MacDonald et al., 2000; Memon et al., 2011; Senitko et al., 2002). Despite the potentially

confounding effect of hypohydration on the PEH response, the relationship between hydration status and blood pressure regulation acutely following exercise remains poorly understood.

To the best of the researcher's knowledge, no previous study has directly investigated the effect of beginning fixed-intensity cycling exercise in a hypohydrated state on the magnitude of PEH. Establishing these responses first in healthy, recreationally active individuals will help inform exercise prescription for other populations, such as those with impaired cardiovascular function (e.g. hypertension).

1.2 Study aims

The objective of this thesis is to extend the current body of knowledge on how hydration status shapes cardiovascular responses to exercise. The aim of the thesis is:

1. To determine the effect of hydration status on the PEH responses to moderate intensity cycling exercise in healthy, normotensive, recreationally active adults.

1.3 Study hypothesis

It is hypothesised that:

1. In healthy normotensive recreationally active adults aged between 18-35 years, reductions in BP will be greater under the hypohydration condition compared with euhydration.

Chapter Two: Literature Review

2.1 Overview

This review focuses on PEH following endurance exercise in healthy normotensive individuals, including the mechanisms underlying the response, the role of hydration status as a key modifier, and how manipulation of exercise variables may inform safe and effective prescription, with consideration of how training status shapes the response. While the review centres on healthy populations, the evidence reviewed is relevant to exercise prescription in clinical populations such as those with hypertension, where the post-exercise BP response carries particular significance. A structured literature search was conducted in PubMed and Scopus using combinations of the following keywords: post-exercise hypotension, blood pressure, hypohydration/dehydration, hydration status, endurance exercise, and aerobic exercise. Titles and abstracts were screened for relevance, followed by full-text review of selected studies. A narrative approach was used to synthesise current evidence on PEH, the effects of hypohydration, and the underlying mechanisms.

2.2 Normal Regulation of BP

Although BP regulation has been extensively researched, it is complex and varies considerably between individuals (Chopra et al., 2011). Multiple systems interact to maintain BP near a tightly regulated set point by adjusting cardiac output (CO), systemic vascular resistance (SVR) and circulating blood volume (Chopra et al., 2011; Raven & Chapleau, 2014). The following provides a brief overview of the key components relevant to BP regulation, including the role of hydration.

2.2.1 Arterial baroreflex control of BP

Short-term control of mean arterial blood pressure (MAP), occurring over seconds to minutes, is governed by the arterial baroreflex, which continuously modulates CO and SVR via the autonomic nervous system (ANS) (Guyenet, 2006). MAP is the product of CO and SVR (MAP = CO $\times$ SVR), reflecting Darcy's law. CO equals heart rate (HR) multiplied by stroke volume (SV), the latter being the amount of blood ejected with each beat (King, 2023). SV depends on several factors including preload, contractility, and afterload (King, 2023). Preload reflects the myocardial wall stress present at end-diastole, which depends on end-diastolic pressure together with ventricular chamber radius and wall thickness, as described by Laplace's law (Norton, 2001). Increased end-diastolic volume, driven by greater venous return and filling pressure, stretches myocardial fibres and produces a stronger contraction and larger SV via the Frank–Starling mechanism (King, 2023; LaCombe et al., 2025). Contractility refers to the intrinsic force of myocardial contraction independent of preload and increases with sympathetic activation and catecholamine stimulation (King, 2023). Afterload represents the myocardial wall tension during systolic ejection, which depends on arterial BP along with chamber radius and wall thickness, with arterial pressure acting as the primary determinant (King, 2023; Norton, 2001). Within this system, the arterial baroreflex acts to stabilise MAP primarily by altering autonomic outflow to the heart and vasculature (Malpas, 2004; Ogoh et al., 2012). When arterial pressure rises, stretch of the baroreceptors in the carotid sinus and aortic arch increases afferent firing, increasing parasympathetic activity and withdrawing sympathetic activity (Dampney, 2017). As a result, HR and contractility fall, producing a reduction in CO, resistance vessels vasodilate which decreases SVR, and MAP returns toward the set point (Dampney, 2017; Ogoh et al., 2012). Conversely, when arterial pressure falls,

sympathetic activity increases and parasympathetic activity decreases. HR and contractility rise which elevates CO, resistance vessels vasoconstrict, SVR increases, and MAP is restored (Dampney, 2017; Ogoh et al., 2012). The baroreflex is therefore essential because BP must be corrected within seconds to maintain adequate perfusion of the brain and other vital organs, particularly during changes in posture, such as moving from seated to standing. Sympathetic tone also helps maintain SVR at rest (Guyenet, 2006), which relates to PEH and the sections that follow, as withdrawal of sympathetic vasoconstrictor activity after exercise reduces SVR and contributes to the fall in BP (Halliwill, 2001; MacDonald, 2002), as discussed in section 2.4.1.

2.2.2 Blood volume and hormonal control of BP at rest

The kidneys influence BP over longer time scales, such as hours to days, by regulating body fluid and sodium balance (Chopra et al., 2011; Nishida et al., 2012). Reduced renal perfusion pressure, sympathetic activation, sodium depletion, or reduced body water content (hypohydration) stimulates renin release (Baffour-Awuah et al., 2023; Bernal et al., 2006). Renin converts angiotensinogen to angiotensin I, which is then converted to angiotensin II by angiotensin-converting enzyme in the pulmonary endothelium (Baffour-Awuah et al., 2023; Bernal et al., 2006). Angiotensin II is a potent vasoconstrictor of the resistance vasculature, increasing SVR and therefore MAP (MacDonald, 2002; Trammel, 2023). It also promotes renal sodium and water retention and stimulates thirst (MacDonald, 2002). Angiotensin II triggers aldosterone release from the adrenal cortex, which increases sodium reabsorption in the distal nephron, specifically the late distal tubule and collecting duct (Bernal et al., 2006). As sodium is retained, water follows osmotically, leading to expansion of extracellular fluid

volume, increased ventricular filling (preload), and increased CO (Magder, 2016). The net effect of renin-angiotensin-aldosterone system (RAAS) activation is a rise in arterial pressure through both factors of Darcy's law (i.e. CO and SVR), with a rapid increase in SVR mediated by angiotensin II-induced vasoconstriction (Triebel & Castrop, 2024) and a slower expansion of blood volume driven by aldosterone, which increases CO (Bernal et al., 2006). Chronically elevated activity of the RAAS contributes to a higher resting BP (Baffour-Awuah et al., 2023). In addition to RAAS activation, antidiuretic hormone (ADH) is released from the posterior pituitary in response to increased plasma osmolality and reduced blood volume (Cuzzo, 2023). Both of these occur during hypohydration, as plasma volume (PV) declines and plasma osmolality rises (Watso & Farquhar, 2019). These changes are detected through two pathways: osmoreceptors in the hypothalamus respond to a rise in plasma osmolality by stimulating ADH release (Cuzzo, 2023), while cardiopulmonary baroreceptors in the atria respond to reduced stretch from a fall in blood volume by signalling the hypothalamus to increase ADH secretion (Ahrabi & Poshtdar, 2025). ADH increases facultative water reabsorption in the collecting ducts and, at higher concentrations, can also promote vasoconstriction, particularly when angiotensin II activity is elevated (Cuzzo, 2023). Together, these responses help expand effective arterial blood volume and raise BP to support tissue perfusion (Cuzzo, 2023; González et al., 2020).

2.2.3 Blood pressure control during endurance exercise

During endurance exercise, blood flow is matched to metabolic demand via local vasodilation in active skeletal muscle. CO rises through increases in HR and, up to moderate intensities (such as 50–70% of $\dot{V}O_{2max}$), SV (Kim & Ha, 2016; Sharman & La Gerche, 2015). As CO increases

during endurance exercise, vasodilation in active skeletal muscle tends to lower SVR, but this is partly offset by sympathetic vasoconstriction in the splanchnic and renal circulations, which helps maintain BP and redistribute blood flow to working muscles (Hayashi et al., 2012). As a result, systolic BP (SBP) typically rises, diastolic BP (DBP) is unchanged or slightly reduced, and MAP increases during dynamic exercise using large muscle groups such as running or cycling, helping muscle perfusion (Kim & Ha, 2016; Sharman & La Gerche, 2015). The arterial baroreflex resets to a higher set point during endurance exercise so it can continue to buffer beat-to-beat fluctuations while allowing the elevated BP and CO required for adequate perfusion and oxygen delivery (Raven et al., 2019). Sympathetic activation during endurance exercise stimulates renin release, activating the RAAS to help maintain BP and circulating volume (Williams et al., 2013). With regular training, resting RAAS activity appears blunted, including reduced circulating angiotensin II, which is consistent with lower vasoconstrictor tone and a lower resting BP outside of exercise (Baffour-Awuah et al., 2023).

2.2.4 Hydration and BP responses during endurance exercise

Much of the evidence on hydration and BP during endurance exercise comes from protocols where hypohydration develops over prolonged cycling in the heat. Compared with a euhydrated state, hypohydration of approximately 5% body mass over 2 hours of cycling at 35°C reduces CO, as SV falls and the compensatory rise in HR is insufficient to fully restore it (González-Alonso et al., 1995). The reduction in CO produces a lower within-exercise MAP when hypohydrated than when euhydrated, driven mainly by a smaller rise in SBP (González-Alonso et al., 1995). Hypohydration alone does not appear to account for this response. When hypohydration and hyperthermia were separated experimentally, each independently

reduced SV only modestly, and a rise in HR was sufficient to maintain CO and MAP near euhydrated levels (González-Alonso et al., 1997). Only when the two occurred together did the reductions in SV compound to the point that HR, already near maximal, could no longer compensate, so CO and MAP fell below euhydrated levels (González-Alonso et al., 1997). The attenuation of MAP therefore appears to require the combination rather than hypohydration alone. The hyperthermic component adds further strain through thermoregulatory demand, where cutaneous vasodilation raises perfusion of the skin, which then competes with the working muscles for blood flow (González-Alonso et al., 1997; Sawka et al., 2015). Even when BP is maintained, cardiovascular strain is still evident, with the degree of fluid loss during prolonged endurance exercise associated with a greater rise in HR as PV falls (McConell et al., 1997). Importantly, this evidence concerns hypohydration developing during exercise, rather than beginning exercise already hypohydrated. Starting in a hypohydrated state means PV is reduced from the outset, which may alter the cardiovascular response to exercise differently from exercise-induced dehydration.

2.3 Post-exercise hypotension

PEH is defined as an acute reduction in arterial BP, including MAP, SBP and DBP, following a single exercise bout (Aly & Yeung, 2023). Although there is no universal threshold for the magnitude or duration of PEH, reductions can occur within minutes after exercise and persist for several hours. For example, reductions of 3-5 mm Hg in SBP have been observed for up to 5 hours following high-intensity interval running in normotensive individuals (Dantas et al., 2017). The magnitude and duration of PEH can vary considerably between individuals and exercise protocols, with the response dependent on exercise intensity, duration, modality and

training status. Whilst each of these variables are discussed in the upcoming sections, exercise intensity has been the most extensively investigated and is often considered a primary driver of the PEH response. However, the effect of exercise intensity on PEH appears to be modified by recovery posture, with differences between exercise intensities being largely abolished when recovery is performed in the supine position (Lu et al., 2024). Furthermore, higher exercise intensities may produce greater PEH and increase the likelihood of orthostatic intolerance or syncope during recovery with superimposed orthostatic stress (Mündel et al., 2015). Orthostatic intolerance presents as light-headedness or dizziness, and in more severe cases, progresses to syncope. These symptoms are underpinned by a reduction in cerebral perfusion (Halliwill et al., 2013; Stewart, 2012). Because recovery posture can affect the magnitude of PEH independent of other exercise variables, recovery posture is noted in the discussion of the relevant literature in the sections that follow, to contextualise some of the variability between studies. While PEH has been observed across a range of populations, the following sections focus on responses observed in normotensive and healthy individuals.

2.3.1 Intensity

Exercise intensity refers to the level of physiological stress imposed during an exercise session and can be measured using various metrics, including relative oxygen consumption (e.g. %$\dot{V}O_{2max}$ or %$\dot{V}O_{2peak}$), heart rate-based measures (e.g. %HR_{max} or %HRR), and subjective rating of perceived exertion (RPE) which correlates with these physiological measures (Garber et al., 2011). Exercise intensity has been shown to affect both the magnitude and duration of the PEH response, although findings across studies are variable. In normotensive individuals, higher exercise intensities generally produce larger and more sustained reductions in BP. For

example, cycling at 70% of $\dot{V}O_{2peak}$ has been associated with greater reductions in MAP post-exercise compared to 30 and 50% $\dot{V}O_{2peak}$, with a maximum decrease in SBP of 9.2 mm Hg at 60 minutes post-exercise, with exercise duration matched across intensities (Forjaz et al., 2004). Findings from running exercise are consistent with this, as Liu et al. (2013) found that running at 80% of $\dot{V}O_{2max}$ produced greater PEH than running at 60% $\dot{V}O_{2max}$ in young and middle-aged recreationally active endurance athletes, with duration matched between intensity conditions. In the same study, during a 60-minute seated recovery period, maximum SBP reductions reached 15.1 mm Hg in middle-aged athletes and 5.7 mm Hg in young athletes (Liu et al., 2013). Similarly, in normotensive recreationally active adults, running at 75 to 80% of $\dot{V}O_{2max}$ has been associated with greater BP reductions at 5, 30 and 60 minutes post-exercise compared to 50 to 55% of $\dot{V}O_{2max}$, again with duration held constant across conditions (Alderman et al., 2007).

Interval exercise provides further support for an intensity-dependent PEH response, as it allows higher intensities to be reached within a single exercise session. Oliveira et al. (2018) compared high-intensity interval exercise (HIIE) and moderate-intensity interval exercise (MIIE) during running in thirteen male normotensive adolescents. HIIE involved eight bouts of 1 minute at 90% of the maximal aerobic running speed, interspersed with 75 seconds of active recovery at 4 km h^{-1}. MIIE consisted of 1-minute bouts at 90% of gas exchange threshold, with the same recovery and was adjusted to match the total distance covered in the HIIE condition. Both protocols resulted in PEH, but only HIIE produced reductions that persisted 60 minutes post-exercise, indicating a more sustained response. Although the adolescent sample limits comparison to broader adult populations, these findings support the idea that higher-intensity exercise may produce a larger or more sustained PEH response.

Recent work in cycling exercise using intensities around critical power (CP) also supports the importance of exercise intensity. CP represents the maximum power output achievable for a sustainable period without fatigue, marking the boundary between heavy and severe exercise intensity domains (Jones et al., 2010; Lipková et al., 2022). In twelve recreationally active, normotensive men, cycling above and below CP was matched for total work, with cycling above CP producing significantly greater PEH (Lei et al., 2023). These findings are supported by a more recent study by Lei et al. (2026), who compared three cycling conditions performed to task failure: heavy cycling below CP, and two supra-CP conditions performed at different intensities above CP, corresponding to power outputs that resulted in task failure at 6 and 12 minutes. The heavy exercise condition was performed halfway between the gas exchange threshold and CP, and therefore lasted longer due to its lower intensity. Supra-CP cycling produced greater PEH than heavy exercise below CP, and the PEH magnitude did not differ between the two supra-CP conditions (Lei et al., 2026). Overall, these findings indicate that intensity is a strong determinant of PEH magnitude, particularly when cycling exercise is performed above CP.

However, the magnitude of PEH is not determined by intensity alone and appears to be influenced by the interaction between intensity and recovery conditions, particularly the degree of orthostatic stress imposed during recovery. When exercise intensity is compared across recovery postures, higher intensities appear to amplify PEH predominantly during standing recovery, with differences between exercise intensities largely abolished when recovery is performed in the supine position (Lu et al., 2024). The superimposed orthostatic stress generates venous pooling, which can reduce CO and consequently BP, contributing to

symptoms of pre-syncope (Halliwill et al., 2014). This is consistent with Mündel et al. (2015) who explored post-exercise orthostatic tolerance with head-up tilt and concurrent lower body negative pressure, and found that one hour of cycling at 70% HR range (calculated by $HR_{max} – HR_{rest}$ x 0.7) produced a greater PEH response and a shorter time to pre-syncope than one hour of cycling at 30% HR range (calculated by $HR_{max} – HR_{rest}$ x 0.3) in eight healthy, recreationally active males. Syncope in this setting has been linked to baroreflex resetting, persistent vasodilation and a downward shift in cerebral autoregulation (Halliwill et al., 2014).

However, not all studies have demonstrated clear differences between exercise intensities. Pescatello et al. (1991) reported no PEH following cycling at 40% and 70% $\dot{V}O_{2max}$ in a small sample of six normotensive men. Other cycling studies in normotensive adults have reported PEH but no significant differences between moderate and higher intensities when exercise duration is held constant and recovery is completed in the seated position (Forjaz et al., 1998; Macdonald et al., 1999). The cohorts in the Forjaz et al. (1998) and Macdonald et al. (1999) studies were young and healthy, with relatively low resting BP in Forjaz et al. (1998). Contrasting findings have also been reported by Leal and Del Vecchio (2022), who compared four cycling protocols matched for caloric expenditure. These included moderate intensity interval cycling, moderate intensity continuous cycling, high-intensity interval cycling and high-intensity continuous cycling. The moderate-intensity protocols were performed at 70 to 75% of maximum aerobic power, while the high-intensity protocols were performed at 85-90% of maximum aerobic power. Interestingly, both continuous exercise sessions produced greater BP reductions than interval protocols (Leal & Del Vecchio, 2022). The authors suggested that sustained vasodilation through histamine receptors and nitric oxide release might explain these findings. However, the unexpectedly low HR during the high-intensity

interval session may reflect the caloric matching approach rather than intended intensity (Leal & Del Vecchio, 2022). The population of normotensive men with a family history of hypertension further limits interpretation. Therefore, while evidence broadly supports an intensity-dependent response, conflicting findings across studies indicate that recovery posture appears to be integral to the PEH response, and since total work performed reflects both intensity and duration combined, exercise duration warrants closer consideration as a determinant of PEH.

2.3.2 Duration

Longer exercise sessions generally increase cardiovascular strain and may enhance the magnitude or persistence of PEH. PEH has been observed in normotensive individuals across a wide range of exercise durations, from brief 10-minute maximal bouts (Cunha et al., 2015) to endurance events such as half marathons (Mourot et al., 2020). Few studies have directly examined the effects of exercise duration while keeping intensity constant. MacDonald et al. (2000) investigated cycling at 70% $\dot{V}O_{2peak}$ for 15, 30 and 45 minutes in normotensive adults. Reductions in MAP, SBP and DBP were observed following all three exercise durations (15, 30 and 45 minutes), with no significant differences between them (MacDonald et al., 2000). When data were pooled across durations, the largest reductions occurred at the 45-minute post-exercise recovery time point, with SBP, DBP and MAP reduced by 12 mm Hg, 4.6 mm Hg and 7 mm Hg, respectively (MacDonald et al., 2000). However, SBP following the shorter cycling durations (15 and 30 minutes) appeared to be returning towards baseline by 60 minutes post-exercise, whereas SBP following the 45-minute exercise duration was still declining at this time point (MacDonald et al., 2000). Therefore, it is possible that longer

exercise durations may extend the PEH response, even if the overall magnitude of PEH is similar. These findings indicate that while intensity and duration are important, total work performed might be a key factor in predicting PEH. Consistent with this, Jones et al. (2007) found that in normotensive physically active males, lower-intensity exercise, when matched for total work through longer duration, can produce a similar PEH response to higher-intensity exercise. However, this study was limited to a small sample size (n = 7) and the absence of HR measurements during exercise, so the cardiovascular strain imposed by each condition could not be compared directly. The 20-minute post-exercise measurement period was also too short to establish whether the duration of PEH differed between conditions.

Overall, duration and intensity appear to act on the PEH response in different ways. Higher intensities produce both larger and more sustained reductions in BP (as described in section 2.3.1), whereas increasing duration appears to extend how long PEH persists without clearly altering its magnitude. Both duration and intensity are also inherently linked, as higher intensities cannot be sustained, meaning any manipulation of one variable limits the other. Duration is therefore best interpreted alongside exercise intensity and total work performed.

2.3.3 Modality

Despite a growing body of literature demonstrating PEH following endurance exercise, the role of exercise modality remains unclear, with most studies utilising cycling or running protocols. Regarding walking, a 7-10 mm Hg reduction in SBP was observed in normotensive men between 30 minutes and two hours after exercise (Headley et al., 1996). Similar reductions in SBP of 10 mm Hg have been reported in normotensive trained females 60

minutes following water walking (Memon et al., 2011). These data indicate that the PEH response is robust even during walking exercise. Evidence from other modalities such as swimming is limited. Lakin et al. (2013) compared moderate-intensity (60-70% HRR) cycling and swimming in healthy young untrained and triathlon-trained men and women. They found that untrained individuals showed similar PEH between cycling and swimming, with the greatest reduction in SBP at the 25-minute time point post-exercise of 3.1 mm Hg following cycling and 5.8 mm Hg following swimming (Lakin et al., 2013). In contrast, triathlon-trained participants showed a significant reduction in SBP (3.7 mm Hg) at 50 minutes post-exercise following cycling, but no reduction following swimming (Lakin et al., 2013). Therefore, training status may be important when interpreting PEH responses following swimming.

Only one known study has specifically compared the exercise modalities of running, cycling and walking. Cunha et al. (2015) investigated maximal short-term incremental exercise in 20 healthy normotensive men and found that only running produced a significant reduction in SBP during a 60-minute supine recovery. However, it is difficult to conclude that running produces the greatest PEH response due to significant limitations. The most notable limitation is that total exercise volume was not controlled, and the running modality involved substantially higher total work than the cycling and walking interventions (Cunha et al., 2015). The study also included only male participants, and recovery was measured in the supine position, which may have reduced the magnitude of the PEH response. Therefore, although PEH occurs across different endurance exercise modalities, direct comparisons remain limited, and future studies should control for intensity, duration and recovery posture. Furthermore, the differing responses observed between trained and untrained individuals following swimming (Lakin et al., 2013) indicate that training status may itself shape the

magnitude and mechanism of the PEH response, and is examined further in the following section.

2.3.4 Training status

The previous sections have shown that exercise characteristics affect the magnitude and duration of PEH. Most of the research has focused on healthy normotensive recreationally active participants, but the response in sedentary or untrained populations is less clear. In normotensive populations, it has been proposed that endurance-trained adults may show a reduced PEH response due to lower baseline BP or a greater capacity to manage sustained vasodilation from repeated exercise exposure (Senitko et al., 2002). Consistent with this, Memon et al. (2011) found greater reductions in BP in normotensive untrained women compared with trained women during immersed water walking. A similar pattern was reported by Lakin et al. (2013), where PEH following swimming was observed in untrained participants but absent in triathlon-trained participants (see Section 2.3.3). The absence of PEH in the trained group was attributed to a slower return in vagal tone and prolonged elevations in sympathetic activity and HR following swimming, which may have offset the fall in SV (Lakin et al., 2013). These findings indicate that training status may modify the post-exercise cardiovascular response, particularly in modalities such as swimming.

Longitudinal evidence also supports a role for training status. Lei, Li, et al. (2025) improved the training status of eleven healthy normotensive men through 2 weeks of high-intensity exercise training, consisting of 15 minutes of cycling at 11% above CP on 14 consecutive days. They found that this short-term training period significantly reduced orthostatic-induced PEH,

reflected by higher MAP, SBP and DBP during seated and standing recovery (Lei, Li, et al., 2025). However, cross-sectional studies have reported more mixed findings. Senitko et al. (2002) compared 16 sedentary and 16 endurance-trained men and women following 60 minutes of cycling at 60% $\dot{V}O_{2peak}$ and found a similar magnitude of PEH across groups. However, the mechanisms differed, with PEH attributed to vasodilation in sedentary men and women and endurance-trained women, but to reduced CO in endurance-trained men (Senitko et al., 2002). Consistent with this, Meade et al. (2018) found that following 60 minutes of cycling at 60% $\dot{V}O_{2peak}$, PEH in endurance-trained men was associated with a reduction in CO, whereas untrained men showed PEH mediated by a reduction in SVR. Similarly, Cote et al. (2015) used high-intensity interval cycling, consisting of 15 bouts of 1 minute at 100% peak power output with 2 minutes of recovery at 50 W, and found that post-exercise MAP reductions were similar regardless of sex or training status. However, post-exercise SBP and the underlying mechanisms did differ, as endurance-trained individuals showed larger reductions, alongside greater depression in baroreflex function and increased sympathetic activity compared with normally active individuals (Cote et al., 2015). Overall, training status may blunt the PEH response in some settings, but the evidence is mixed. Importantly, even when the magnitude of PEH is similar between trained and untrained individuals, the underlying post-exercise cardiovascular mechanisms may differ. The mechanisms behind sustained lower CO in trained participants remain unclear, although increased sensitivity to local vasodilators may play a role, as discussed in Section 2.4.

2.4 Mechanisms of PEH

2.4.1 Autonomic and haemodynamic mechanisms

After exercise, autonomic control shifts, with reduced sympathetic vasoconstrictor activity, a return of parasympathetic activity toward resting levels, and the baroreflex operating at a lower set point (Halliwill, 2001; MacDonald, 2002). The overall result is sustained vasodilation, a fall in SVR and lower arterial pressure (Cunha et al., 2015; Halliwill et al., 1996). Haemodynamically, SVR is often reduced after endurance exercise, but the effect on CO depends on the interaction between training-related cardiovascular adaptations, the orthostatic demand during recovery, and whether venous return is adequately maintained (de Brito et al., 2015; Dujić et al., 2006; Hamer & Boutcher, 2006). For example, CO is often maintained during post-exercise recovery when HR rises sufficiently to counteract the post-exercise fall in SV, particularly during seated or supine recovery, temperate conditions, and when hydration is sufficient to maintain venous return and preload (Fecchio et al., 2021; Halliwill et al., 2014; Lynn et al., 2009). In contrast, CO falls when venous return is compromised, such as during upright recovery due to loss of the muscle pump, heat stress due to high skin blood flow, or hypohydration with subsequent reduced blood volume (Halliwill et al., 2014; Lynn et al., 2009; Watso & Farquhar, 2019). Variations in posture, hydration and environmental conditions may help explain differences in CO and SVR responses reported across studies (Halliwill et al., 2013; Halliwill et al., 2014). Time of day may also play a role, with lower CO values observed in the morning compared with the afternoon (de Brito et al., 2015).

2.4.2 Local vascular mechanisms

Local factors from active muscle and the vascular wall also contribute to post-exercise vasodilation and the PEH response (Halliwill et al., 2013). In response to acute endurance exercise, increased blood flow and shear stress stimulate the release of nitric oxide (NO) from the vascular endothelium (Halliwill, 2001). In normotensive individuals, NO may also remain elevated after acute cycling exercise (Jungersten et al., 1997). Therefore, due to its potent vasodilatory effect, NO may contribute to post-exercise vasodilation and the sustained reduction in SVR. However, blocking NO synthase or prostaglandin pathways alone does not fully abolish PEH, suggesting that multiple overlapping mechanisms modulate vascular resistance following endurance exercise (Halliwill et al., 2000). Histamine release in active skeletal muscle, acting on H1 and H2 receptors, has also been linked to local vasodilation after exercise (McCord & Halliwill, 2006). Overall, these findings indicate that local vasodilatory mechanisms contribute to PEH, but no single local mediator fully explains the response.

Taken together, PEH appears to result from the combined effects of systemic autonomic adjustments and local vascular mechanisms. During prolonged PEH, CO may return toward baseline while SVR remains reduced, allowing arterial pressure to remain lower after exercise (Halliwill, 2001; Kenney & Seals, 1993). The persistence of reduced SVR may explain why PEH can persist beyond the immediate recovery period, with reductions of 3–5 mm Hg in SBP observed for up to 5 hours following high-intensity interval running in normotensive individuals (Dantas et al., 2017). Therefore, the magnitude and duration of PEH likely depend on the interplay between sustained vasodilation and the recovery of CO following exercise. While PEH describes the acute BP reduction following a single exercise bout, repeated bouts

of endurance exercise can produce longer-term reductions in resting BP through structural and functional vascular adaptations (Farrell & Turgeon, 2025). Increased perfusion and shear stress during endurance exercise promote arterial and arteriolar remodelling, including increased vessel diameter and reduced wall thickness, which enhance blood flow capacity (Farrell & Turgeon, 2025; Saltin, 1988). Shear stress also stimulates NO production and promotes vascular endothelial growth factor secretion, supporting angiogenesis and arterial growth (Lloyd et al., 2005). These adaptations help regulate BP over the long term and are clinically relevant for people with hypertension.

2.4.3 Sex differences in PEH mechanisms

Sex may alter the haemodynamic pattern underlying PEH, rather than the overall magnitude of the response. Similar PEH magnitudes have been reported in males and females, although men more often show CO-mediated reductions, with PEH in women mediated by decreases in SVR (Queiroz et al., 2013; Senitko et al., 2002). During recovery from endurance exercise, it has been suggested that women show a reduced ability to vasoconstrict the arteriolar bed compared with men, possibly reflecting reduced sympathetic vasoconstrictor activity and greater parasympathetic cardiac modulation, which may contribute to an SVR-mediated BP reduction (Barnett et al., 1999; Bassareo & Crisafulli, 2020). Other mechanisms have also been proposed, including the effects of ovarian hormones, particularly oestrogen, which may promote vasodilation and reduce afterload (Charkoudian, 2001; Wallin et al., 2010). Together, these factors may help explain the more frequent occurrence of PEH in females (Convertino, 1998; Senitko et al., 2002). The greater frequency of PEH in females is important because females often have a lower resting BP, so post-exercise reductions may result in lower

absolute BP values, potentially increasing the risk of orthostatic intolerance or syncope and highlighting the importance of routine post-exercise BP monitoring. Rossow et al. (2010) found that endurance-trained men and women showed a similar PEH response following high-intensity interval cycling, with both sexes demonstrating increased CO and reduced SVR during recovery. However, following steady-state cycling, the mechanisms differed. Women showed an increase in SV at 60 minutes post-exercise and a greater reduction in SVR, whereas men showed no significant change in SVR, despite reduced vascular resistance in active muscle (Rossow et al., 2010). Overall, while sex does not consistently alter the magnitude of PEH, it appears to shape the underlying haemodynamic pattern.

2.5 Hypohydration and PEH

Hypohydration reduces PV, which reduces venous return, preload and consequently SV via the Frank-Starling mechanism (Magder, 2016). The baroreflex compensates by increasing HR in an attempt to offset the fall in SV, although this compensation is often incomplete and CO remains reduced (Watso & Farquhar, 2019). Under normal conditions, a fall in CO would typically be offset by a compensatory rise in SVR through increased sympathetic vasoconstriction (Halliwill et al., 1996). However, because SVR is already reduced during post-exercise recovery due to sustained vasodilation, this compensatory mechanism is blunted, which may contribute to a larger fall in BP (Jimenez et al., 1999; Watso & Farquhar, 2019). Adequate fluid replacement during exercise helps maintain PV and preload, blunting the reduction in SV during post-exercise recovery, limiting the HR rise and preserving CO, particularly when sweat losses are high (Lynn et al., 2009). However, BP responses following prolonged endurance exercise are not explained by PV loss alone. Holtzhausen and Noakes

(1995) reported a significant fall in SBP after an 80 km footrace in endurance-trained athletes, but suggested this response was multifactorial, reflecting factors such as loss of the skeletal muscle pump aiding venous return and persistent cutaneous vasodilation from thermoregulation. Similarly, Meade et al. (2018) found that even when exercise-induced fluid loss was matched, the haemodynamic pattern of PEH differed between endurance-trained and untrained men. In endurance-trained men, PEH was associated with a reduction in CO, whereas in untrained men it was mediated by a reduction in SVR (Meade et al., 2018). These findings suggest that hydration-related changes cannot be interpreted in isolation, as the post-exercise haemodynamic response is shaped by the interaction between fluid balance, training status and the physiological demands of the exercise performed.

Sweating during prolonged exercise causes water and electrolyte loss, with sweat rate determined by exercise intensity, fitness, clothing and environmental conditions such as heat, wind, humidity and altitude (Maughan, 1991). Because hypohydration is linked to greater PEH and reduced orthostatic tolerance, hydration is essential for safe recovery after exercise. Drinking water during or soon after exercise reduces these risks in healthy adults and can attenuate or even abolish PEH in some protocols, likely by supporting preload (via PV restoration) and normalising cardiac autonomic activity and baroreflex function during early recovery (Lei, Wei, et al., 2025; Tajima et al., 2025). However, much of the available literature has focused on the immediate effect of fluid ingestion before, during or after exercise, with relatively little attention given to the effect of beginning exercise in a hypohydrated state.

These fluid-ingestion studies have often examined the acute pressor response to drinking rather than the hydration status itself. For example, Tajima et al. (2025) examined the

immediate effect of post-exercise water ingestion on orthostatic hypotension, while others have examined BP responses in heat stress (Kajiki et al., 2024; Kay et al., 2005). In similar conditions, Lynn et al. (2009) showed that fluid replacement to match sweat loss mitigated the post-exercise decrease in CO, although this was examined in highly trained endurance participants. These findings involving heat stress are difficult to apply to temperate conditions, as heat stress confounds the mechanisms underlying PEH, with typically amplified responses due to higher skin blood flow, core temperature (T_{core}), and larger sweat-induced PV losses that sustain peripheral vasodilation (Cunha et al., 2020; Horiuchi & Oliver, 2023). Among studies in temperate conditions, Mendonca and Fernhall (2016) examined the effect of 50ml and 500ml of water ingested immediately before a cycling ramp protocol to volitional exhaustion in healthy adults. They found similar magnitudes of PEH between conditions during 30 minutes of seated recovery followed by 5 minutes of standing recovery. However, participants were hydrated before ingesting water and body mass was similar before and after exercise, as the study focused on the pressor response to water ingestion rather than the effect of hypohydration. Endo et al. (2012) administered water during cycling exercise compared with water restriction and found that fluid replacement equivalent to exercise-induced body mass loss prevented PEH, indicating fluid intake during exercise can meaningfully alter the post-exercise BP response. However, pre-exercise hydration status was not measured, and the PEH response was small following the relatively low exercise intensity used (60% HRR) and supine recovery. Overall, these studies show that fluid intake can alter the PEH response, but there is limited research on whether starting exercise in a hypohydrated state alters the magnitude or duration of PEH.

Most studies focusing on PEH are performed under typical laboratory conditions where hydration status is assumed to be euhydrated, but not always directly assessed (Forjaz et al., 2004; Forjaz et al., 1998; MacDonald et al., 2000; Memon et al., 2011; Senitko et al., 2002). The distinction matters because athletes and recreationally active adults may begin exercise hypohydrated. Hypohydration at the start of exercise can arise through more than one pathway, including fluid restriction, inadequate replacement of fluid losses during previous exercise, or diuretic use (James et al., 2019). In research settings, fluid restriction is often used to induce pre-exercise hypohydration because it reduces body water before exercise without the additional physiological effects of prior exercise, heat stress, or diuretic use. Volpe et al. (2009) assessed hydration status through spontaneous spot urine tests before sport practice in 263 Division 1 collegiate athletes and found that 66% were hypohydrated, indicated by a urine specific gravity $\geq$ 1.020. Similarly, Chapelle et al. (2020) reported that 55% of recreational players and over 60% of elite players were hypohydrated before training or game scenarios. These findings indicate that pre-exercise hypohydration is common among athletic populations, highlighting the importance of understanding how hydration status shapes cardiovascular responses during and after exercise.

Beginning exercise hypohydrated means PV is already reduced from the outset, increasing cardiovascular strain during exercise and potentially altering the subsequent post-exercise BP response. Pre-exercise fluid restriction reduces PV more than exercise-induced hypohydration (Barr, 1999), and this lower PV increases blood viscosity and shear stress, contributing to greater cardiovascular drift during exercise (Heaps et al., 1994). The added cardiovascular strain is evident when exercise is performed at a fixed workload. In recreational athletes, hypohydration induced through 12 h of fluid restriction reduced

workload and oxygen uptake during cardiopulmonary exercise testing with a cycle ramp protocol, reflecting a lower CO driven by reduced preload (Strüven et al., 2023). Consistent with this, Aldridge et al. (2005) found that in moderately trained rugby players, HR, RPE and relative $\dot{V}O_2$ were all significantly higher during cycling exercise at a fixed intensity of 75 watts when participants were hypohydrated through fluid restriction than when they were euhydrated. The same absolute workload therefore represents a higher relative exercise intensity when hypohydrated, which Aldridge et al. (2005) proposed increases the likelihood of premature fatigue. Interestingly, RPE during the first 5 minutes of exercise was similar between hypohydrated and euhydrated conditions, with differences emerging only as exercise continued (Aldridge et al., 2005). A hypohydrated state may therefore go undetected in the opening stages of an exercise session, with perceived effort rising only once the physiological demands become greater. Starting exercise in a hypohydrated state consequently matters, as the reduced pre-exercise PV may alter the post-exercise BP response differently from hypohydration that develops gradually during exercise.

Only one known study has directly examined whether beginning exercise hypohydrated alters the PEH response, manipulating hydration status before cycling and comparing rehydration strategies during recovery. Lei, Wei, et al. (2025) examined the PEH response in recreationally active, normotensive men who completed severe-intensity cycling to volitional exhaustion, followed by 1 hour of seated recovery, under four hydration conditions. These were euhydration, hypohydration with no fluid intake during recovery, hypohydration with fixed fluid intake during recovery, and hypohydration with *ad libitum* fluid intake during recovery, with hypohydration induced through fluid restriction before exercise. The hypohydration condition without rehydration produced the largest post-exercise BP reductions from

baseline, with MAP, SBP and DBP remaining lower across the whole 60-minute recovery period. A peak reduction in DBP of 12 mm Hg was shown in this condition, compared with 6 mm Hg in euhydration. The authors proposed that reduced blood volume and lower baroreflex sensitivity contributed to the exaggerated PEH response. When hypohydrated participants rehydrated after exercise, either with a prescribed or *ad libitum* strategy, the additional PEH caused by pre-exercise hypohydration was abolished (Lei, Wei, et al., 2025). Post-exercise fluid restoration therefore appears to offset the exaggerated BP fall caused by pre-exercise hypohydration.

The work of Lei, Wei, et al. (2025) is highly relevant to the present study, as it examined recreationally active, normotensive males starting exercise in a hypohydrated state from fluid restriction and exercising in a temperate laboratory environment. However, participants completed short-duration, severe-intensity cycling to volitional exhaustion, with subsequent trials matched in duration to one of the hypohydration trials completed first, rather than each condition being cycled to exhaustion independently. Consequently, the findings may not translate directly to longer-duration endurance exercise performed at a fixed submaximal intensity. Lei, Wei, et al. (2025) reported similar post-exercise HR responses across conditions but did not report on HR during exercise, making it difficult to determine whether hypohydration altered the physiological responses during cycling, which could in turn have affected the subsequent PEH response. A clear evidence gap therefore remains, as few studies have isolated hypohydration at the start of endurance exercise in the absence of heat stress and without in-exercise or post-exercise water ingestion. Pre-exercise hypohydration is common, as athletes and recreationally active adults often begin exercise hypohydrated (Maughan & Shirreffs, 2010) and may not rehydrate during activity. Prolonged team sports,

long-distance rowing events and exercise while fasting during Ramadan are examples where athletes may be particularly vulnerable to the compounding effects of pre-exercise hypohydration with limited rehydration options. Therefore, the present study addresses this gap by examining whether starting exercise whilst hypohydrated alters the magnitude and duration of PEH during standing recovery following 30 minutes of fixed-intensity cycling.

Chapter Three: Methods

3.1 Ethics and informed consent

All participants were informed of the study procedures, aims, and risks of participation prior to providing written informed consent. This study was conducted in accordance with the latest revision of the Declaration of Helsinki and was approved by the Central Health and Disability Ethics Committee (2025 EXP 24123) and registered in a clinical trials database (Australia New Zealand Clinical Trials Registry, ACTRN12625001253493).

3.2 Participants

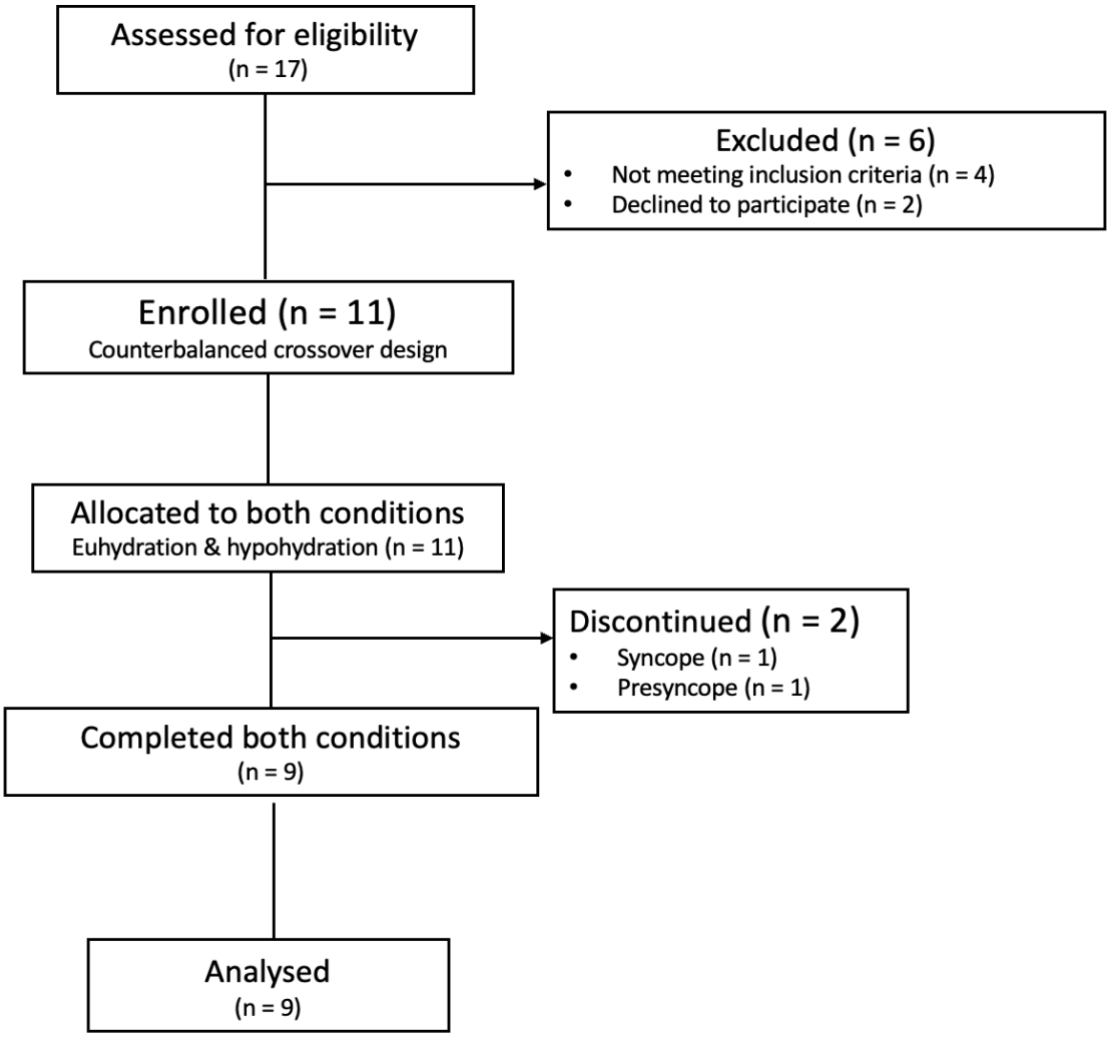


Figure 1. Participant recruitment overview. Consort diagram based off framework by Hupalo et al. (2023).

An a priori power analysis (G*Power version 3.1.9.4; Heinrich Heine University Düsseldorf, Düsseldorf, Germany) was conducted using data from Endo et al. (2012), with the same primary dependent variable as the current study of post-exercise reduction in MAP, resulting in an effect size of 0.51. Based on conventional α (0.05) and β (0.80) values, a minimum of 6 participants were required. To account for drop-out, 11 participants were recruited, with 2 being discontinued (Figure 1). Participant characteristics, including age, height, body mass, resting BP, resting HR and functional threshold power (FTP) are reported in Table 1. All

participants were healthy, had no history of cardiovascular, cerebrovascular, pulmonary, metabolic or renal disease, were non-smokers and were not taking any medication that might influence BP, hydration status or HR. Female participants self-reported their pregnancy status and menstrual cycle phase, with all visits occurring in the mid-luteal phase.

Participants were all considered recreationally active, but not elite or professionally trained athletes, defined as over a typical week in the past 6 weeks, performing at least 50% of the lower limit of the WHO physical activity guidelines (World Health Organisation, 2020) but not exceeding the upper limit (acceptable range of moderate 75-300 min/week or vigorous 37.5-150 min/week exercise), measured via the Global Physical Activity Questionnaire (GPAQ, see Appendix E). Participants were instructed to maintain their usual exercise habits throughout the study period and to refrain from making substantial changes to their training volume or intensity between visits.

3.3 Study design

Each participant visited the laboratory on three occasions, with all visits completed at the same time of day (±1 hour), starting no later than 9:00am. The first visit was the familiarisation visit, which included an FTP cycling test (see Appendix G). The FTP protocol consisted of a warm-up, followed by a ramp-up test in which resistance increased incrementally until participants reported an RPE of 10, reflecting the maximal effort sustainable for 5 minutes. After a period of active recovery, participants completed a 5-minute FTP test at the resistance reached during the ramp. This visit was designed to familiarise the participant with all experimental procedures, risks and benefits, determine eligibility, obtain informed consent, and establish individual exercise workloads for subsequent experimental visits. Pilot testing

was conducted prior to the start of data collection and indicated that cycling at 80% of FTP was a tolerable exercise intensity for the target population and elicited a robust PEH. All cycling exercise was completed on a stationary IC6 indoor cycle (Life Fitness, USA). During cycling exercise, heart rate was recorded (Polar Electro, Finland) and RPE was measured using a modified Borg CR10 RPE scale. Visits 2 and 3 involved identical procedures, with the only difference being initial hydration status as described below. All visits were separated by ≥5 days.

3.4 Experimental design

Participants arrived at the laboratory having abstained from strenuous exercise and alcohol for 12 hours, and caffeinated beverages for 4 hours before testing. Before visit 2, participants were asked to record their diet and timing of food intake for 12 hours prior to coming into the laboratory. Additionally, participants were asked to avoid meals that are high in sodium and a list of foods with vasoactive components (e.g. nitrates) was provided and participants were asked to avoid as listed on the diet recall sheet (see Appendix F). Adherence to these diet requirements was self-reported and the diet recall sheet was collected at the last visit. To assess hydration status, upon arrival participants were asked to provide a urine sample for assessment of urine specific gravity (USG). Visits 2 and 3 were completed in a counterbalanced order and were defined as hypohydration (USG ≥1.020) and euhydration (USG ≤1.020). Water intake was restricted following provision of the USG sample in both visits. Water was then consumed *ad libitum* once the final blood pressure was recorded (30 minutes post exercise).

3.5 Hypohydration visit

Participants were instructed to consume a bolus of water up to 1L, 18 hours before the visit. After this, participants were asked to refrain from consuming water or other liquids (e.g. coffee, milk, tea, energy drinks, soups/broths) for the next 18 hours prior to visiting the laboratory. The target USG for this visit was ≥1.020. If the USG of the sample provided upon arrival to the laboratory was <1.018, the visit was rescheduled to the earliest convenience to the participant. If the USG was <1.020 but >1.018, another urine sample was obtained after 30 minutes. If USG remained <1.020, the visit was rescheduled. The total number of rescheduled visits across visits 2 and 3 due to USG outside the target range was capped at 2 per participant. Water consumption was then restricted until the visit was complete (e.g. during exercise and the post exercise standing period).

3.6 Euhydration visit

For the euhydration visit, participants were instructed to maintain euhydration during the 12h prior to exercise. They were asked to keep their normal hydration habits while ensuring they consumed a minimum of 500ml the night before the visit and the same volume again on the morning of the visit. Once the participant provided a urine sample, water consumption was restricted until the visit was complete. If the USG was >1.020 but <1.023, and if the participant consented, they were asked to consume another 500ml of water and another urine sample was obtained after 30 minutes. If the USG returned was still >1.020, then the visit was rescheduled. If the initial USG returns a value of >1.023, then the visit was rescheduled without obtaining another sample.

3.7 Exercise protocol

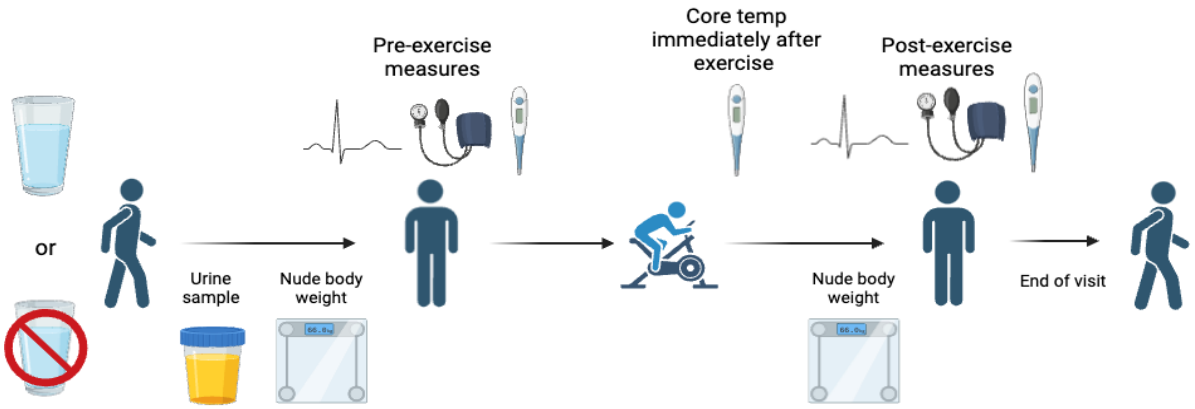


Figure 2. Experimental protocol overview for visits 2 and 3. Pre- and post-exercise measures include HR, BP and core temperature. Warm-up was 50% of FTP for 5 minutes and cool-down was 40% of FTP for 3 minutes. Image created with BioRender.com.

Participants arrived at the temperature-controlled laboratory, first provided a urine sample and measured their nude body weight in a private bathroom. Following this, participants were seated for electrocardiogram (ECG) instrumentation. After 5 minutes of seated and quiet rest, resting seated BP was recorded. Participants then stood up for 8 minutes of HR recording, and then standing BP was recorded. Tympanic T_{core} was recorded after 10 minutes of standing. The ECG leads were removed and the participant donned a heart rate monitor for measurement of HR during exercise. Participants completed a 5-minute warm up at 50% of their estimated FTP. Following this a 30-minute fixed intensity cycling bout was completed at 80% of the participant's FTP with power output and HR recorded every 2 minutes and RPE every 5 minutes. Finally, a 3-minute cool-down was done at 40% of the participants FTP. Post-exercise, T_{core} was measured immediately, the participant dried themselves, removed the HR monitor and nude body weight was obtained. The ECG was then refitted and the participant

stood for 30 minutes of HR measurements, with BP and T_{core} measured every 5 minutes. The post exercise period began when the participant dismounted the ergometer.

3.8 Measurements

3.8.1 Cardiovascular

BP was measured using an automated sphygmomanometer (Suresigns, VM4, Philips Medical Systems, Best, The Netherlands). For baseline seated blood pressures participants rested in a quiet room and were instructed to have their feet flat on the floor with their palm facing up and to refrain from talking. When standing, participants were asked to remain as still as possible. Pre-exercise resting seated BP was measured in triplicate following 5 minutes of seated rest, followed by resting standing BP measured in duplicate after 8 minutes of standing rest. If two consecutive SBP readings differed by >5 mm Hg, an additional measure was taken. During recovery, standing BP was measured every 5 minutes for 30 minutes. The participants were reminded about the signs and symptoms of syncope.

A safety threshold of an SBP below 100 mm Hg was used as a termination criterion for the present study, applied both to the average pre-exercise standing BP and to any timepoint during the post-exercise standing recovery period. When this threshold was exceeded, or when the participant developed any symptoms associated with fainting, including light-headedness, nausea, blurry vision, weakness, or shortness of breath, the visit was terminated and the participant was assisted to a seated position, if this occurred, the visit was not repeated.

HR was measured pre- and post-exercise using a 3-lead ECG (lead II) at rest and using a Polar HR monitor during exercise (Polar Electro, T31-coded transmitter, model N2 965, Kempele, Finland). During the fixed-intensity cycling bout, HR was recorded every 2 minutes.

3.8.2 Core temperature

As T_{core} can influence post-exercise vascular compliance, and may therefore affect the magnitude of PEH, T_{core} was measured to gain insight to this potential mechanism. T_{core} was measured using a tympanic infrared thermometer (Microlife Corporation, model IR 1DE1, Berneck, Switzerland). T_{core} was measured after 10 minutes of standing pre-exercise, immediately post-exercise, and every 5 minutes during the 30-minute recovery period.

3.8.3 Anthropometric

Body height was measured using a stadiometer and body mass was measured using electronic scales. Nude body mass was measured before and immediately after exercise to determine exercise-induced body mass loss. Percentage of body mass loss was calculated from the change in pre- to post-exercise nude body mass.

3.8.4 USG

USG was measured using a hand-held electronic refractometer (Atago Co., Ltd, Tokyo, Japan). It was assessed from a urine sample provided on arrival to the laboratory and used to confirm hydration status prior to each experimental visit.

3.8.5 Rating of perceived exertion

RPE was measured using a modified Borg CR10 RPE scale, where 0 represents no exertion and 10 represents maximal effort. RPE was recorded every 5 minutes during the 30-minute fixed-intensity cycling bout.

3.9 Data acquisition

All data were collected through Labchart (ADInstruments, Bella Vista, NSW, Australia) and saved in an Excel spreadsheet.

3.10 Calculations

MAP was calculated as per MAP = DBP + (SBP – DBP)/3.

3.11 Statistical analysis

All data were analysed using SPSS statistical software v.28 (IBM Corp., Armonk, NY, USA) and are presented as mean ± SD. Statistical significance was set *a priori* at $P < 0.05$. HR at baseline and during exercise were analysed by a two-way repeated measures analysis of variance (ANOVA, time x condition, 16 x 2 – where time refers to baseline prior to exercise and the measurement every 2 minutes from exercise onset to termination). Power output during exercise were analysed by a two-way repeated measures analysis of variance (ANOVA, time x condition, 15 x 2 – where time refers to the measurement every 2 minutes from exercise onset to termination). Pre and post T_{core}, nude body weight, heart rate (visits 2 and 3) were

analysed by a two-way repeated measures analysis of variance (ANOVA, time x condition, 2 x 2 – where time refers to the pre- and post-exercise timepoints). RPE during exercise were analysed by a two-way repeated measures analysis of variance (ANOVA, time x condition, 6 x 2 – where time refers to the measurement made every 5 minutes from exercise onset until termination). Absolute SBP, DBP, and MAP pre- and post-exercise (either hypohydration or euhydration) were analysed by two-way repeated measures analysis of variance (ANOVA, time x condition, 7 x 2), where time refers to pre- (baseline) and post-exercise measurements (6 timepoints post-exercise recovery at 5-minute intervals for 30 minutes total), and condition refers to the hypohydration and euhydration visits. Absolute changes in SBP, DBP, and MAP from pre-exercise baseline were analysed by two-way repeated measures of analysis of variance (ANOVA, time x conditions, 6 x 2). HR during recovery were analysed by a two-way repeated measures analysis of variance (ANOVA, time x condition, 7 x 2 – where time refers to the final measurement at end of exercise and every 5 minutes thereafter during standing recovery for 30 minutes). Post-hoc pairwise comparisons were used to isolate interaction effects. Effect sizes were estimated using partial eta squared (η^2) and are reported for the two-way ANOVA main and interaction effects, with large effect sizes defined as >0.14, medium 0.06-0.14, and small <0.06 (Cohen, 2013). Average HR during exercise, pre-exercise USG, change in body weight from pre- to post-exercise, maximum reduction (nadir) in SBP, DBP, and MAP and change from baseline (e.g. ΔSBP) during post-exercise standing recovery, and mean power output during the fixed-intensity exercise were compared via paired t-test. FTP was determined in visit 1 and is reported in Table 1 alongside participants physical characteristics and baseline blood pressure data.

Chapter Four: Results

Table 1. Participant physical characteristics, resting cardiovascular variables, and cycling power outputs	
Sex (Male/Female)	7/2
Age (years)	27 ± 3
Height (cm)	183 ± 12
Weight (kg)	81.2 ± 12.5
Resting SBP (mm Hg)	115 ± 5
Resting DBP (mm Hg)	74 ± 8
Resting MAP (mm Hg)	88 ± 7
Resting HR (beats·min^{-1})	64 ± 11
FTP (W)	197 ± 52
80% FTP (W)	157 ± 41
Moderate PA (min/week)	110 ± 88
Vigorous PA (min/week)	89 ± 41

SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial blood pressure; HR = heart rate; FTP = functional threshold power; PA = physical activity. (n = 9, 2 female)

4.1 Pre-exercise measures

Table 1 shows the participant characteristics, mean resting haemodynamics, FTP, and the power output during the steady state cycling exercise (80% of FTP). By design, participants' self-reported moderate and vigorous physical activity (Table 1) fell within the WHO physical activity guidelines (see Section 3.2) (World Health Organisation, 2020). All nine participants completed both exercise bouts. For exercise variables and analyses of change from baseline to nadir $n = 9$. By design, USG was greater in the hypohydration trial compared to euhydration (1.023 ± 0.002 vs. 1.009 ± 0.007, $P = 0.004$). One participant did not reach the required USG

threshold for the hypohydration condition and was rescheduled with hypohydration achieved on the subsequent visit. Analysis of body mass revealed a significant main effect of time (P = 0.002, η^2 = 0.719) and hydration (P = 0.050, η^2 = 0.393), but no time x hydration interaction effect (P = 0.117, η^2 = 0.278). Body mass was 81.1 $\pm$ 12.4 kg in the euhydration condition and 80.2 $\pm$ 11.9 kg in the hypohydration condition prior to exercise, and 80.8 $\pm$ 12.4 and 79.8 $\pm$ 11.8 kg post-exercise, respectively. Body mass fell by 0.3 $\pm$ 0.4 kg across the euhydration visit and 0.4 $\pm$ 0.2 kg across the hypohydration visit, with no difference in the magnitude of change between conditions (P = 0.117).

4.2 Power

Power output was matched across conditions. There was no effect of time (P = 0.220, η^2 = 0.138), hydration (P = 0.529, η^2 = 0.051) or a time x hydration interaction (P = 0.920, η^2 = 0.103). Mean power across the 30 minutes was 159 $\pm$ 41 W for euhydration and 159 $\pm$ 42 W for hypohydration, with no difference between conditions (P = 0.529).

4.3 Core temperature

Tympanic T_{core} increased from pre-exercise to immediately post-exercise and declined across recovery in both conditions (n = 7, main effect of time, P < 0.001, η^2 = 0.477). There was no effect of hydration (P = 0.671, η^2 = 0.032) and no time x hydration interaction (P = 0.236, η^2 = 0.188). T_{core} was greatest immediately post-exercise in both the hypohydration condition (35.8 $\pm$ 0.7 °C) and the euhydration condition (35.6 $\pm$ 0.6 °C). After 30 minutes of recovery, T_{core} was equivalent between conditions (34.9 $\pm$ 1.1 °C).

4.4 Rating of perceived exertion

RPE increased over time in both conditions and was higher with hypohydration (see Figure 3 for *P* values). Post-hoc testing identified differences between conditions at minute 25, where RPE was 6 ± 2 for euhydration and 7 ± 1 for hypohydration (*P* = 0.009), and at minute 30, where RPE was 6 ± 2 for euhydration and 8 ± 1 for hypohydration (*P* < 0.001).

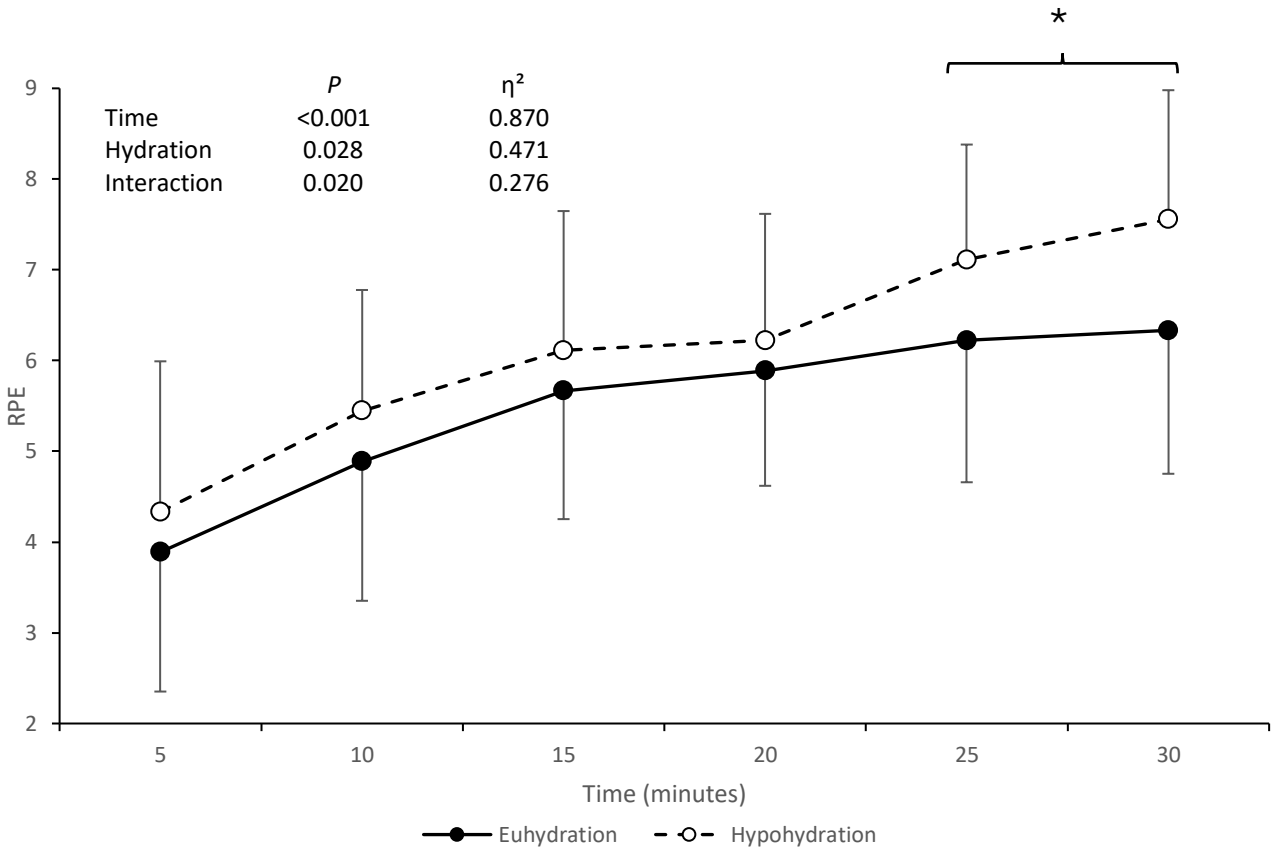


Figure 3. Rating of perceived exertion (RPE) during exercise in the euhydration and hypohydration conditions (n = 9). Data are mean ± SD. * indicates a difference between conditions at minutes 25 and 30 (P ≤ 0.009).

4.5 Heart rate

During exercise, HR was higher with hypohydration (main effects of time and hydration and a time x hydration interaction – see Figure 4 for *P* values), with post-hoc tests revealing difference from baseline and all time points except minutes 8 (*P* = 0.058) and 22 (*P* = 0.074). The higher HR with hypohydration persisted at every time point throughout recovery, where main effects of time and hydration were present, but no time x hydration interaction, so the difference between conditions was consistent across the recovery period (see Figure 5 for *P* values).

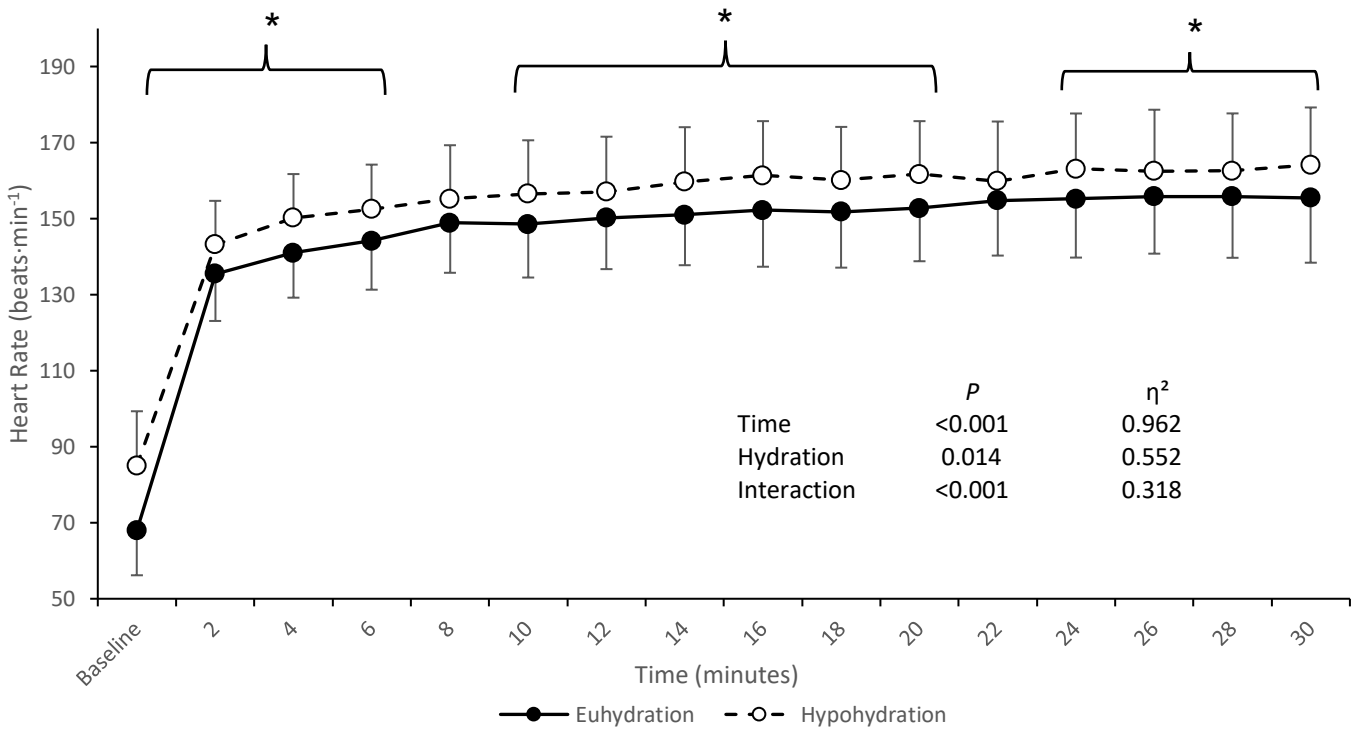


Figure 4. Heart rate during exercise (n = 9). Data are mean ± SD. * indicates a difference between conditions (P ≤ 0.05).

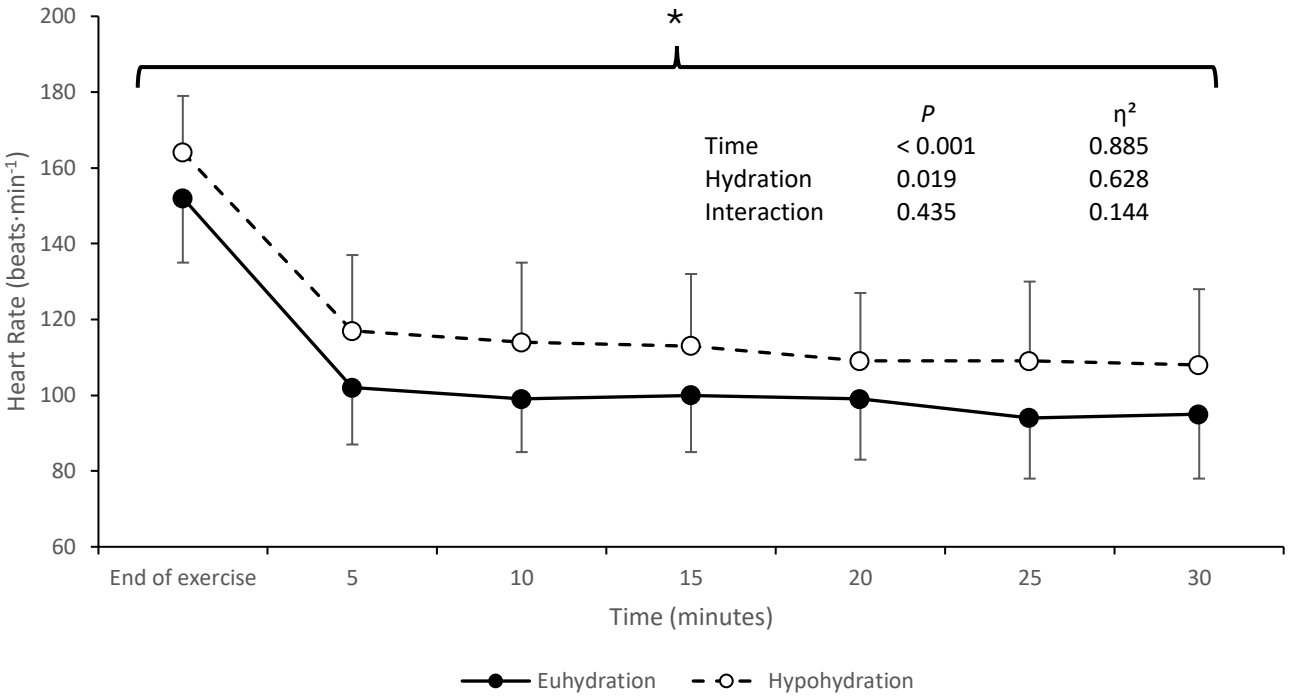


Figure 5. Heart rate during recovery (n = 7). Data are mean ± SD. * indicates a difference between conditions (P < 0.05). End of exercise denotes HR at the final minute of the 30-minute exercise bout.

4.6 Blood pressure

Absolute blood pressures following exercise are shown in Figure 6. Briefly, there was no difference in SBP or DBP between conditions during the recovery period following exercise. However, the effect sizes for hydration on SBP were large, suggesting the analysis was underpowered to detect a difference (see Figure 6 for *P* values). For MAP, a time x hydration interaction with no main effect of time or hydration, and post-hoc testing located the difference between conditions at minute 25 (*P* = 0.010), where MAP was 93 ± 3 mm Hg for euhydration and 88 ± 3 mm Hg for hypohydration (Figure 6).

The maximum reduction in SBP post-exercise (i.e. ΔSBP nadir) was greater following exercise in the hypohydration condition (-12 ± 3 mm Hg) than euhydration (-6 ± 5 mm Hg) (*P* = 0.002,

Figure 7). No difference between conditions was found for ΔDBP (-8 ± 6 vs -8 ± 6 mm Hg, *P* = 0.965) or ΔMAP (-8 ± 3 vs -5 ± 5 mm Hg, *P* = 0.138). When ΔSBP and ΔMAP were analysed at each recovery time point, there was a significant main effect of hydration for both ΔSBP and ΔMAP, with a greater reduction from baseline in the hypohydration condition (Figure 8). However, absolute nadir values did not differ between condition for SBP (112 ± 9 vs 106 ± 6 mm Hg, *P* = 0.068), DBP (71 ± 7 vs 71 ± 6 mm Hg, *P* = 0.962) or MAP (86 ± 7 vs 84 ± 4 mm Hg, *P* = 0.358). Time to nadir did not differ between conditions for SBP (16 ± 10 vs 13 ± 8 min, *P* = 0.482), DBP (14 ± 9 vs 13 ± 8 min, *P* = 0.865) or MAP (15 ± 10 vs 16 ± 7 min, *P* = 0.746).

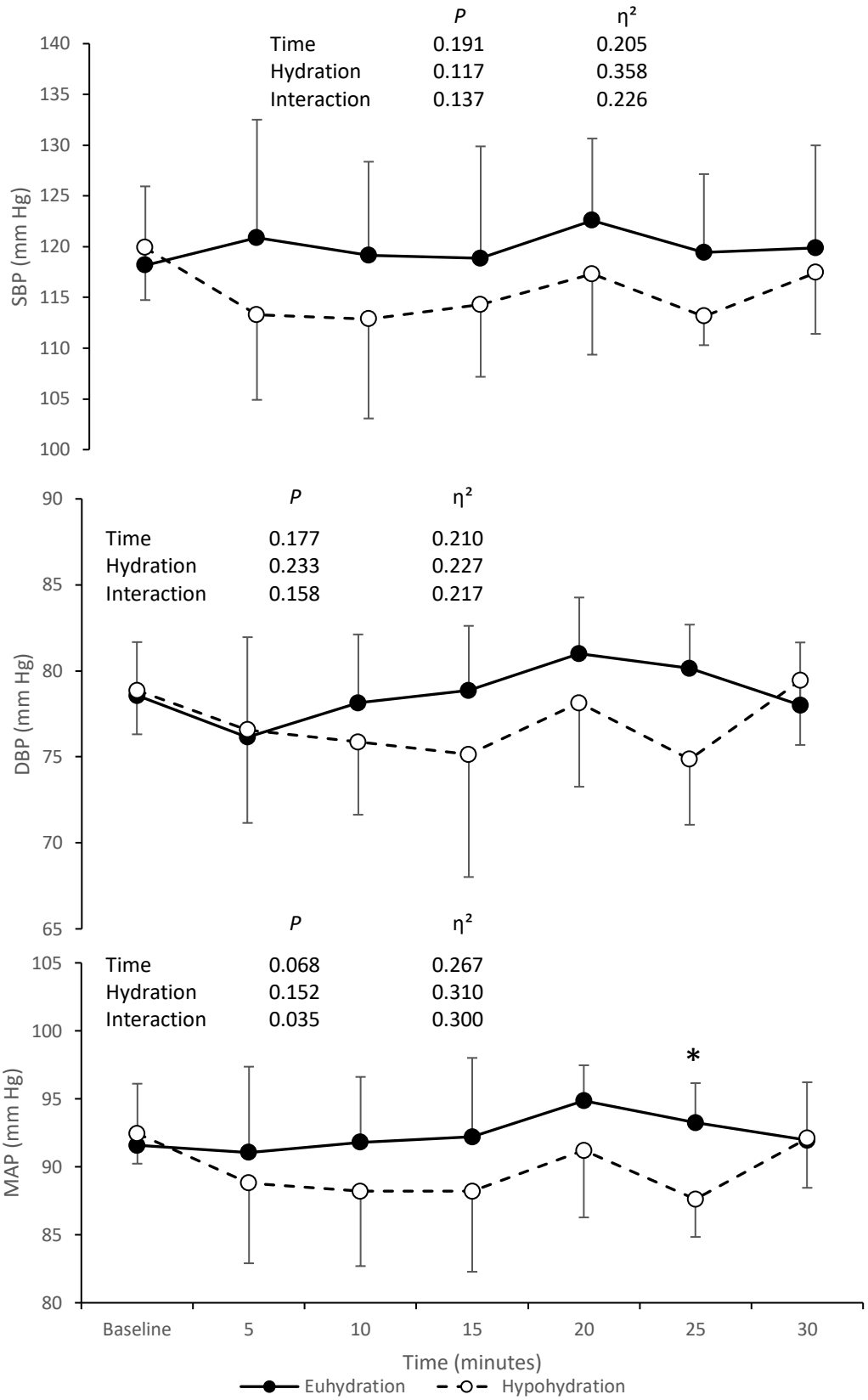


Figure 6. Systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) during recovery from cycling exercise (n =7). Data are mean ± SD. * indicates a difference between conditions ($P < 0.05$).

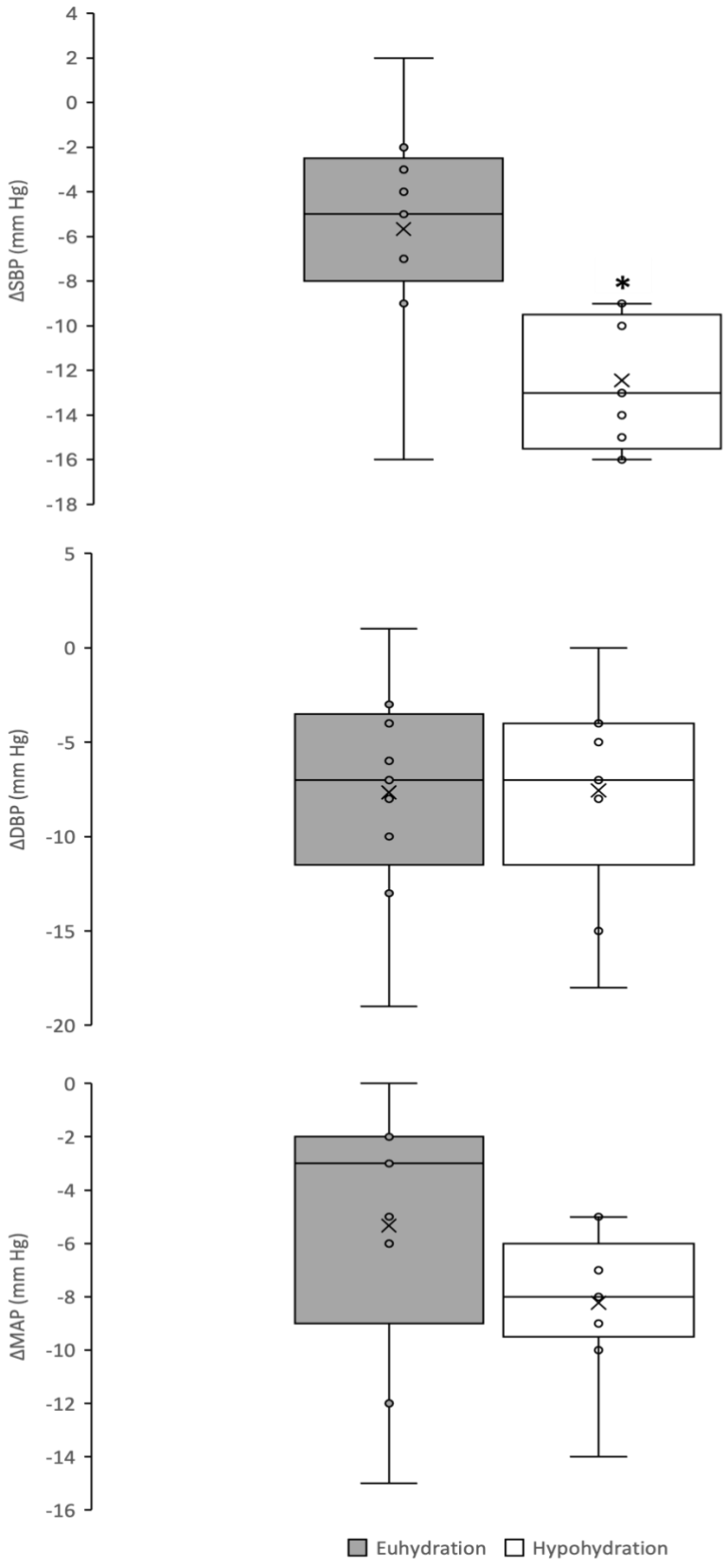


Figure 7. Change from baseline to nadir for systolic blood pressure (ΔSBP), diastolic blood pressure (ΔDBP) and mean arterial pressure (ΔMAP) (n = 9). * indicates a significant difference between conditions ($P < 0.05$).

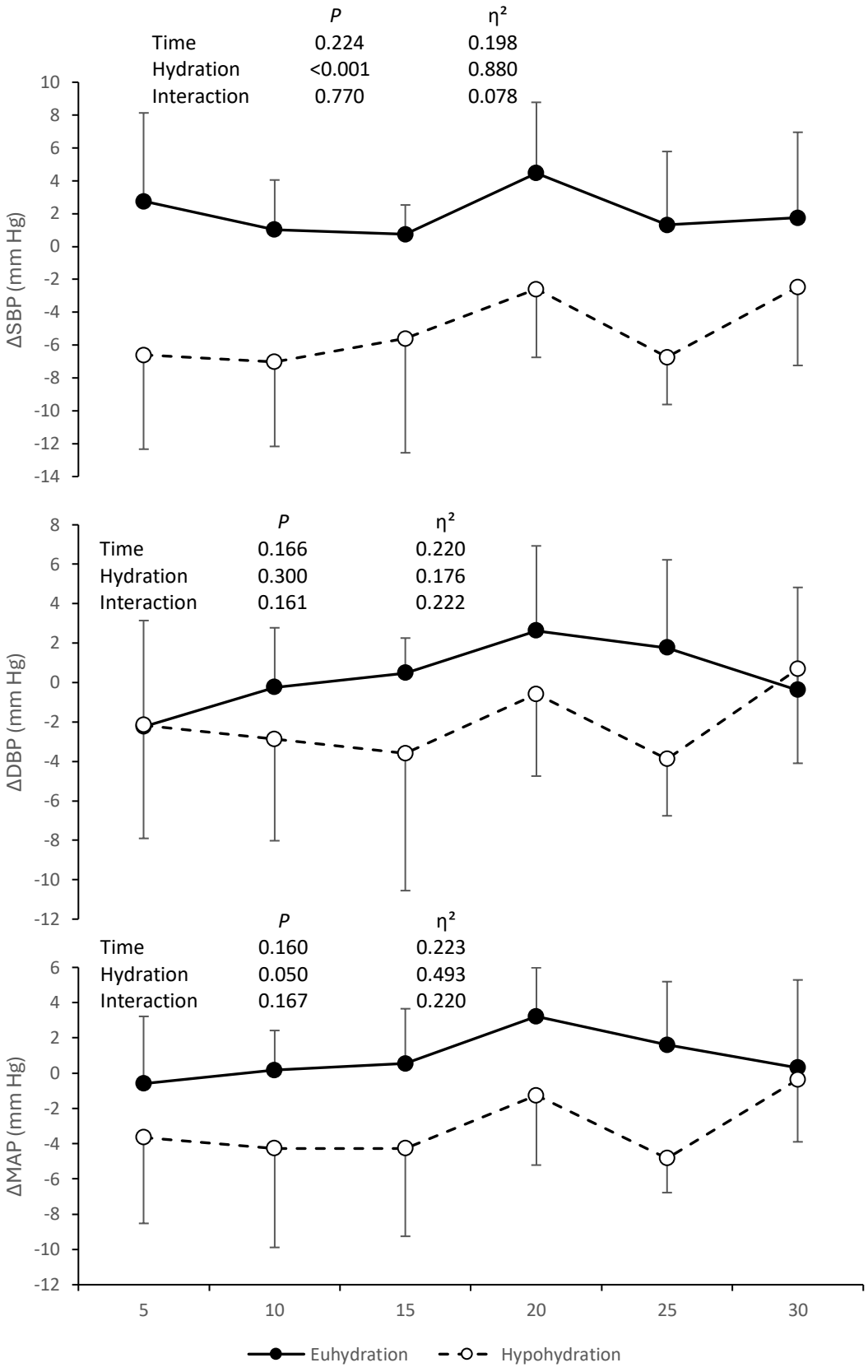


Figure 8. Absolute change from baseline at each recovery time point for systolic blood pressure (ΔSBP), diastolic blood pressure (ΔDBP) and mean arterial pressure (ΔMAP) (n = 7). Data are mean ± SD.

4.7 Safety/Adverse events

Four participants experienced adverse events during the study, each associated with an SBP below the pre-determined safety threshold of 100 mm Hg, at which point the visit was terminated. Three occurred in the hypohydration condition and one in the euhydration condition. During the hypohydration condition, one participant experienced syncope after the 5 minute time point post-exercise, and a second reported pre-syncopal symptoms during the pre-exercise standing period and did not begin the exercise bout. Both were unable to complete the visit and were excluded from all analyses. A third participant during the hypohydration condition, and one during the euhydration condition, reported pre-syncopal symptoms and an SBP of under 100 mm Hg at minute 15 of recovery and were asked to sit down. These two participants completed the exercise bout for both conditions and are included in the during-exercise analyses and the change from baseline to nadir (n = 9, Figure 7), but not in the analyses across the recovery time points (n = 7). This accounts for the difference in sample size as seen in the figure captions and as detailed in the CONSORT diagram (Figure 1).

Chapter Five: Discussion

The aim of this study was to determine the effect of hydration status on the PEH responses to moderate intensity cycling exercise in healthy, normotensive, recreationally active adults. It was hypothesised that reductions in BP post-exercise would be greater in the hypohydration condition compared with euhydration. The main finding of this study was that when BP was analysed as the absolute change from baseline (pre-exercise): 1) greater nadir reductions (i.e. greatest reduction) in SBP were observed in the hypohydration condition and 2) the analysis of BP across the entire recovery period (30 minutes) revealed a significant main effect of hydration for both ΔSBP and ΔMAP, with larger reductions in the hypohydration condition (Figure 8). The time taken to reach nadir blood pressures did not differ between conditions, indicating hypohydration altered the magnitude of the SBP response but not its timing. Despite power output during the cycling bouts being matched, the physiological strain was greater in the hypohydration condition, with a higher HR during exercise and throughout recovery, and higher RPE at minutes 25 and 30 of the exercise bout. Collectively, this partially supported our hypothesis, as reductions in BP were greater following hypohydration, with the largest effect on SBP.

5.1 Hydration status

Hypohydration was successfully achieved through fluid restriction, as indicated by the USG values in the results. In the hypohydration condition, USG was above the threshold of 1.020 commonly used to classify hypohydration (Volpe et al., 2009), and comfortably below this threshold in the euhydration condition. Consistent with Lei, Wei, et al. (2025), body mass in

the hypohydration condition was lower at baseline and after exercise, but there was no difference between conditions from pre- to post-exercise. Fluid restriction was used to induce hypohydration because it reduces body water before exercise without the additional physiological effects of prior exercise, heat stress or diuretic use (James et al., 2019). This allowed hydration status to be isolated, with participants starting exercise hypohydrated, which can commonly occur (Volpe et al., 2009). If additional stress such as heat or prior exercise were superimposed on this, the risk of adverse effects may increase. Resting HR was approximately 17 beats·min^{-1} higher at baseline in the hypohydration condition, consistent with reduced PV and a compensatory rise in HR to defend CO (Watso & Farquhar, 2019).

5.2 Physiological responses during exercise

As per the study design, power output was matched between conditions, with no effect of hydration on the power output at any timepoint, or mean power across the bout. T_{core} rose from pre-exercise to immediately post-exercise and declined across recovery in both conditions, with no effect of hydration. Participants therefore completed the same work under the same thermal conditions in both visits. The absence of a difference in T_{core} is expected as the environment was temperate, and it allows the findings to be attributed to hydration status alone rather than to hydration combined with additional heat stress. This distinction is important because much of what is known about hydration and cardiovascular physiology during exercise comes from heat stress conditions, where the impairment in cardiovascular function depends on the combination of hypohydration and hyperthermia rather than hypohydration alone (González-Alonso et al., 1997). By using a temperate environment, the present study examined hypohydration in isolation, meaning the

differences observed in the post-exercise BP response can be attributed to hydration status without a confounding thermal component.

Despite the matched workload, HR was higher with hypohydration at almost every time point during exercise with an average difference of ~8 beats·min^{-1} (Figure 4). Our findings are in agreement with those of Aldridge et al. (2005) who reported elevated HR throughout 30 minutes of fixed-intensity cycling when hypohydrated, reflecting the added cardiovascular strain of exercising in this state, although they confirmed hydration status using urine osmolality rather than USG. Reduced PV lowers venous return and preload, decreasing SV via the Frank-Starling mechanism, with a compensatory rise in HR to defend CO (Magder, 2016; Watso & Farquhar, 2019). The absence of any difference in power output indicates the fixed workload was achievable and the rise in HR was sufficient to maintain CO and muscle perfusion, sustaining the required work during exercise. Aldridge et al. (2005) proposed that the added strain of exercising hypohydrated increases the likelihood of premature fatigue. No difference in work completed was observed in the present study, although the fixed intensity was achievable for both conditions and the exercise bout was not continued to exhaustion, so time to fatigue was not tested. The associated increase in cardiovascular demand likely underpins the increase in RPE seen during exercise in the hypohydration condition. It is interesting to note, however, that the difference in RPE was not present from the outset and only diverged between conditions at minutes 25 and 30 (Figure 3). Again, our findings are supported by Aldridge et al. (2005), who reported that RPE during the first 5 minutes of exercise was similar between hypohydration and euhydration conditions with differences occurring only as exercise continued. Thus, the strain of starting exercise hypohydrated was not perceived until the bout was well underway, despite HR being elevated

from baseline and throughout exercise. Athletes who begin exercise hypohydrated, which is common (Chapelle et al., 2020; Hew-Butler et al., 2018; Volpe et al., 2009), may not experience an increased RPE initially, but may notice the added effort as exercise progresses. Taken together, the responses during exercise indicate that hypohydration increased cardiovascular strain at a fixed workload without altering the work completed. The compensatory rise in HR was adequate to sustain the exercise bout, consistent with HR being sufficient to maintain cardiovascular function when hypohydration occurs without hyperthermia (González-Alonso et al., 1997). The elevated HR persisted into the recovery period, as described next in section 5.3.

5.3 Physiological responses post-exercise

HR remained elevated in the hypohydration condition throughout the recovery period with an average difference of ~14 beats·min^{-1} (Figure 5). The sustained elevation in HR accompanied the central post-exercise finding of a larger absolute reduction in SBP. The absolute change from baseline to nadir was significant for SBP, whereas the change in MAP and DBP did not differ between conditions. When BP was instead analysed as the change from baseline at each time point, a significant effect of hydration was present for MAP as well as SBP. As MAP is weighted toward DBP, which did not differ between conditions, this effect was likely driven by the SBP response. Although the fall in SBP was larger with hypohydration, the time taken to reach nadir did not differ between conditions, so hypohydration altered the magnitude of the SBP reduction rather than shifting when it occurred. The timing of the nadir did, however, vary considerably between individuals in both conditions. The absence of a

fixed point at which the lowest post-exercise SBP occurs emphasises the value of monitoring across the full recovery period rather than at a single time point or immediately after exercise.

Few studies provide a direct comparison for the present findings, as almost no work has examined pre-exercise hypohydration on the PEH response, which is why the following comparison draws largely on a single study. These findings support those of Lei, Wei, et al. (2025), who reported the greatest magnitude of PEH in the hypohydration condition where participants began exercise hypohydrated and received no fluid during exercise or recovery. However, where the present study found the greatest absolute change from baseline in SBP, Lei, Wei, et al. (2025) found a significant maximum reduction in DBP and MAP but not SBP. Differences in method may explain this. As discussed in section 2.3.1 of the literature review, exercise intensity is a strong determinant of the PEH response, so the severe-intensity, short-duration cycling to exhaustion used by Lei, Wei, et al. (2025) may have driven the reductions in MAP and DBP. Recovery posture is also a determinant, and their participants recovered in the seated position rather than standing as in the present study. Notably, dehydration has been shown to produce a selective reduction in SBP under head-up tilt, accompanied by severe pre-syncopal symptoms that were absent when participants were rehydrated (Harrison et al., 1986). This supports the notion that hypohydration exacerbates the orthostatic component of the post-exercise response, and that the resulting fall in SBP is linked to the onset of pre-syncopal symptoms. Lei, Wei, et al. (2025) also did not report HR during exercise, so it is unclear whether cardiovascular strain differed between their conditions, in contrast to the clear difference shown in the present study (section 5.2). Their finding of no difference in HR post-exercise may also reflect the seated rather than standing recovery and the reduced orthostatic component.

A caveat to the comparison with Lei, Wei, et al. (2025) is that the present study had a smaller sample size, yet we report large effect sizes for the absolute BP results (all >0.14) (Cohen, 2013). It is possible the study was underpowered, as suggested by these large effect sizes, and that the findings would more closely match those of Lei, Wei, et al. (2025) with a larger sample. This reduced sample resulted from participants not completing the full standing recovery period, and one stopping pre-exercise in the hypohydration condition, as described in section 5.4. This may explain why differences appeared in the change from baseline to nadir rather than in absolute BP values, where a larger sample may have been needed to reach significance. The clearest effect of hydration was therefore found in the change from baseline to nadir, which retained the larger sample. Where large effect sizes were accompanied by non-significant results, such as the change in DBP and the analyses of absolute BP across recovery, it is possible an effect of hydration was present but could not be detected in the current sample.

5.4 Orthostatic intolerance and safety

The larger post-exercise fall in SBP with hypohydration carries a clear safety implication. A greater PEH, particularly when recovery is completed in the standing position, can be accompanied by orthostatic intolerance and, as evidenced by the current study, syncope (Halliwill et al., 2013; Halliwill et al., 2014). Four participants experienced orthostatic intolerance with three occurring during the hypohydration condition and one during the euhydration condition, as described in section 4.7. Each recorded an SBP below the pre-determined safety threshold of 100 mm Hg, at which point the visit was terminated. Three of

the four events occurred in the hypohydration condition, indicating that hypohydration increased the likelihood of orthostatic intolerance. Notably, one of these events occurred before exercise had begun, when a participant developed pre-syncopal symptoms during the pre-exercise standing period. This demonstrates that hypohydration, combined with the orthostatic demand of standing, was sufficient to compromise orthostatic tolerance on its own.

The pattern of events is consistent with the mechanisms underlying post-exercise orthostatic intolerance. Standing recovery removes the support of the skeletal muscle pump, which limits venous return and, combined with sustained post-exercise vasodilation, promotes venous pooling. This reduces CO, BP and cerebral perfusion, generating pre-syncopal symptoms (Halliwill et al., 2014; Stewart, 2012). Orthostatic tolerance, however, is not fixed, and higher-intensity exercise produces a greater PEH and a shorter time to syncope (Mündel et al., 2015). Hypohydration adds to this through a reduced PV, which exacerbates the fall in preload on standing. Although not the aim of the current study, the occurrence of outright syncope and several participants achieving an SBP of <100 mm Hg with accompanying pre-syncopal symptoms indicates that orthostatic tolerance is impaired in the hypohydrated condition described herein. Moreover, the participants recruited were healthy, normotensive, recreationally active adults, with no history of orthostatic intolerance or syncope, so these events were not attributable to any pre-existing susceptibility. Given that recreationally active and elite athletes frequently begin exercise hypohydrated (Chapelle et al., 2020; Hew-Butler et al., 2018; Volpe et al., 2009), and may recover in the standing position, this carries practical relevance for the safe management of the post-exercise period, and is considered further in the implications section.

5.5 Implications

Establishing how hydration status shapes the BP responses to exercise is relevant beyond the immediate recovery period. A single bout of endurance exercise can produce PEH that persists for several hours (Aly & Yeung, 2023), and these acute reductions appear to accumulate, with each post-exercise drop stacking on the last to contribute to the lower resting BP seen with regular training (Cornelissen & Fagard, 2005; da Nobrega, 2005). Because these long-term reductions result from repeated acute responses, the factors that alter the magnitude and duration of the acute response are relevant to how exercise is prescribed. The present study demonstrates that hydration status is one of these factors.

By design, the most direct implication of the current study relates to healthy, recreationally active adults. Hypohydration resulted in a significant fall in SBP after exercise and, as described in section 5.4, was occasionally accompanied by orthostatic intolerance and syncope. A deficit in total body water may not be perceived, and as discussed, recreationally active adults and athletes may start exercise hypohydrated and possibly recover in the standing position. This combination may result in an exaggerated PEH, indicating that hydration status deserves attention both before and following exercise. It reinforces the value of assessing hydration status before exercise rather than assuming a euhydrated starting state, as is common in PEH research (Forjaz et al., 2004; Forjaz et al., 1998; MacDonald et al., 2000; Memon et al., 2011; Senitko et al., 2002). In response to this, several practical strategies can be implemented for safety around exercise, hydration and BP. These include ensuring adequate hydration pre-exercise, replacing expected fluid loss during and after exercise, and

taking extra care when fluid loss is expected to be high, for example when exercising in the heat. In addition, recovering in the seated or supine position is a simple, achievable strategy to minimise the risk of post-exercise orthostatic symptoms while preserving the beneficial BP-lowering effects of exercise. Where standing recovery is unavoidable, active recovery may be preferable to passive standing, as movement maintains the skeletal muscle pump and helps limit venous pooling. Where rehydration is used, a prescribed strategy appears more reliable than drinking to thirst for limiting post-exercise cardiovascular strain (Lei, Wei, et al., 2025). The advantage of these strategies is that they are straightforward to implement and can be applied in most settings.

The findings also raise questions for populations beyond healthy young adults. Hypertension is a modifiable risk factor for cardiovascular disease and stroke, so tools that help lower BP can contribute to reducing non-communicable disease risk (Alpsoy, 2020). People with hypertension typically experience greater PEH (Pescatello et al., 2019), so awareness of the risks associated with acute reductions in BP is particularly relevant in this population. In hypertensive individuals, ambulatory reductions in BP have been reported for up to 24 hours after a single 45-minute aerobic exercise session (Taylor-Tolbert et al., 2000). The acute response is therefore clinically meaningful in itself and may help individualise exercise prescription (Cornelissen & Smart, 2013). Hydration status may therefore be relevant to how safely and effectively this response is achieved in clinical populations. No known studies have directly examined pre-exercise hypohydration in a clinical cohort such as hypertension, however the different post-exercise haemodynamic patterns in these individuals mean that beginning exercise hypohydrated may shift the balance between CO and SVR during recovery. The hypohydrated state could exaggerate the post-exercise fall in BP and increase the risk of

orthostatic symptoms, particularly in individuals taking diuretics, who are more prone to hypohydration through increased fluid loss as well as having a reduced capacity to defend BP (Duarte & Cooper-DeHoff, 2010). Diuretics are also used in conditions such as chronic heart failure, where fluid management and physical activity are key (Magdy et al., 2022), extending the relevance to populations beyond hypertension. As the present study examined healthy normotensive adults, the response in clinical populations cannot be established from the findings, but evidence points to a likely risk that warrants future research, given how commonly endurance exercise is prescribed in this cohort.

5.6 Limitations

There are limitations in the present study that should be considered when interpreting these findings. Participants were advised to make no changes to their training in the period between visits, however, adherence was self-reported and their activity between visits could not be controlled. Similarly, participants self-reported diet and while they reported following the same diet leading up to both conditions, variance cannot be fully excluded. Power output was matched between conditions within each participant, but differences in fitness across the study period cannot be fully excluded. There were also differences in the time between visits. This occurred due to participant unavailability and, in the two female participants, the requirement to complete both visits within the same menstrual cycle (mid-luteal). While this impacted scheduling, it kept menstrual phase consistent and the counterbalanced design of the study meant this variation was distributed evenly across both conditions.

The study sample was small (n = 11), with only nine participants available for the during-exercise analyses and the analysis of change from baseline to nadir, while just seven were available for the analyses across the recovery period. This reduced the power of the recovery analyses, as reflected by the large effect sizes. Recovery was also monitored for only 30 minutes, in line with the protocol used in other PEH studies (Kay et al., 2005; Leal & Del Vecchio, 2022), although PEH can persist for several hours after exercise (Dantas et al., 2017). The present study was therefore limited to examining more acute effects and cannot determine whether the difference between conditions was sustained, or became greater, beyond this point. However, the orthostatic stress of standing during recovery combined with hypohydration raises safety considerations as seen in the present study. Therefore, it was unrealistic to have participants standing for a longer period during recovery.

Blood pressure was recorded every five minutes during recovery, so the true nadir may have occurred between measurements. A shorter interval or continuous beat-to-beat measurement could capture the nadir more precisely, but more frequent cuff measurement was not practical in this study. The cuff occlusion, and the reactive hyperaemia that follows it, was sufficient to induce pre-syncopal symptoms in two participants. Measuring more often may therefore have increased the frequency of adverse events in those with low BP, illustrating the challenge of measuring a variable such as BP, where the act of the measurement can itself alter the response being observed.

Finally, hydration status was assessed using USG rather than plasma osmolality. Plasma osmolality is often regarded as a more direct marker of hydration status, but it requires a blood sample and is not always practical. USG and plasma osmolality do not always align

perfectly, and USG can lag behind acute changes in hydration status (Oppliger et al., 2005). However, USG still differed clearly between conditions in the present study and this was enough to produce a difference in the cardiovascular response. This shows the manipulation created a genuine physiological difference, even if USG does not perfectly reflect total body water. USG is also simple and easily obtained, making it a practical way to check hydration status before exercise. Urine can be assessed using a handheld refractometer or, where this is not available, urine colour can be used as a marker.

5.7 Future directions

A clear path for future work is to identify the haemodynamic pattern behind the SBP fall observed with hypohydration. The greater maximum reduction in SBP seen in the hypohydration condition cannot currently be attributed to a reduction in CO or SVR, or a combination of the two. Direct measurement of SV, CO and SVR during recovery would resolve this and allow comment on the mechanism behind the fall. This is of particular interest given that HR remained elevated by 14 beats·min^{-1} during recovery, which suggests an ongoing compensatory response to a reduced blood volume that direct measurement of CO could confirm. A local vascular effect of hypohydration is also worth exploring. While the reduced PV and preload associated with hypohydration may lower CO, a local vascular mechanism contributing to lower SVR could also contribute to the fall in BP. Short-term elevations in blood viscosity, such as during hypohydration, can increase shear stress on the vessel wall and promote vasodilation (Salazar Vázquez et al., 2008). As there is some evidence this could occur, measuring viscosity and vascular responses during recovery may help confirm whether this pathway is involved alongside the CO and SVR balance.

As discussed in Section 2.4.3 of the literature review, the haemodynamic pattern underlying PEH may differ between males and females, with reductions in CO more commonly reported in males and reductions in SVR in females. The present study included only two females and did not measure SV, CO or SVR, however it would be valuable to see whether hydration interacts with these sex-specific mechanisms. Extending this work toward clinical populations such as those with hypertension would also provide valuable insight, given the altered cardiovascular control in these individuals and the use of endurance exercise in exercise prescription.

5.8 Conclusions

The present study examined whether hydration status alters the PEH response to fixed-intensity cycling in healthy, normotensive, recreationally active adults. Beginning exercise in a hypohydrated state resulted in a greater maximum reduction in SBP from baseline upon completion of exercise, and a greater reduction in both SBP and MAP when analysed across the standing recovery. Hypohydration produced PEH sufficient, in some cases, to induce orthostatic intolerance and syncope. Because recreationally active adults and athletes often begin exercise hypohydrated and may recover in the standing position, these findings carry practical relevance for safe management of recovery from exercise. Hydration status is therefore a modifiable factor that shapes the acute BP response to exercise and the risk of post-exercise orthostatic intolerance. Simple attention to hydration and recovery posture can reduce this risk while preserving the BP-lowering benefits of exercise. Establishing this

response in healthy adults provides a foundation for future work in clinical populations, where both the benefits and risks of the post-exercise BP response are greatest.

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Appendices

Appendix A: Ethics Approval Letter

Health and Disability Ethics Committees

Health and Disability Ethics Committees
Ministry of Health
133 Molesworth Street
PO Box 5013
Wellington
6011
hdecs@health.govt.nz

Ethics reference: 2025 EXP 24123

17 October 2025

Dr Blake Perry

Wallace Street
Mt Cook
Wellington 6021
New Zealand

Tēnā koe Dr Perry

APPROVAL OF APPLICATION

Study title: The effects of hydration status on post-exercise hypotension in healthy normotensive recreationally active adults

I am pleased to advise that your application was **approved** by the Northern B Health and Disability Ethics Committee (the Committee) with **non-standard conditions**. This decision was made through the expedited pathway.

Conditions of HDEC approval

HDEC approval for this study is subject to the following conditions being met prior to the commencement of the study in New Zealand. It is your responsibility, and that of the study's sponsor, to ensure that these conditions are met. No further review by the Northern B Health and Disability Ethics Committee is required.

Standard conditions:

- Before the study commences at *any* locality in New Zealand, all relevant regulatory approvals must be obtained.
- Before the study commences at *any* locality in New Zealand, it must be registered in a clinical trials registry. This should be a registry approved by the World Health Organization (such as the Australia New Zealand Clinical Trials Registry, www.anzctr.org.au or https://clinicaltrials.gov/).
- Before the study commences at *each given* locality in New Zealand, it must be authorised by that locality in Ethics RM. Locality authorisation confirms that the locality is suitable for the safe and effective conduct of the study, and that local research governance issues have been addressed.

Non-standard conditions:

- The Committee requested the participant information sheet (PIS) be updated to provide reassurance that the nude body weight taken in a private bathroom is recorded by the participant alone. Please also consider revising the wording around using oral contraception and reporting placebo-pill phase as not all contraceptive pill regimens include inactive (placebo) tablets.
- The Committee requested the broad statement under Māori data sovereignty which allows "Māori organizations to access de-identified study data" should be revised, or accompanied by a supplementary option in the Consent Form (CF), as related to future unspecified use of coded data.
- The Committee requested the Researchers consider their duty of care with respect to anticipated findings of clinical significance which may require direct referral to a General Practitioner (which should be consented), rather than merely suggesting to participants that they visit their doctor.
- The Committee requested the Researchers consider how they will ensure participants have the PIS/CF in their possession for at least 7 days prior to consenting (per the submission form).
- The Committee requested the data management plan (DMP) be updated as data should be retained for 10 years following completion of the study (contrary to the 1 year stated in the form). Please also update the wording around tissue sample destruction to indicate that samples will be discarded in accordance with established guidelines for the disposal of bio-hazardous materials.
- The Committee requested the protocol be updated to define what is meant by "normotensive", or, indicate the blood pressure range being used. Please take into account that the study will recruit physically active individuals so it is expected participants may have lower baseline blood pressure. Please update table 1 to clarify what will be considered an adverse event / serious adverse event for this study. Please also clarify how the data collected in the study will be analysed, and what the expected or potential outcomes are.
- The Committee requested the Researchers consider whether any costs will be reimbursed for participants e.g. parking.

Non-standard conditions must be completed before commencing your study, however, they do not need to be submitted to or reviewed by HDECs.

If you would like an acknowledgement of completion of your non-standard conditions you may submit a post approval form amendment through the Ethics Review Manager. Please clearly identify in the amendment form that the changes relate to non-standard conditions and ensure that supporting documents (if requested) are tracked/highlighted with changes.

For information on non-standard conditions please see paragraphs 125 and 126 of the *Standard Operating Procedures for Health and Disability Ethics Committees (SOPs)*.

After HDEC review

Please refer to the *SOPs* for HDEC requirements relating to amendments and other post-approval processes.

Your next progress report is due by 17 October 2026.

Participant access to compensation

The Northern B Health and Disability Ethics Committee is satisfied that your study is not a clinical trial that is to be conducted principally for the benefit of the manufacturer or distributor of the medicine or item being trialed. Participants injured as a result of treatment received as part of your study may therefore be eligible for publicly-funded compensation through the Accident Compensation Corporation.

Appendix B: Ethics Amendment Letter

*Note the amendments to this ethical application was the addition of a 100 mm Hg systolic BP termination criterion.

Health and Disability Ethics Committees

Health and Disability Ethics Committees
Ministry of Health
133 Molesworth Street
PO Box 5013
Wellington
6011
hdecs@health.govt.nz

Ethics reference: 2026 AM 24123

2 February 2026

Dr Blake Perry

Wallace Street
Mt Cook
Wellington 6021
New Zealand

Tēnā koe Dr Perry

APPROVAL OF AMENDMENT

Study title: The effects of hydration status on post-exercise hypotension in healthy normotensive recreationally active adults

I am pleased to advise that this amendment was **approved** by the Northern B Health and Disability Ethics Committee (the Committee). This decision was made through the post-approval pathway.

Further information and assistance

Please contact the HDECs Secretariat at hdecs@health.govt.nz or visit our website at www.ethics.health.govt.nz for more information, as well as our General FAQ and Ethics RM manual.

Nāku noa, nā

Ms Kate O'Connor

Chair

Northern B Health and Disability Ethics Committee

Encl: Appendix A: documents submitted

Appendix C: Participant Information Sheet (first page only)

MASSEY UNIVERSITY
TE KUNENGA KI PŪREHUROA
UNIVERSITY OF NEW ZEALAND

COLLEGE OF HEALTH
TE KURA HAUORA TANGATA

PARTICIPANT INFORMATION SHEET

Hydration and post-exercise hypotension

Formal study title: The effects of hydration status on post-exercise hypotension in healthy normotensive recreationally active adults

Locality: Massey University, Wellington
Ethics reference: 2025 EXP 24123
Lead investigator: Dr Blake Perry **Contact phone number:** +64 4 979 3492

You are invited to take part in a study on the impact of hydration status on post-exercise hypotension. This study is part of a Massey University thesis paper. **Whether or not you take part is your choice.** If you don't want to take part, you don't have to give a reason, and it won't affect the care you receive. If you do want to take part now, but change your mind later, you can pull out of the study at any time.

This Participant Information Sheet will help you decide if you'd like to take part. It sets out why we are doing the study, what your participation would involve, what the benefits and risks to you might be, and what would happen after the study ends. We will go through this information with you and answer any questions you may have. You do not have to decide today whether or not you will participate in this study. Before you decide, you may want to talk about the study with other people, such as family, whānau, friends, or healthcare providers. Feel free to do this.

If you agree to take part in this study, you will be asked to sign the Consent Form on the last page of this document. You will be given a copy of both the Participant Information Sheet and the Consent Form to keep.

This document is 17 pages long, including the Consent Form. Please make sure you have read and understood all the pages.

VOLUNTARY PARTICIPATION AND WITHDRAWAL FROM THIS STUDY

Your participation in this study is entirely voluntary. If you do not wish to participate, or change your mind after agreeing to participate, then you may withdraw from the study without consequence and at any time. If you do decide to withdraw after agreeing to participate and signing the consent form then we kindly ask that you contact the lead investigator to indicate your withdrawal.

Appendix D: Consent Form

MASSEY UNIVERSITY
TE KUNENGA KI PŪREHUROA
UNIVERSITY OF NEW ZEALAND

COLLEGE OF HEALTH
TE KURA HAUORA TANGATA

CONSENT FORM

Hydration and post-exercise hypotension

Please read the statements below and sign at the end if you agree (consent):		
I have read the Participant Information Sheet and I fully comprehend what it says.		
I have been given sufficient time to consider whether or not to participate in this study.		
I have had the opportunity to use a legal representative, whanau/ family support or a friend to help me ask questions and understand the study.		
I am satisfied with the answers I have been given regarding the study and I have a copy of this consent form and information sheet.		
I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time.		
I consent to the research staff collecting and processing my information, including information about my health.		
I consent to my de-identified information being sent overseas if reasonably requested by researchers outside of the study team.		
I consent to my de-identified information being made available to appropriate Māori organisations for uses that may benefit Māori.		
If I decide to withdraw from the study, I agree that the information collected about me up to the point when I withdraw may continue to be processed.	Yes ☐	No ☐
I understand that my participation in this study is confidential and that no material, which could identify me personally, will be used in any reports on this study.		
I understand the compensation provisions in case of injury during the study.		
I know who to contact if I have any questions about the study in general.		
I understand my responsibilities as a study participant.		
I wish to receive a summary of the results from the study.	Yes ☐	No ☐

Declaration by participant:
I hereby consent to take part in this study.

Participant's name: _____

Signature: _____ Date: _____

Appendix E: GPAQ

Physical Activity

Next I am going to ask you about the time you spend doing different types of physical activity in a typical week. Please answer these questions even if you do not consider yourself to be a physically active person.

Think first about the time you spend doing work. Think of work as the things that you have to do such as paid or unpaid work, study/training, household chores, harvesting food/crops, fishing or hunting for food, seeking employment. *[Insert other examples if needed].* In answering the following questions 'vigorous-intensity activities' are activities that require hard physical effort and cause large increases in breathing or heart rate, 'moderate-intensity activities' are activities that require moderate physical effort and cause small increases in breathing or heart rate.

Questions		Response	Code
Activity at work			
1	Does your work involve vigorous-intensity activity that causes large increases in breathing or heart rate like *[carrying or lifting heavy loads, digging or construction work*] for at least 10 minutes continuously? *[INSERT EXAMPLES] (USE SHOWCARD)*	Yes 1 No 2 *If No, go to P 4*	P1
2	In a typical week, on how many days do you do vigorous-intensity activities as part of your work?	Number of days ⌊__⌋	P2
3	How much time do you spend doing vigorous-intensity activities at work on a typical day?	Hours : minutes ⌊__⌋⌊__⌋ : ⌊__⌋⌊__⌋ hrs mins	P3 (a-b)
4	Does your work involve moderate-intensity activity that causes small increases in breathing or heart rate such as brisk walking *[or carrying light loads*] for at least 10 minutes continuously? *[INSERT EXAMPLES] (USE SHOWCARD)*	Yes 1 No 2 *If No, go to P 7*	P4
5	In a typical week, on how many days do you do moderate-intensity activities as part of your work?	Number of days ⌊__⌋	P5
6	How much time do you spend doing moderate-intensity activities at work on a typical day?	Hours : minutes ⌊__⌋⌊__⌋ : ⌊__⌋⌊__⌋ hrs mins	P6 (a-b)
Travel to and from places			
The next questions exclude the physical activities at work that you have already mentioned. Now I would like to ask you about the usual way you travel to and from places. For example to work, for shopping, to market, to place of worship. [insert other examples if needed]			
7	Do you walk or use a bicycle (*pedal cycle*) for at least 10 minutes continuously to get to and from places?	Yes 1 No 2 *If No, go to P 10*	P7
8	In a typical week, on how many days do you walk or bicycle for at least 10 minutes continuously to get to and from places?	Number of days ⌊__⌋	P8
9	How much time do you spend walking or bicycling for travel on a typical day?	Hours : minutes ⌊__⌋⌊__⌋ : ⌊__⌋⌊__⌋ hrs mins	P9 (a-b)
Recreational activities			
The next questions exclude the work and transport activities that you have already mentioned. Now I would like to ask you about sports, fitness and recreational activities (leisure), [insert relevant terms].			
10	Do you do any vigorous-intensity sports, fitness or recreational (*leisure*) activities that cause large increases in breathing or heart rate like [*running or football,]* for at least 10 minutes continuously? *[INSERT EXAMPLES] (USE SHOWCARD)*	Yes 1 No 2 *If No, go to P 13*	P10
11	In a typical week, on how many days do you do vigorous-intensity sports, fitness or recreational (*leisure*) activities?	Number of days ⌊__⌋	P11
12	How much time do you spend doing vigorous-intensity sports, fitness or recreational activities on a typical day?	Hours : minutes ⌊__⌋⌊__⌋ : ⌊__⌋⌊__⌋ hrs mins	P12 (a-b)

Continued on next page

Appendix E (continued)

Physical Activity (recreational activities) contd.			
Questions		**Response**	**Code**
13	Do you do any moderate-intensity sports, fitness or recreational *(leisure)* activities that causes a small increase in breathing or heart rate such as brisk walking,(*cycling, swimming, volleyball*)for at least 10 minutes continuously? *[INSERT EXAMPLES] (USE SHOWCARD)*	Yes 1 No 2 *If No, go to P16*	P13
14	In a typical week, on how many days do you do moderate-intensity sports, fitness or recreational (*leisure*) activities?	Number of days ⌊__⌋	P14
15	How much time do you spend doing moderate-intensity sports, fitness or recreational (*leisure*) activities on a typical day?	Hours : minutes ⌊__⌋⌊__⌋ : ⌊__⌋⌊__⌋ hrs mins	P15 (a-b)
Sedentary behaviour			
The following question is about sitting or reclining at work, at home, getting to and from places, or with friends including time spent [sitting at a desk, sitting with friends, travelling in car, bus, train, reading, playing cards or watching television], but do not include time spent sleeping. *[INSERT EXAMPLES] (USE SHOWCARD)*			
16	How much time do you usually spend sitting or reclining on a typical day?	Hours : minutes ⌊__⌋⌊__⌋ : ⌊__⌋⌊__⌋ hrs min s	P16 (a-b)

Appendix F: Diet Recall Sheet

For the 12 hours prior to your first study visit please record your diet in the table below. You will be asked to repeat the same diet for the remaining study visits. Please return this sheet at your last study visit.

Hypohydration condition: 18 hours before your visit, you may consume a bolus of water up to 1 L. Record the time you consumed the bolus. After this, please refrain from consuming water or other liquids (e.g. coffee, milk, tea, energy drinks, soups/broths) and foods with high water content (e.g. watermelon, peaches, oranges, cucumbers, lettuce, celery, tomatoes) for the 18 hours before your visit.

Time of visit: _____ *Planned bolus time:* _____

Time bolus was consumed: _____

Euhydration condition: Please consume water as you normally would. However, ensure you consume 500 ml the night before the visit and 500 ml the morning of the visit. Record the time of water consumption.

Time of visit: _____

Planned time (night before, 500 ml): _____ *Time consumed:* _____

Planned time (morning, 500 ml): _____ *Time consumed:* _____

A reminder to avoid:

- Strenuous exercise and alcohol for 12 hours, and caffeine (e.g. coffee, tea, or energy drinks) for 4 hours, prior to arriving at the laboratory.

Food to avoid 12 hours prior to your laboratory visit:

- Beetroot and beetroot juice
- Dark chocolate
- Berries including blackcurrants and grapes
- Nutritional supplements containing high nitrate or anthocyanin content
- Any meals high in sodium (e.g. fast food, processed meats)

Food – record approximate volumes/weights where appropriate	Time

Appendix G: FTP Protocol

1. **Warm-up (5 min):**
    - Resistance: Level 30 for males, Level 20 for females
    - Cadence: 50–60 RPM
    - Participants adjust effort to feel comfortable, targeting RPE ~5/10
2. **Ramp-up test (4–8 min):**
    - Starting resistance: Level 30 for males, Level 20 for females
    - Cadence: 85–90 RPM
    - Resistance increases by 5 levels every minute
    - Rate of Perceived Exertion (RPE) recorded each minute
    - The ramp concludes when the participant reports RPE of 10, reflecting the maximal effort sustainable for 5 minutes
3. **Active recovery 1 (7 min):**
    - Resistance: Level 30 for males, Level 20 for females
    - Cadence: 40–50 RPM
    - Targeting RPE ~5/10
4. **FTP test (5 min):**
    - Resistance: Level reached at the end of ramp-up test
    - Cadence: 85–90 RPM
    - Participants may reduce resistance slightly if needed to maintain target cadence
    - An estimated FTP will be calculated from test data
5. **Active recovery 2 (3 min):**
    - Resistance: Level 30 for males, Level 20 for females
    - Cadence 40–50 RPM
    - Targeting RPE ~5/10
6. **FTP calculation:**
    - 80% of the estimated FTP will be used to determine workload for subsequent fixed-intensity cycling visits
7. **Workload testing (5 min):**
    - Participants will cycle to familiarise themselves with the workload planned for subsequent visits
8. **Cool-down (3 min):**
    - Resistance: Level 30 for males, Level 20 for females
    - Cadence 40–50 RPM
    - Targeting RPE ~5/10