OPTIMISING TRAINING PRESCRIPTION AND PERIODISATION DURING ALTITUDE TRAINING IN ELITE RUNNERS

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For Louise and Chris, two remarkable scientists,

but more importantly human beings, from whom

I have learnt so much. I am incredibly grateful.

"Your attitude, not your aptitude, determines your altitude"

Zig Ziglar

"I used to run to school, 10 km every day. And this at altitude, perfect preparation, really"

Haile Gebrselassie

"It is clearly not the journey for everyone. People succeed in as many ways as there are people. Some can be completely fulfilled with destinations that are much closer to home and more comfortable. But if you long to keep going, then I hope you are able to follow my lead to the places I have gone. To within a whisper of your own personal perfection. To places that are sweeter because you worked so hard to arrive there. To places at the very edge of your dreams."

Michael Johnson

"What I have is a malevolent curiosity. That's what drives my need to write and what probably leads me to look at things a little askew."

David Bowie

Never stop learning

i. ABSTRACT

Altitude training is frequently utilised by elite runners to improve performance in subsequent competition at sea-level. Alongside the beneficial physiological adaptations which can be obtained with a sufficient period of hypoxic residence, periodisation and distribution of training intensity likely have a strong influence on subsequent athletic performance. Moreover, understanding the limitations of exercise in-, and physiological responses to- hypoxia, and how they differ across the spectrum of intensities at which runners are required to train, may assist in effective programming of training at altitude. Whilst sound principles exist regarding sea-level training practices, there is continuing uncertainty regarding the efficacy of altitude training due in part to the neglect of these principles in many studies. Furthermore, the characterisation and periodisation of training is seldom discussed amongst the reasons contributing to observed performance or physiological changes within the altitude training literature. As such, the primary theme of this thesis was a focus on training during altitude exposure, with the aim of optimising altitude training for performance improvement during subsequent sea-level competition.

Study One observed differences in running speed and perceived exertion when elite runners completed the same training sessions (covering four different intensities relevant to middle-distance running), at sea-level and during a live high train high (LHTH) camp at 2100 m. Study Two examined the differences in oxygen uptake and anaerobic contribution between various interval training sessions completed in normoxia, low (1400 m) and moderate normobaric hypoxia (2100 m). Study Three followed a group of elite runners completing a LHTH intervention at 2100 m to prepare for competition within a week of return to sea-level, with training monitored during both the lead-in period at sea-level and the LHTH intervention to identify training periodisation strategies, as well as the effect of LHTH on training load. Finally, utilising a parallel-groups, repeated measures design, Study Four compared the effects

of completing a block of living and intensified training at sea-level, 1600 or 1800 m on performance throughout a subsequent competitive season.

The key findings of this research were: i) compared to sea-level, running speed in elite runners is adversely affected at 2100 m in an intensity-dependent manner (Study One); ii) completing high-intensity interval running at 2100 m simulated altitude, but not 1400 m, is likely to induce a lower $\dot{V}O_2$ and greater anaerobic contribution to exercise during threshold and maximal aerobic sessions when compared to training at 580 m; however race-pace training is largely unaffected (Study Two); iii) elite runners achieved personal best performances in sea-level competition immediately following LHTH at 2100 m (Study Three); and iv) a pre-competition, three week block of LHTH at 1600 or 1800 m yielded greater performance improvements in subsequent sea-level races than undertaking similar training at sea-level (Study Four).

Taken together, the positive performance outcomes noted following altitude training may be due to the greater overall load of training in hypoxia compared to normoxia, effective tapering strategies, individualisation of training and competition schedules, as well as a hypoxia induced increase in haemoglobin mass (Studies Three and Four). Moreover, the wide time frame for peak performances observed following LHTH suggests that the window for optimal performance is highly individual, and factors other than altitude exposure *per se* may be important (Study Four). Contrary to existing guidelines, during natural altitude camps involving elite runners with prior altitude experience, remaining at moderate altitude to complete some high-intensity training may be beneficial, as is integrating established training practices such as overload (utilising hypoxic stress to facilitate the increase in load) and taper into a periodised and monitored training program. In summary, the findings of this thesis may be used to optimise the altitude training process at both low and moderate altitudes, with beneficial implications for elite athletes utilising this strategy during their competition preparation.

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Kevin, I think I gave you a heart attack in our first meeting saying I wanted to start collecting data in a few weeks. Sorry about that, but it looks like it might've all turned out ok

(though that first study was rejected for publication three times - who's counting right?) Thanks for your mentorship and advice, for looking out for my career, as well for sending Julien, your first or second favourite scientist, to get me over the line.

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iv. PUBLICATIONS

Peer-reviewed publications

1. **Sharma AP**, Mujika I. Quantification of training and competition loads in endurance sports: a key to recovery-stress balance and performance. In: Kellmann M, Beckmann J, editors. *Sport, Recovery, and Performance: Interdisciplinary Insights.* Abingdon: Routledge; 2018. p. 132-147, 2018. [Appendix 1]
2. **Sharma AP**, Saunders PU, Garvican-Lewis LA, Clark B, Stanley J, Robertson EY, Thompson KG. Training at 2100 m altitude affects running speed and session RPE at different intensities in elite middle-distance runners. *Int J Sports Physiol Perform*. 2017;12(Suppl 2):S2-147-S2-152. [Chapter 3]
3. **Sharma AP**, Saunders PU, Garvican-Lewis LA, Clark B, Gore CJ, Thompson KG, Périard JD. Normobaric hypoxia reduces $\dot{V}O_2$ at different intensities in highly trained runners. *Med Sports Sci Exerc.* 2018 Aug 7. doi: 10.1249/MSS.0000000000001745. [Chapter 4]
4. **Sharma AP**, Saunders PU, Garvican-Lewis LA, Périard JD, Clark B, Gore CJ, Raysmith BP, Stanley J, Robertson EY, Thompson KG. Training periodisation during Live High Train High at 2100 m improves sea-level performance in elite runners. *J Sports Sci Med.* 2018;17(4):607-616. [Chapter 5]
5. **Sharma AP**, Saunders PU, Garvican-Lewis LA, Clark B, Welvaert M, Gore CJ Thompson KG. Increased training load at 1600 and 1800 m improves performance in national level runners. *Int J Sports Physiol Perform.* 2018 Aug 6. doi: 10.1123/ijspp.2018-0104. [Chapter 6]

Conference proceedings

1. **Sharma AP**, Saunders PU, Raysmith BP, Garvican-Lewis LA, Clark B, Gore CJ, Thompson KG. (2016). Increased training load during 3-4 weeks LHTH at 2100 m improves race performance within 1 week post-altitude in elite runners. 2nd Aspire Academy Sports Science Conference: Monitoring Athlete Training Loads – the Hows and Whys, Doha, Qatar. 24th February.
2. **Sharma AP**, Garvican-Lewis LA, Clark B, Gore CJ, Saunders PU, Thompson KG. (2017). Training periodisation during LHTH at various altitudes improves performance in elite runners. American College of Sports Medicine (ACSM) 64th Annual Meeting, Denver, USA. 31st May.
3. **Sharma AP**, Saunders PU, Garvican-Lewis LA, Clark B, Welvaert M, Gore CJ, Thompson KG. (2017). Performance changes following LHTH at 1600 or 1800 m in national level runners. 22nd annual Congress of the European College of Sport Science (ECSS), Essen, Germany. 5th July.
4. **Sharma AP.** (2018). Optimising training prescription and periodisation during altitude exposure in elite runners. University of Canberra Research Institute for Sport and Exercise (UCRISE) Higher Degree by Research Student Conference. Canberra, Australia. 15th February.

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vii. LIST OF ABBREVIATIONS

%$\dot{V}O_{2max}$	$\dot{V}O_{2peak}$ as % of altitude specific $\dot{V}O_{2max}$
%HbCO	Percentage of bound carboxyhaemoglobin
[BLa]	Blood lactate concentration
ALT1600	Experimental group completing LHTH at 1600 m (Study 4)
ALT1800	Experimental group completing LHTH at 1800 m (Study 4)
ANOVA	Analysis of Variance
AOD	Accumulated oxygen deficit
CL	Confidence limits
CO	Carbon monoxide
CON	Control group
d	Cohen's effect size
eENT1	erythrocyte equilibrative nucleoside transporter 1
EPO	Erythropoietin
FiO_2	Fraction of inspired oxygen
g	Hedges' effect size
GPS	Global positioning system
Hb_{mass}	Haemoglobin mass
HIF-1	Hypoxia inducible factor-1
HR	Heart rate
IAAF	International Association of Athletics Federations
LHTH	Live High Train High
LHTL	Live High Train Low
LHTLH	Live High Train Low and High

LT	Lactate threshold
MAOD	Maximal accumulated oxygen deficit
O_2	Oxygen
PhD	Doctor of Philosophy
RE	Running economy
RER	Respiratory exchange ratio
RPE	Rating of Perceived Exertion
RS	Running speed
SaO_2	Arterial oxyhaemoglobin saturation
SD	Standard deviation
SpO_2	Peripheral capillary oxygen saturation
sRPE	Session rating of perceived exertion
TL	Training load
TSB	Training stress balance
TV	Training volume
$\dot{V}CO_2$	Minute carbon dioxide production
$\dot{V}_E$	Minute ventilation
$\dot{V}O_2$	Oxygen uptake
$\dot{V}O_{2max}$	Maximal oxygen uptake
$\dot{V}O_{2peak}$	Peak oxygen uptake
$v\dot{V}O_{2peak}$	Velocity at peak oxygen uptake
η^2	Eta-squared effect size

1.0 INTRODUCTION

Altitude training is often utilised by elite athletes to improve performance in subsequent competition at sea-level. Live high train high (LHTH) or classic altitude training is the original and preferred method of elite athletes and is frequently observed in their competition preparation plans (Friedmann-Bette, 2008; Tønnessen *et al*. 2014; Solli *et al*. 2017; Turner *et al*. 2018). LHTH refers to athletes living and training at natural altitude (usually 2000 to 2500 m) for a period of two or more weeks to prepare for competitions at altitude, or more frequently, to improve their sea-level performance subsequent to adaptations gained during altitude acclimatisation and/or associated training in hypoxia (Saunders *et al*. 2009a). Whilst many anecdotal reports featuring world-class performances of elite athletes at sea-level following LHTH exist, well-controlled studies of elite athletes using altitude training under ecologically valid conditions with training well characterised are still lacking (Friedmann-Bette, 2008).

In response to the lower partial pressure of oxygen characteristic of higher altitudes, a number of acute and chronic physiological and metabolic adjustments occur to maintain homeostasis and levels of tissue oxygenation both at rest and during exercise (Mazzeo *et al*. 2008). Several chronic adaptations, including an increase in haemoglobin mass (Hb_{mass}), improved oxygen utilisation (Saunders *et al*. 2004) and better muscle buffering (Mizuno *et al*. 1990) are viewed as beneficial to endurance exercise performance, and can be obtained with a sufficient period of hypoxic residence and training (Gore *et al*. 2007, Gore *et al*. 2013). Accordingly, numerous studies have reported improved performance following LHTH (Daniels and Oldridge, 1970, Gore *et al*. 1998, Bonne *et al*. 2014, Rodriguez *et al*. 2015). However, there are also several potentially detrimental effects of hypoxic exposure including depressed immune function, increased oxidative stress and the risk of experiencing acute mountain sickness (Bailey and Davies, 1997). Additionally, upon exposure to moderate

altitude, a number of physiological responses occur to compensate for the reduced arterial oxygen content, both immediately and with continued exposure, which may negatively affect exercise performance. These include increased ventilation (leading to increased risk of dehydration), decreased stroke volume, reduced plasma volume and lower maximal aerobic power (Saunders *et al*. 2009a). As such, several investigations have reported impaired performance following altitude training (Adams *et al*. 1975; Jensen *et al*. 1993; Levine and Stray-Gundersen, 1997; Gore *et al*. 1997; Bailey *et al*. 1998; Gough *et al*. 2012), leading to a sceptical view regarding its efficacy for elite athletes amongst the scientific community (Lundby *et al*. 2012).

The reasons often given in the literature for inconsistent findings between investigations (Bonetti and Hopkins, 2009) are often related to compromising either the hypoxia induced acceleration of erythropoiesis and production of red blood cells, or the maintenance of oxygen flux and training intensity at altitude – two adaptive pathways identified and frequently cited as explanatory for observed performance changes (Levine and Stray-Gundersen, 1997, Chapman *et al*. 1998). Regarding the former, iron status and supplementation (Stray-Gundersen *et al*. 1992), as well as insufficient hypoxic dose (Wilber *et al*. 2007) are often mentioned. Concerning the latter, reduced oxygen availability at altitude leading to athletes training at lower absolute intensities resulting in detraining (Chapman *et al*. 1998) and the relative intensity of training sessions being clamped as equivalent to sea-level, thus also reducing absolute training intensity (Lundby *et al*. 2012) are reasons discussed. However, training prescription (i.e. structure and design of individual training sessions including interval training, work-to-rest ratio, race specific intensities, altitude selection for training sessions) and periodisation (i.e. intra- and inter-week structure of training) during altitude training are often forgotten in the discussion when interpreting subsequent sea-level performances. The lack of training discussion is despite recent studies and reviews highlighting specifically the

importance of individualised training, maintenance of training intensity at altitude, training intensity distribution, and consistent training between parallel athlete groups as being crucial in explaining subsequent changes in sea-level performance (Chapman *et al*. 1998; Friedmann-Bette, 2008; Chapman *et al*. 2014a; Brocherie *et al*. 2017; Robach *et al*. 2018).

Critical to any performance is the prior training completed by an athlete, and it is difficult to assess a competitive performance without placing it within the context of at least the recent training load and overall periodisation of an athlete's preparation (Mujika, 2013). Sound principles exist regarding effective training practices (e.g. overload, tapering, interval training) to enhance endurance performance (Bompa, 1999; Billat, 2001; Issurin, 2010; Buchheit and Laursen, 2013, Mujika *et al*. 2018), however continuing uncertainty about the efficacy of altitude training persists through minimal training quantification in certain studies, and neglect of training principles in others.

A large volume of the altitude training literature has investigated the hypoxia induced increase in erythropoietin (EPO) and Hb_{mass}, based on the established belief this is the primary physiological pathway enhancing post-altitude sea-level performance (Levine and Stray-Gundersen, 1997; Wilber *et al*. 2007, Gore *et al*. 2013, Garvican-Lewis *et al*. 2016). However, exposure to hypoxic conditions has an influence on training itself – and this aspect has received less attention in the literature despite its obvious and acknowledged importance (Chapman *et al*. 1998; Friedmann-Bette, 2008; Mujika, 2013), as well as its ergogenic potential. Periods of intensified training are inherent to the schedules of elite athletes and have been shown to improve performance in endurance sports when combined with a suitable subsequent period of taper (Aubry *et al*. 2014; Clark *et al*. 2014, Rønnestad *et al*. 2016, Rønnestad *et al*. 2017). Training under hypoxic stress allows athletes to experience higher physiological loads than those achieved when completing equivalent training in normoxic conditions (Saunders *et al*. 2009a), and as such one of the benefits of altitude camps may be their use as an additional

physiological stressor to regular training conducted at sea-level. When combined with altitude residence (necessary to stimulate the erythropoietic pathway), as observed during LHTH, training under hypoxia presents an attractive strategy for endurance athletes to stimulate an overload, super-compensation training response without additional mechanic load, which may increase the risk of injury and illness and be counterproductive to maximising performance (Hausswirth *et al*. 2014, Raysmith and Drew, 2016).

Training under moderate hypoxic stress is a key point of difference when considering LHTH compared with live high train low (LHTL) altitude training. Here, training at lower altitudes (0 to 1600 m) is combined with residence at moderate altitudes (2000 to 3000 m), with the goal of maintaining oxygen delivery and exercise intensity during aerobic training, whilst still acquiring physiological adaptations conferred by moderate altitude residence (Levine and Stray-Gundersen, 1997). LHTL has largely replaced LHTH in the scientific literature, however both methods are still widely used in practice by elite athletes (Gough *et al*. 2012, Rodriguez *et al*. 2015, Solli *et al*. 2017). It is important to note, even in athletes utilising the LHTL method, training is often conducted at low altitudes (800 to 1600 m) well above sea-level (Levine and Stray-Gundersen, 1997; Stray-Gundersen *et al*. 2001; Chapman *et al*. 2014a; Robach *et al*. 2018). At these low altitudes, there is still some degree of impairment to aerobic training and maximal oxygen uptake ($\dot{V}O_{2max}$) due to the reduced oxygen availability, with elite athletes appearing to be more susceptible to a performance decline than untrained individuals (Gore *et al*. 1996). As such, it is important to consider the effects altitude may have on performance over the range of intensities at which endurance athletes train, because some elite athletes may be unable to maintain the training velocities required for competitive fitness (Chapman *et al*. 1998). Physiological responses attempting to compensate for the reduced oxygen availability at altitude are insufficient to maintain performance at certain intensities and exercise durations more so than others (Deb *et al*. 2018). Understanding the limitations of

exercise in-, and physiological responses to- hypoxia, and how they might differ across the spectrum of intensities at which endurance athletes are required to train (Billat, 2001), may assist coaches and sports scientists in effective programming of training at altitude. In practical terms, knowledge of these responses and how they apply to different intensities would inform intensity specific modifications to training sessions (stay high or descend to lower altitude, increase recoveries, modify pace) designed to maintain exercise intensity and oxygen flux, or alternatively amplify the anaerobic contribution to interval training at altitude. Such modifications have the purpose of enhancing competitive performance during middle-distance and distance events, where both aerobic and anaerobic contributions to performance are relevant (Gastin, 2001).

There are potentially large benefits in having training camps where athletes can train together, be monitored well, and have training and recovery programs individualised based on prior experience, physiology and training theory. A well monitored and prescribed training program accounting for the physiological limitations of exercise in hypoxia may mitigate the risk of detraining arising from the impairment of training quality at altitude, and therefore facilitate a beneficial adaptive response which may lead to improved performances in sea-level competition. However, to achieve these outcomes, further understanding in two key areas is required. Firstly, the physiological and performance responses during individual training sessions conducted in hypoxia across the spectrum of intensities at which endurance athletes train. Secondly, the effects of an intensified block of training conducted at low and moderate altitudes on sea-level performance, with training load periodisation quantified to contextualise any observed performance changes. Optimising the altitude training process at both low and moderate altitudes can have beneficial implications for elite athletes utilising this strategy during their competition preparation.

Therefore, the primary theme of this PhD thesis is a focus on training prescription (Studies One and Two) and periodisation (Studies Three and Four) during LHTH, with the aim of optimising this strategy for performance improvement during subsequent sea-level competition in elite runners. The aim will be addressed by the achievement of the following objectives:

1. Study One - Determine the effect of training at 2100 m natural altitude on running speed during training sessions covering a range of training intensities in elite runners.
2. Study Two - Determine the effect of low (1400 m) and moderate (2100 m) normobaric hypoxia on $\dot{V}O_2$, anaerobic contribution and other physiological parameters during interval training at three different intensities in highly-trained runners.
3. Study Three - Describe the training load periodisation and consequent physiological and performance changes in a group of elite runners undertaking LHTH at 2100 m, specifically to prepare for sea-level competition immediately afterwards.
4. Study Four - Determine the effect of an in-season, pre-competition block of living and training at 580, 1600 or 1800 m in national-level runners on haemoglobin mass and sea-level performance during the subsequent competition period. Training volume and load were also monitored to help contextualise any changes in performance.

2.0 LITERATURE REVIEW

2.1 INTRODUCTION

Elite endurance athletes frequently utilise altitude training as part of their competition preparations. Various altitude training modalities exist, including classical altitude training, or live high train high (LHTH). LHTH refers to athletes living and training at natural moderate altitude (2000 to 3000 m [Bärtsch and Saltin, 2008]) for two or more weeks to prepare for competitions at altitude, or more frequently to improve their sea-level performance subsequent to adaptations gained during altitude acclimatisation and/or associated training in hypoxia (Saunders *et al*. 2009a). LHTH has largely been replaced in the literature by the more contemporary live high train low (LHTL) strategy. Here, athletes similarly reside at moderate altitudes, (can be achieved at natural altitude, or utilising simulated methods, e.g. hypoxic tents), however complete most training, particularly at high intensities, at a lower altitude, usually at or close to sea-level, to overcome the negative effect of moderate hypoxia on maximal oxygen uptake ($\dot{V}O_{2max}$) and training intensity in elite athletes (Gore *et al*. 1996; Levine and Stray-Gundersen, 1997; Chapman *et al*. 1998).

Several studies have investigated the effect of LHTH and LHTL on sea-level performance, with the results being largely equivocal (Bonetti and Hopkins, 2009), leading to scepticism within the scientific community regarding its use amongst elite athletes (Bailey and Davies, 1997; Lundby *et al*. 2012). Whilst the scientific consensus appears to favour LHTL over LHTH as an ergogenic training strategy in elite endurance athletes (Levine and Stray-Gundersen, 1997; Chapman *et al*. 1998; Bonetti and Hopkins, 2009), many continue to regularly engage in LHTH camps, evidenced by several case studies of champion athletes utilising LHTH with positive performance outcomes (Daniels and Oldridge, 1970; Gore *et al*. 1998; Pugliese *et al*. 2014; Tønnessen *et al*. 2014; Solli *et al*. 2017; Turner *et al*. 2018). Whilst

many anecdotal reports featuring world-class performances of elite athletes at sea-level following LHTH exist, controlled studies of elite athletes using altitude training under ecologically valid conditions with training well characterised are still lacking (Friedmann-Bette, 2008).

The potential benefits of LHTH are that altitude acclimatisation provides the stimulus for several physiological adaptations, and furthermore provides the additional stress of training under hypoxic conditions. Given that relative intensity is higher at any given absolute intensity (i.e. speed/power output) due to the hypoxia induced reduction in $\dot{V}O_{2max}$, altitude training may be used to train at higher relative intensities than at sea-level in experienced athletes (Friedmann-Bette, 2008; Saunders *et al*. 2009a). Such a period of intensified training may be used to increase training load to achieve a super-compensation effect (Aubry *et al*. 2014). Furthermore, the increased intensity of training is not associated with the increased mechanical trauma associated with exercising at higher workloads in weight bearing sports, and so can be particularly beneficial for runners (Saunders *et al*. 2009a). Whilst this may appear an attractive strategy, overcoming the limitations of hypoxia upon exercise at the variety of intensities at which endurance athletes train (in particular the reduction of $\dot{V}O_{2max}$ and consequent reduction of speed/power for a given effort) is critical in facilitating a positive adaptive response and mitigating the risk of detraining. Despite this, the continued use of LHTH by elite athletes suggests it is a key factor in optimising competition performance (Friedmann-Bette, 2008; Tønnessen *et al*. 2014). Therefore, designing and implementing effective training strategies at altitude could provide a competitive advantage for elite endurance athletes (Saunders *et al*. 2009a).

The extensive volume of research concerning altitude training has focused on understanding: i) the physiological adaptations associated with acclimatisation to altitude, ii) performance at sea-level following a period of altitude training and, iii) the factors affecting

the performance response to altitude (for reviews see: Bailey and Davies, 1997; Rusko *et al*. 2004; Mazzeo, 2005; Gore *et al*. 2007; Wilber *et al*. 2007; Bärtsch and Saltin, 2008; Mazzeo, 2008; Friedmann-Bette, 2008; Bonetti and Hopkins, 2009; Saunders *et al*. 2009a; Millet *et al*. 2010; Lundby *et al*. 2012; Gore *et al*. 2013; Chapman *et al*. 2014a; Hawley *et al*. 2018; Ploszczyca *et al*. 2018; Bejder *et al*. 2018). Whilst sound principles exist regarding effective training practices (e.g. overload, tapering, interval training) to enhance endurance performance (Bompa, 1999; Billat, 2001; Issurin, 2010; Buchheit and Laursen, 2013, Mujika *et al*. 2018), the literature concerning the integration of these principles into an altitude setting is lacking compared to other areas, with a majority of training recommendations for altitude sojourns based on coaching anecdotes and elite athlete case reports, rather than scientific studies (Baumann *et al*. 1994; Wilber, 2004; Issurin 2007; Saunders *et al*. 2009a; Sperlich *et al*. 2016; Solli *et al*. 2017). The following review of literature therefore aims to summarise the literature in the first three areas to provide context necessary for the thesis, before exploring the available evidence regarding established endurance training practices, and how they may apply in an altitude setting to optimise sea-level performance.

2.2 PHYSIOLOGICAL RESPONSES TO ALTITUDE TRAINING

The physiological responses to exercise upon acute exposure to moderate altitudes, as well as the adaptations induced through acclimatisation have been extensively reviewed (Berglund, 1992; Bailey and Davies, 1997; Fulco *et al*. 2000; Rusko *et al*. 2004; Gore *et al*. 2007; Bärtsch and Saltin, 2008; Mazzeo, 2008; Gore *et al*. 2013; Hawley *et al*. 2018; Ploszczyca *et al*. 2018). The current section will briefly summarise the aspects that underpin performance enhancement upon return to sea-level.

In response to the lower partial pressure of inspired oxygen that is characteristic at altitude, a number of acute physiological and metabolic adjustments occur to maintain homeostasis and levels of tissue oxygenation both at rest and during exercise (Mazzeo *et al*. 2008). Upon ascent to moderate altitudes, resting arterial oxyhaemoglobin saturation (SaO_2) decreases into the low 90% range (Mazzeo, 2008). Almost immediately, a peripheral chemoreceptor mediated increase in ventilation occurs, continuing over 10-14 days during chronic exposure and resulting in an increase in SaO_2 (Bärtsch and Saltin, 2008). Within the first two days at natural altitude an increase in haemoglobin concentration is also observed, predominantly in response to plasma volume contraction and haemoconcentration (Berglund, 1992; Lobigs *et al*. 2018). Upon initial exposure, heart rate and thus cardiac output increase to compensate for the reduced oxygen content, allowing for sufficient oxygen delivery to the working muscles during submaximal exercise, and unchanged submaximal $\dot{V}O_2$ (Clark *et al*. 2007; Mazzeo, 2008). However, due to the contraction in plasma volume, preload to the heart is reduced, thus reducing maximal cardiac output (Vogel *et al*. 1974). These responses serve to impair endurance exercise performance, necessitating modifications to training, particularly during the initial acclimatisation phase (Saunders *et al*. 2009a; Millet *et al*. 2010).

Perhaps the dominant paradigm behind the potential efficacy of altitude training is the hypoxia induced increase in erythropoiesis and haemoglobin mass (Hb_{mass}), resulting in a concomitant increase in $\dot{V}O_{2max}$ (Berglund, 1992; Levine and Stray-Gundersen, 1997; Levine and Stray-Gundersen, 2005; Gore *et al*. 2013; Saunders *et al*. 2013). Hypoxia inducible factor-1 (HIF-1) has been identified as a major transcription factor with respect to oxygen homeostasis and a key factor in the cascade of adaptations to altitude training (Semenza, 2000).Parameters activated by HIF-1 include erythropoietin (EPO) and transferrin for red cell production and iron metabolism, as well as other genes involved in angiogenesis, upregulation of glycolysis and oxygen transport through the circulatory system (Wang *et al*. 1995; Sasaki *et al*. 2000; Semenza, 2000).

The HIF-mediated responses to hypoxia associated with red blood cells and oxygen transport gained attention because $\dot{V}O_{2max}$ is closely associated with performance (di Prampero, 1986). At moderate altitude, the lower partial pressure of oxygen stimulates EPO production in the kidneys, which in turn stimulates the production of red blood cells in the bone marrow and yields downstream increases in Hb_{mass}, which may translate to improvements in $\dot{V}O_{2max}$ and performance (Levine and Stray-Gundersen, 1997; Stray-Gundersen *et al*. 2001; Saunders *et al*. 2013; Rodriguez *et al*. 2015). EPO levels tend to peak in the blood within one to three days at altitude (Berglund, 1992; Jelkmann, 2011), after which they begin to fall and return to, or drop below baseline, the rate of which may depend on the hypoxic dose (Garvican *et al*. 2012; Chapman *et al*. 2014a; Ploszczyca *et al*. 2018).

Whether altitude training can induce accelerated erythropoiesis and increase Hb_{mass} in elite athletes has been debated in the literature (Robach and Lundby, 2012; Millet *et al*. 2017). Investigations in elite athletes have shown both increases (Stray-Gundersen *et al*. 2001; Heinicke *et al*. 2005; Garvican *et al*. 2012; Gough *et al*. 2012; Bonne *et al*. 2014; Garvican-Lewis *et al*. 2015a; Rodriguez *et al*. 2015) and no change/decreases (Gore *et al*. 1997; Gore *et*

al. 1998; Robach *et al*. 2018) in Hb_{mass} or red cell volume following periods of natural altitude training. Factors affecting the erythropoietic response to altitude likely include the dose of altitude administered, iron supplementation and status, training status, training loads, and presence of illness (Berglund, 1992; Gore *et al*. 1998; Wachsmuth *et al*. 2013; Garvican-Lewis *et al*. 2016; Ploszczyca *et al*. 2018). The balance of the available evidence would suggest that altitude training does induce increases in Hb_{mass} in elite athletes, provided the altitude dose is sufficient (Gore *et al*. 2013; Garvican-Lewis *et al*. 2016). However, the contribution of Hb_{mass} to improved performance following altitude training is less clear, with performance improvements, especially in middle-distance events, reported both with and without increases in Hb_{mass} (Gore *et al*. 1998; Garvican *et al*. 2011).

Several non-haematological adaptations which may be beneficial to sea-level performance have been reported (Gore *et al*. 2007). Foremost amongst these are improvements in buffering capacity and exercise economy. Given the immediate and rapid increase in ventilation characteristic of exposure to altitude, expiration of larger volumes of carbon dioxide than usual reduces its partial pressure in the blood and leads to respiratory alkalosis (Hansen *et al.* 1967). Consequently, increased renal excretion of bicarbonate occurs, which is the main buffer of lactic acid and hydrogen ions (Gore *et al*. 2007). Combined with compensatory respiratory alkalosis, elevated muscle lactate and hydrogen ion concentration may serve to increase overall buffer capacity (Gore *et al*. 2007). Studies in runners, skiers and cyclists residing at altitudes between 2000 and 3000 m have reported increases of 6 to 18% in buffer capacity of the gastrocnemius and vastus lateralis (Mizuno *et al*. 1990; Svedenhag *et al*. 1991; Gore *et al*. 2001), which has been demonstrated to occur alongside improvements in short term running time (Mizuno *et al*. 1990) and maximal accumulated oxygen deficit (Svedenhag *et al*. 1991). These improvements in anaerobic capacity may offer a potential benefit to athletes, particularly during middle-distance events where the anaerobic contribution to performance is

significant (Gastin, 2001). However, other investigations have reported no change in buffer capacity or anaerobic performance following altitude training (Bailey *et al*. 1998; Clark *et al*. 2004).

Several investigations have reported improvements in exercise economy following periods of altitude training (Gore *et al*. 2001; Saunders *et al*. 2004; Saunders *et al*. 2007). Given that endurance performance is attributable to $\dot{V}O_{2max}$, fractional utilisation of $\dot{V}O_{2max}$ (i.e. lactate threshold), and exercise economy (di Prampero *et al*. 1986), enhancement in any of these variables is likely to translate into performance improvements. Potential mechanisms for the improvement in economy could be greater use of carbohydrate for oxidative phosphorylation, an increase in the production of adenosine triphosphate (ATP) per unit of oxygen consumed, enhanced tissue oxygen extraction, a decrease in the ATP cost of muscle contraction, and an increase in efficiency in the excitation-contraction coupling process (Green, 2000; Gore *et al*. 2007; Mazzeo, 2008). Elite distance runners completing three weeks of simulated LHTL residing between 2000 and 3100 m experienced a 3.3% improvement in running economy, a finding which was subsequently replicated in a similar group of elite runners completing 46 nights of LHTL at 2860 m (Saunders *et al*. 2004; Saunders *et al*. 2007). However, other investigations have failed to replicate these findings in trained athletes (Lundby *et al*. 2007), and furthermore, the effect of LHTH at moderate altitudes on exercise economy is less clear. A study of elite runners completing four weeks of LHTH between 1500 and 2000 m revealed no significant changes in submaximal running economy, however post-intervention measurements weren't collected until three weeks following return to sea-level (Bailey *et al*. 1998).

Chronic adaptations to altitude may have negative effects on performance, both at altitude, and upon return to sea-level. Increased ventilation which persists at altitude, and for some time upon return to sea-level (Levine and Stray-Gundersen, 1997), may increase the cost

of breathing, given that a greater proportion of oxygen is required at the level of the lungs to facilitate the additional work of these muscles (Wilhite *et al*. 2013). With acclimatisation to altitude, maximal cardiac output is reduced due to a decrease in stroke volume induced by less venous filling, as well as a decrease in heart rate due to lower cardiac contractility (Gore *et al*. 2007; Mazzeo *et al*. 2008). Muscle blood flow to exercising muscles during maximal exercise may also be reduced after chronic exposure to altitude – whilst improvements in red cell mass and thus SaO_2 occur with acclimatisation, oxygen delivery is tightly regulated such that supply meets demand; as such a decrease in muscle blood flow occurs, as well as a re-distribution of blood flow to non-exercising tissues (Bailey and Davies, 1997; Mazzeo, 2008). Finally, hypoxia can lead to a depression of immune function and increased levels of tissue damage mediated by oxidative stress (Bailey and Davies, 1997; Gore *et al*. 2007).

In summary, physiological adaptations in response to acute and chronic exposure to hypoxic environments are well-established and range from short-term detrimental effects in response to the acute reduction in arterial oxygen content, to longer-term adaptations that can improve endurance performance (Sinex and Chapman, 2015). Balancing the positive adaptations that may result from training in and acclimatisation to hypoxia, while minimising effects that can lead to detraining or maladaptation, is necessary in achieving benefit from altitude training (Sinex and Chapman, 2015).

2.3 PERFORMANCE FOLLOWING ALTITUDE TRAINING

The ergogenic potential of altitude training for sea-level performance has been investigated for over 60 years. The general consensus from coaches and athletes is that LHTH improves performance, a notion evidenced by its frequent and continued use by elite athletes (Pugliese *et al*. 2014; Tønnessen *et al*. 2014; Bellenou *et al*. 2017; Solli *et al*. 2017; Turner *et al*. 2018) and supported by several case reports and controlled studies (Daniels and Oldridge, 1970; Mizuno *et al*. 1990; Gore *et al*. 1998; Bonne *et al*. 2014; Rodriguez *et al*. 2015). Accordingly, a meta-analysis (Bonetti and Hopkins, 2009) reported a 1.6 ± 2.7% improvement in performance for elite athletes following LHTH and noted that this could be increased to as much as 5% with enhancements to altitude protocols to bring them further in line with established recommendations. However, the large variability in these results is confirmed by studies reporting no change or a decrement in performance following LHTH (Faulkner *et al*. 1967; Buskirk *et al*. 1967; Faulkner *et al*. 1968; Adams *et al*. 1975; Jensen *et al*. 1993; Levine and Stray-Gundersen, 1997; Bailey *et al*. 1998; Gough *et al*. 2012), and scepticism regarding its efficacy for elite athletes (Bailey and Davies, 1997; Lundby *et al*. 2012). Additionally, the majority of controlled studies featuring elite athletes show impaired performance (Adams *et al*. 1975; Jensen *et al*. 1993; Levine and Stray-Gundersen, 1997; Bailey *et al*. 1998). Factors that may explain these divergent performance outcomes will be discussed subsequently in this review.

Assessing the performance of elite athletes completing altitude training presents a unique challenge (Gore, 2014). Several factors (see below) exclusive to altitude confound interpretation of any changes, along with other concerns such as motivational issues associated with completing repeated time trials, as well as measurement precision (Gore, 2014). Several studies have demonstrated improved $\dot{V}O_{2max}$ and/or Hb_{mass} following a period of altitude training but unfortunately did not include measures of performance, whether in time trials or

competition (Heinicke *et al*. 2005; Brugniaux *et al*. 2006). Whilst these studies have benefit in demonstrating the adaptive potential of altitude training in elite athletes, the frequent dissociation between physiological and performance adaptations observed within the altitude training literature makes it difficult to extrapolate any changes (Daniels and Oldridge, 1970; Levine and Stray-Gundersen, 1997; Gore *et al*. 1998; Fulco *et al*. 2000; Garvican *et al*. 2011; Rodriguez *et al*. 2015).

The predominant source of evidence for the continued use of classic altitude training by elite athletes exists in the form of case reports and uncontrolled studies (Daniels and Oldridge, 1970; Gore *et al*. 1998; Pugliese *et al*. 2014; Solli *et al*. 2017). For example, six world class runners completing one to two week blocks of LHTH at 2300 m, interspersed with periods at sea-level, collectively achieved 14 personal records in competition, including a world record (Daniels and Oldridge, 1970). World class pursuit cyclists ($\dot{V}O_{2max}$ ~ 80 $mL \cdot kg^{-1} \cdot min^{-1}$) completing four weeks of LHTH at 2690 m achieved a 4% improvement in 4000 m time trial performance at sea-level within 21 days of descent from altitude (Gore *et al*. 1998). Olympic champions in the 20 km race walk and marathon completed three weeks of LHTH at 2090 m, concluding three to four weeks prior to their gold medal winning performances, with performance improvements of 1-4% reported (Pugliese *et al*. 2014). Finally, an Olympic champion cross-country skier undertook five altitude camps (each of 10 to 16 days duration) annually during the five most successful years of her career, accounting for 18-25% of annual training volume (Solli *et al*. 2017). Interestingly, each of these reports was characterised by an increase in training volume and/or load at altitude compared to the preceding period of training at sea-level – when replicated in controlled studies, this has also resulted in performance improvements in swimmers (Bonne *et al*. 2014). While these reports provide an ecologically valid insight regarding the practices of elite athletes at altitude, it is not possible to determine whether sea-level performance improvements are due to adaptations induced by

acclimatisation to altitude, changes in training load, placebo effects, or otherwise (Fulco *et al*. 2000; Lundby *et al*. 2016). For elite athletes chasing marginal improvements in performance to gain a competitive advantage (Hopkins and Hewson, 2001), such matters are likely trivial, yet the lack of controlled studies in the literature leaves the above question relatively unanswered for sport scientists. Unfortunately, the detailed aspects of training unique to these altitude reports in elite athletes (e.g. intensified training, repeated exposures, maintained absolute intensity) are not apparent in many laboratory controlled studies (Adams *et al*. 1975; Bailey *et al*. 1998), perhaps accounting for the divergent findings and gap between science and practice.

Early studies into the effects of altitude training (Buskirk *et al*. 1967; Dill and Adams, 1971; Adams *et al*. 1975) clearly demonstrated an impaired performance capacity at altitude. One aspect of these studies was the matched relative intensity of exercise between altitude and sea-level, allowing the relative contributions of exercise training and altitude acclimatisation to be assessed (Fulco *et al*. 2000). In a well-controlled crossover study, elite athletes completed 19.3 km of running each day for three weeks at 75% of altitude specific $\dot{V}O_{2max}$ (Adams *et al*. 1975). The authors reported no differences in performance when training occurred during residence at sea-level, or 2300 m altitude. However, in keeping the relative intensity consistent between altitude and sea-level, the beneficial aspects associated with altitude acclimatisation (e.g. increase in Hb_{mass}) may be negated due to the consequent reduction in absolute workload, and absence of race-like intensities required to maintain competitive fitness, an important consideration especially if competition is to occur immediately following altitude exposure (Chapman *et al*. 1998; Lundby *et al*. 2012; Chapman *et al*. 2014a). Based on this study (Adams *et al*. 1975) it would appear that adaptations induced by altitude acclimatisation alone may be insufficient to improve performance in elite runners, with training related factors also making a substantial contribution. Whilst adaptations to altitude acclimatisation may be ergogenic, the

potential of hypoxic stress to increase the physiological load of endurance training may result in training adaptations beyond what can be achieved at sea-level, a particularly pertinent consideration for world-class athletes close to the limit of their adaptive potential. However, the efficacy of completing intensified training at altitude compared to sea-level remains largely un-investigated in the literature.

2.4 FACTORS AFFECTING THE PERFORMANCE RESPONSE TO ALTITUDE TRAINING

A variety of reasons are presented in the literature when explaining impaired or unchanged performance following classic altitude training. These include the reduction of $\dot{V}O_{2max}$ in hypoxia, the dose (i.e. living altitude of exposure) of altitude administered, changes in immune function, iron supplementation, the individual response to altitude training and the timing of testing or competition post-exposure. These aspects will be reviewed in this section.

2.4.1 REDUCTION IN $\dot{V}O_{2MAX}$ AND TRAINING INTENSITY

Due to the reduced partial pressure of inspired oxygen at natural altitude, or reduced fraction of inspired oxygen in simulated altitude environments, $\dot{V}O_{2max}$ is reduced (Wehrlin and Hallén, 2006; MacInnis *et al*. 2015). The decline of $\dot{V}O_{2max}$ (~ 6% per 1000 m) increases with altitude (Wehrlin and Hallén, 2006 – **Figure 2.1**) and has been observed in elite athletes (who appear to be more adversely affected than untrained counterparts) at altitudes as low as 580 m (Gore *et al*. 1996). Whilst acclimatisation over time results in improvements in performance and $\dot{V}O_{2max}$ at moderate altitude, these do not recover to sea-level values (Daniels and Oldridge, 1970; Dill and Adams, 1971; Chapman *et al*. 2016).

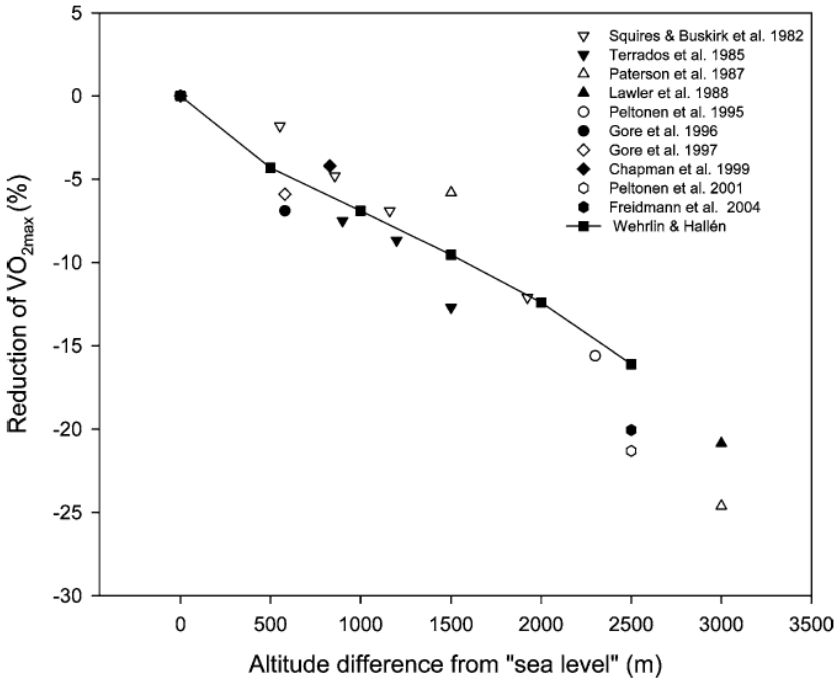


Figure 2.1. The decline of $\dot{V}O_{2max}$ with ascending altitude. Reproduced from Wehrlin and Hallén, 2006.

As a consequence of a lower $\dot{V}O_{2max}$, the relative intensity of exercise for the same speed or power output increases at altitude compared to sea-level, resulting in greater physiological and metabolic adjustments being necessary to maintain homeostasis and performance for a given absolute workload when performed at altitude compared with sea-level (Mazzeo, 2008). A greater physiological cost, increased anaerobic contribution and increased perception of effort in hypoxia (Fulco *et al*. 1998; Weyand *et al*. 1999; Hahn *et al*. 2001; Mazzeo, 2008; McLean *et al*. 2013a) may alter self-selected intensity (i.e. speed, power output) during training sessions at altitude. The reduction in intensity has been observed for a number of training durations, in particular during single interval bouts of exercise greater than two minutes, as well as intermittent (i.e. interval training) exercise (Deb *et al*. 2018 – **Figure 2.2**). In elite female cyclists completing interval training at a simulated altitude of 2100 m, self-selected exercise intensity during prolonged intervals (3 x 10 min) and repeated sprinting (3 x 6 x 15 s) was reduced by 6% (Brosnan *et al*. 2000).

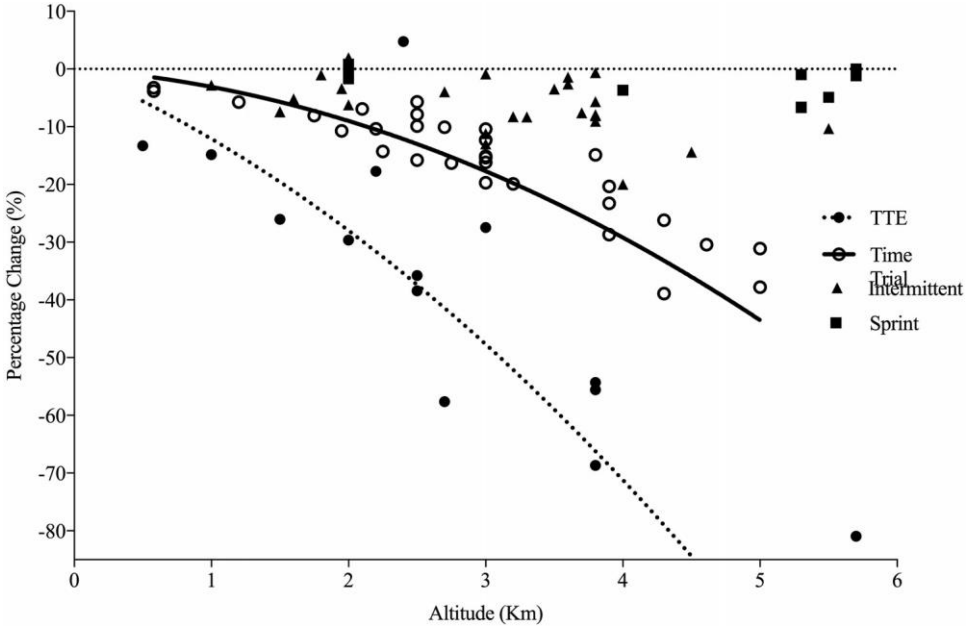


Figure 2.2. The decline in exercise performance with ascending altitude. Reproduced from Deb *et al*. 2018.

The lower absolute intensity of training (i.e. work rate) observed at altitude may have implications for performance at sea-level. In those runners with a substantial decrease in running speed (~ 9%) during interval training at 2500 m, a significant decrement in 5000 m race performance (24 s slower) following altitude training was observed (Chapman *et al*. 1998) compared with those better able to maintain sea-level running speed. Additionally, interval training $\dot{V}O_2$ was also lower in these runners with impaired running speed. Hence, the reduction in training intensity at altitude may be coupled with a lower oxygen flux. At 2500 m simulated altitude, running speed at an equivalent relative intensity (in this case anaerobic threshold) is reduced by 13%, leading to a 19% reduction in $\dot{V}O_2$ (Friedmann *et al*. 2004). Whilst training at near sea-level exercise intensities would therefore seem important in maintaining oxygen flux, certain mechanisms that may allow this (e.g. downhill running or completing shorter intervals at the same speed) would still result in a lower interval training $\dot{V}O_2$. As a result, potential deconditioning may still occur by virtue of training with a lower oxygen flux, impairing subsequent sea-level performance (Levine and Stray-Gundersen, 1997; Chapman *et al*. 1998).Therefore, whilst training at near sea-level intensity is likely an important factor

contributing to improved sea-level performance following altitude training, it is important to ensure adequate oxygen flux during training is also maintained. Together, these are key arguments in favour of selecting LHTL over LHTH (Levine and Stray-Gundersen, 1997; Chapman *et al*. 1998; Brosnan *et al*. 2000 – **Figure 2.3**).

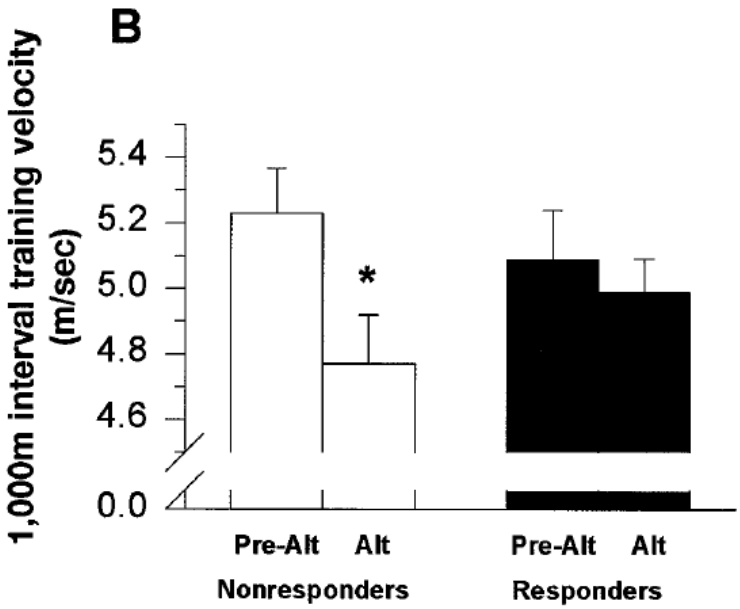


Figure 2.3. Maintenance of exercise intensity at altitude influences subsequent performance at sea-level. Responders were those athletes who had a small, non-significant reduction in interval training velocity at 2500 m, and subsequently improved running performance at sea-level relative to pre-altitude trials. Non-responders had a significant, 9% reduction in interval training velocity, and were slower in subsequent sea-level time-trials. Reproduced from Chapman *et al*. 1998.

2.4.2 HYPOXIC DOSE

Recommendations for altitude training typically suggest living at 2000 to 2500 m for three to four weeks to elicit haematological adaptations purported to improve endurance performance (Wilber *et al*. 2007). A dose-response model of altitude training has been proposed, based on the premise that a hypoxia-induced increase in EPO is the primary physiological pathway enhancing post-altitude sea-level performance (Levine and Stray-Gundersen, 2006; Wilber *et al*. 2007). Recent reviews of the literature suggest that Hb_{mass}, a key measurable outcome of the erythropoietic cascade, increases in a dose dependent manner

following altitude training (Gore *et al*. 2013; Garvican-Lewis *et al*. 2016 – **Figure 2.4**). Accordingly, several investigations of elite athletes completing altitude training in line with the above recommendations report concurrent improvements in Hb_{mass} and sea-level performance (Stray-Gundersen *et al*. 2001; Bonne *et al*. 2014; Rodriguez *et al*. 2015). Whilst improvements in haematological, physiological and performance measures are observed in athletes following low altitude (600 to 2000 m) training (Roels *et al*. 2006; Frese and Friedmann-Bette, 2010; Wachsmuth *et al*. 2013; Garvican-Lewis *et al*. 2015a), other investigations report impaired or unchanged performance, Hb_{mass} and $\dot{V}O_{2max}$ following altitude training between 1619 to 1822 m (Gore *et al*. 1997; Bailey *et al*. 1998; Chapman *et al*. 2014a). Though residence and training at lower altitudes would minimise the impairment to aerobic training and thus potentially be advantageous to performance (Saunders *et al*. 2009a), these venues likely reside below the threshold required to stimulate erythropoiesis and induce a meaningful change in $Hb_{mass.}$ Consequently, improvements following training at lower altitudes are not as consistently observed in comparison to adaptations induced by moderate altitudes greater than 2000 m (Wilber *et al*. 2007; Chapman *et al*. 2014a). In a parallel groups study of runners all completing interval training at 1250 m during a four week LHTL protocol, it was observed that at a group level, athletes residing at 1780 m did not achieve significant improvements in 3000 m time-trial performance nor VO_{2max}, a finding in contrast to their counterparts residing at 2085 and 2454 m (Chapman *et al*. 2014a). However, at an individual level, performance improvements were observed in seven of 10 runners residing at 1780 m (Chapman *et al*. 2014a). It has been previously noted that certain individuals, based on genetic predisposition, may well have a beneficial response to low "doses" of altitude, whilst others may require exposure to higher altitudes to induce adaptations (Levine and Stray-Gundersen, 2006). These conflicting results provide the impetus for further inquiry into the efficacy of low altitude training for sea-level performance enhancement in elite athletes.

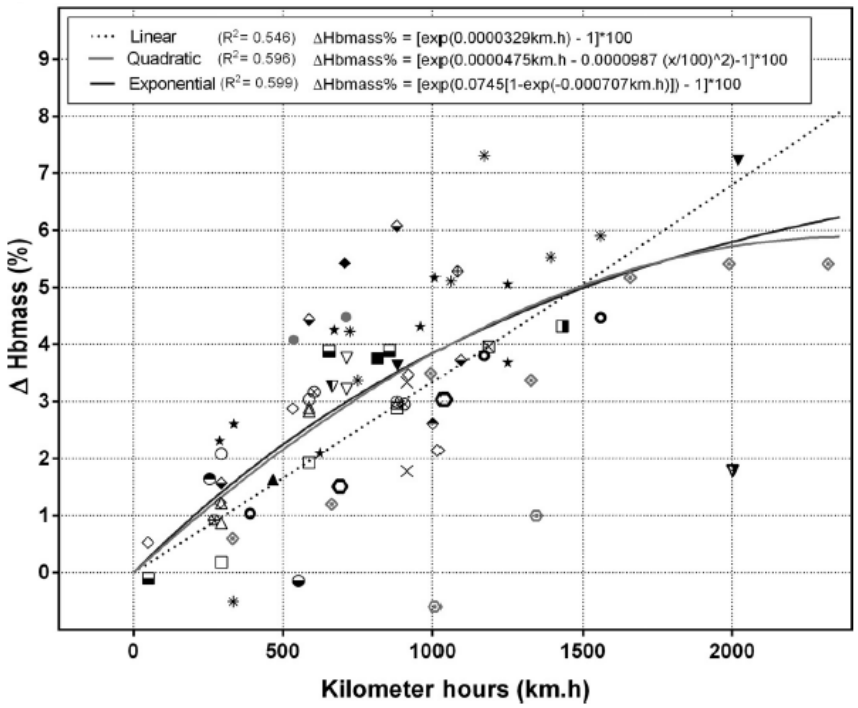


Figure 2.4. Haemoglobin mass increases during altitude training in a dose-dependent manner. Reproduced from Garvican-Lewis *et al*. 2016.

2.4.3 IMMUNE FUNCTION

Hypoxic exposure and exercise are stressors that can impact on immune function (Mazzeo, 2005; Gleeson, 2007). Acute exercise and acute exposure to hypoxia mediate similar circulating lymphocyte and neutrophil responses, resulting in temporary immunosuppression (Pedersen and Steensburg, 2002; Mazzeo, 2005). In response to reduced arterial oxygen content in hypoxia, increased epinephrine release and sympathetic nerve activity serve to improve oxygen delivery through increasing ventilation and cardiac output, redistributing blood flow to key tissues, and altering substrate selection to ensure economical use of oxygen (Mazzeo, 2005). The combined stress of training and hypoxia has been associated with immune suppression through the increased release of epinephrine and impairment of T-cell activation and proliferation, which may increase the incidence of infection and illness in elite athletes, particularly during the initial exposure to hypoxia, compromising beneficial physiological or performance adaptations (Bailey and Davies, 1997; Mazzeo, 2005; Friedmann-Bette, 2008).

German elite swimmers reporting symptoms of illness during altitude training camps experienced no improvements in Hb_{mass}, with a negative effect of sickness also found on sea-level competition performance following altitude training (Wachsmuth *et al*. 2013). British elite runners completing four weeks of moderate altitude training at 1500 to 2000 m did not improve performance in maximal or severe intensity tests, nor submaximal running economy, and reported a 50% greater incidence of illness during altitude training compared to sea-level training (Bailey *et al*. 1998). Resting levels of plasma glutamine were observed to be decreased during altitude training in this study and proposed as a potential reason for the increased incidence of illness, given the role of glutamine in maintaining healthy immune function (Bailey *et al*. 1998). Given the cumulative effects of hypoxic exposure and exercise on immune function, the authors proposed a model whereby an increased training load exacerbated by hypoxia may decrease glutamine below the physiological range, thus increasing the risk of infection (Bailey *et al*. 1998).

It would appear that the control and modulation of training load at altitude is important for maintaining the immune status of athletes, as well as to facilitate training adaptations, and suggests the need for close monitoring of training load and wellness at altitude. Indeed, overtraining symptoms (e.g. illness, reduced serum testosterone) were reported in Australian world champion track cyclists who failed to improve Hb_{mass} or $\dot{V}O_{2max}$ after completing four weeks of altitude training at 2690 m, with the timing of the symptoms coinciding with an increased training volume (Gore *et al*. 1998). It has been previously shown that high intensity interval exercise completed at a similar absolute intensity or relative metabolic stress in moderate hypoxia compared to sea-level induces a much greater sympathoadrenal stress response (Mazzeo *et al*. 2001; Niess *et al*. 2003). As a result, it has been recommended the additional impact of moderate hypoxia on the stress response to training should be considered if repeated training sessions are performed within a short period of time (Niess *et al*. 2003).

Whilst the overall training volumes completed by the cyclists in the previous study compared favourably with their training in the previous months, the nature of their training involving high intensity efforts on consecutive days, as well as training load spikes which may have contributed to the illness observed (Gore *et al*. 1998).

2.4.4 IRON STATUS AND SUPPLEMENTATION

Given the interrelationship between changes in Hb_{mass}, $\dot{V}O_{2max}$ and endurance performance observed with altitude training (Saunders *et al*. 2013), facilitating an increase in Hb_{mass} during altitude exposure is often viewed as a required contributor towards a positive performance outcome following altitude training (Levine and Stray-Gundersen, 2005). Sufficient iron stores are a necessary component to support hypoxia mediated increases in erythropoiesis and Hb_{mass} with chronic altitude exposure (Stray-Gundersen *et al*. 1992; Levine and Stray-Gundersen, 1997; Govus *et al*. 2015). Upon activation of erythropoiesis at altitude, the iron demand increases, and a concomitant reduction in hepcidin production (endogenous regulator of iron homeostasis) allows for increased iron absorption and release of iron stores (Ploszczyca *et al*. 2018). However, in the absence of adequate iron stores pre-altitude, haematological adaptations during initial altitude exposure may be absent (Govus *et al*. 2015). Stray-Gundersen and colleagues (1992) have previously shown red cell volume remained unchanged in non-supplemented, iron deficient runners, contrasting to their non-iron deficient counterparts, who experienced an increase in red cell volume. It has therefore been recommended to supplement with iron prior to altitude exposure to normalise iron stores and promote adaptation upon arrival to altitude, as well as during altitude exposure (Govus *et al*. 2015; Constantini *et al*. 2017; Garvican-Lewis *et al*. 2018). Compared with placebo or no iron supplementation, greater increases in Hb_{mass} following altitude training have been observed in athletes supplementing with either oral iron in a dose-dependent manner (Govus *et al*. 2015), or intravenous iron, with no differences observed between oral and intravenous iron (Garvican-

Lewis *et al*. 2018). No increases in Hb_{mass} were observed in the placebo supplementation group, and given that all athletes in the study were non-iron deficient, collectively this suggests the greater relative importance of iron supplementation during altitude training over pre-altitude iron stores (Garvican-Lewis *et al*. 2018).

2.4.5 INDIVIDUAL RESPONSE TO ALTITUDE

The individual variation in physiological and performance responses to hypoxia has been well established (Chapman *et al*. 1998; Chapman, 2013). A meta-analysis (Bonetti and Hopkins, 2009) reported 1.6 ± 2.7% and 4.0 ± 3.7% improvements in performance in elite athletes completing LHTH and LHTL respectively, highlighting substantial variation between investigations, likely indicative of the variety of protocols used, as well as a degree of individual variability. Furthermore, substantial individual variation greater than the mean response has been frequently reported in different studies concerning both LHTH and LHTL. In a well-controlled study investigating the effects of three weeks altitude exposure at 2300 m on performance, elite athletes completed 19.3 km of running each day at 75% of $\dot{V}O_{2max}$, and it was observed that half of the participants improved their two mile running time at sea-level following altitude training, whereas the other half were slower (Adams *et al*. 1975). Whilst the training completed during this study may not be indicative of how elite athletes typically train, the controlled design allows for the effects of altitude to be examined independent of any training effects, and therefore highlights the marked differences between individuals regarding their response to altitude.

The potential of acquiring a beneficial performance response from altitude training has traditionally been attributable to two major adaptive pathways – the hypoxia induced acceleration of erythropoiesis and production of red blood cells, and the maintenance of oxygen flux and training intensity at altitude (Levine and Stray-Gundersen, 1997; Chapman *et al*.

1998). Factors pertaining to each of these have been shown to exhibit a wide degree of both inter- and intra- subject variability. Numerous studies have shown between athlete variation regarding the hypoxia induced increase in EPO and Hb_{mass} (Chapman *et al*. 1998; Friedmann *et al*. 2005; Wachsmuth *et al*. 2013; McLean *et al*. 2013b). In a retrospective analysis of collegiate runners, it was observed that athletes who improved performance in sea-level 5000 m time-trials following altitude training had a larger increase in EPO upon ascent to altitude compared to those who failed to improve performance, and EPO remained elevated after 14 days (Chapman *et al*. 1998). Furthermore, these athletes, termed "responders" also had significant increases in red cell volume and $\dot{V}O_{2max}$ compared to their "non-responder" counterparts (Chapman *et al*. 1998). A recent study in elite swimmers demonstrated that the acute EPO increase was highly reproducible (r = 0.95) in individuals completing two LHTH camps at 2320 m interspersed by three months at sea-level (Wachsmuth *et al*. 2013). However, a non-significant relationship between the EPO response and Hb_{mass} was reported (Wachsmuth *et al*. 2013), as were no significant relationships between the change in Hb_{mass} and swimming performance, replicating a previous study in elite junior swimmers (Friedmann *et al*. 2005). A study in elite Australian Rules footballers undertaking two pre-season LHTH camps at 2100 m in consecutive years reported a similar (4%) mean increase in Hb_{mass} after each camp (McLean *et al*. 2013b). An individual response (Hopkins, 2015) of approximately half the group mean effect was reported, indicating that most players gained benefit. However, a small, non-significant correlation (r = 0.21, p = 0.59) was reported for Hb_{mass} change between the two camps, indicating the same individuals generally did not change their Hb_{mass} consistently from year to year (McLean *et al*. 2013b). Finally, in highly trained runners completing two, three week blocks of LHTL interspersed by a five week washout period near-to sea-level, reproducible mean improvements in $\dot{V}O_{2max}$ and Hb_{mass} of ~ 3% were reported after each block, however these did not translate to reproducible improvements in time-trial performance, with

mean changes of 1.4% faster and 0.7% slower being observed respectively (Robertson *et al*. 2010a). Additionally, there was a lack of association between changes in block one versus block two, with moderate but unclear correlations for $\dot{V}O_{2max}$ and Hb_{mass}, and only a trivial correlation for time-trial performance (Robertson *et al*. 2010a – **Figure 2.5**). Together, these studies suggest the erythropoietic and Hb_{mass} response to altitude does not appear to be a fixed trait, and thus it would appear unlikely that the individual variability in the performance response to hypoxia is related solely to this pathway. Furthermore, the intra-individual differences reported (Saunders *et al*. 2009b; Robertson *et al*. 2010a; McLean *et al*. 2013b), indicate that the performance response to altitude is not consistent and dependent solely on physiological adaptations, but also fitness, training status, fatigue and timing of competition, which require individual management to ensure optimal performance (Robertson *et al*. 2010a).

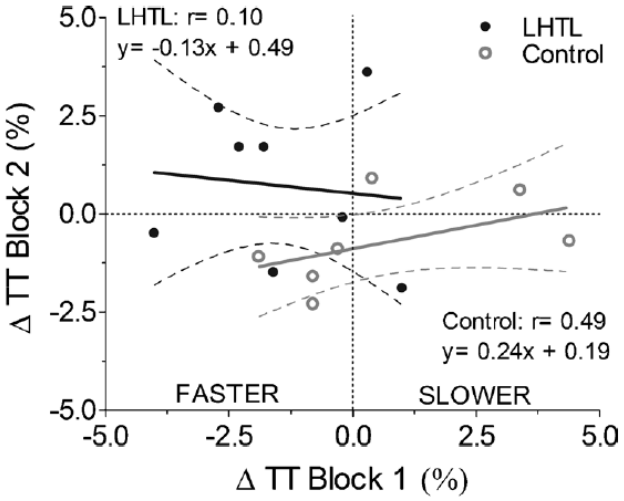


Figure 2.5. Intra- and inter-individual differences in performance in runners completing two blocks of LHTL altitude. Within individual changes in performance show little reproducibility following similar bouts of LHTL, and there are also wide inter-individual responses observed. Reproduced from Robertson *et al*. 2010a.

Measurement of peripheral oxygen saturation at rest (SpO_2) may reveal information regarding an athlete's haematological response to altitude (Ploszczyca *et al*. 2018). The increase in EPO levels is proportional to the level of hypoxia and decline in SpO_2 (Eckardt *et al*. 1989; Roberts and Smith, 1999). Typical metrics proposed to quantify the hypoxic dose

administered, concern the hypoxic stimulus or altitude achieved (Garvican-Lewis *et al*. 2016), whereas the measurement of "saturation hours" refers to the hypoxic response, and accounts for the large inter-individual variability frequently demonstrated to a given hypoxic dose (Chapman *et al*. 1998; Friedmann *et al*. 2005; Millet *et al*. 2016). Athletes with a higher saturation value or faster rise in SpO_2 over the course of an altitude exposure may need to extend the length of an exposure, increase exposure to a higher altitude, or complete more high intensity exercise at altitude to induce greater desaturation (Ploszczyca *et al*. 2018). Alternatively, athletes with low values may be more suited to lower altitudes, and greater modifications to training, however this assertion is speculative and warrants further investigation.

The ability of individuals to maintain SpO_2 has been strongly linked to the maintenance of both $\dot{V}O_{2max}$ and performance at altitude (Chapman *et al*. 1999; Chapman *et al*. 2011; Chapman, 2013). In elite runners, it was demonstrated that low saturation athletes (SpO_2 during race pace exercise at sea-level < 91%) had a significantly greater slowing of 3000 m time-trial performance at 2100 m altitude compared to high saturation athletes (SpO_2 > 93%) (Chapman *et al*. 2011). The mechanism behind arterial oxyhaemoglobin desaturation, leading to exercise induced arterial hypoxemia, can be traced to limitations of oxygen diffusion, either at the lung or muscle microvasculature, as well as inadequate hyperventilation (Chapman, 2013). Furthermore, elite athletes would appear to be more susceptible to this phenomenon than lesser trained individuals, and even within elite athletes frequently utilising altitude, it is evident some athletes are more negatively affected during exercise in hypoxia than others (Gore *et al*. 1996; Chapman, 2013). As a result, pre-screening of an athlete's desaturation response during maximal exercise, as well as the hypoxic ventilatory response, have been proposed to provide information pertaining to their response to altitude, which could be used to make alterations to

training at altitude (e.g. lengthened recoveries, more sessions at lower altitude) necessary to help maintain exercise intensity (Saunders *et al*. 2009a; Chapman, 2013).

2.4.6 TIMING OF COMPETITION POST-ALTITUDE EXPOSURE

The optimal time to compete following altitude training is an important issue for athletes, coaches and scientists. A recent review on this issue highlighted the decay of physiological adaptations conferred by altitude training (Chapman *et al*. 2014b), including the increase in Hb_{mass} and ventilatory acclimatisation as key factors to consider when scheduling competition. Given the marked individual variability displayed in the induction of these adaptations, it would follow that the de-acclimatisation response would be similarly variable, and likely not feature a simple reversal of the altitude acclimatisation response (Chapman *et al*. 2014b).

An altitude induced increase in Hb_{mass} is unlikely to persist at sea-level for a prolonged period. In elite biathletes, Hb_{mass} had returned to baseline values within 16 days at sea-level following three weeks of altitude training (Heinicke *et al*. 2005). In elite cyclists, a 3.5% increase in Hb_{mass} observed after 19 days at 2760 m had dropped to 2% above baseline 11 days after return to sea-level (Garvican *et al*. 2012). A meta-analysis revealed than within 20 days of LHTH at a median altitude of 2320 m, Hb_{mass} remained elevated by 3.4%, however when Hb_{mass} was assessed following 20 days at sea-level, no significant differences with baseline were observed (Gore *et al*. 2013). In elite, high altitude native, Kenyan runners, Hb_{mass} remained stable for the first 14 days at sea-level but declined by 6% after five weeks (Prommer *et al*. 2010). In sea-level natives, this decline in red cell mass seems to be driven by a process termed neocytolysis, or the selective haemolysis of young circulating erythrocytes in times of red blood cell excess (Rice *et al*. 2001). Therefore, if altitude induced increases in Hb_{mass} are quickly negated upon return to sea-level, this could affect the optimal timing of competition, especially during aerobically dominant events, where oxygen delivery is a key determinant of performance. Alternatively, training with additional Hb_{mass} is also beneficial, and may explain

increased performance several weeks following altitude training, regardless of whether Hb_{mass} has returned to baseline levels (Chapman *et al*. 2014a).

Increases in submaximal and maximal ventilation mediated by altitude exposure often persists for a number of days, however the exact time-frame for the return to baseline levels is yet to be clearly elucidated (Daniels and Oldridge, 1970; Levine and Stray-Gundersen, 1997; Stray-Gundersen *et al*. 2001). Whether or not increased ventilation can help improve performance is a contentious issue; on one hand, higher ventilation can induce alkalosis, which has been shown as beneficial to performance during 800 m running (Wilkes *et al*. 1982). Conversely, the work of breathing may increase with added ventilation, which results in a greater fraction of $\dot{V}O_{2max}$ being diverted to the lungs, thus reducing oxygen delivery to locomotor muscles (Wilhite *et al*. 2013; Chapman *et al*. 2014b). As such, the timing of competition post-altitude competition with respect to ventilatory adaptations merits further investigation.

Anecdotal evidence from coaches suggests competing i) within 48-72 hours upon descending from altitude, whilst adaptations conferred by altitude acclimatisation and training are at their peak or ii) after two to three weeks of re-acclimatisation to sea-level, benefitting from training with heightened physiological capacity for a period after the altitude camp, is optimal (Baumann *et al*. 1994; Millet *et al*. 2010; Chapman *et al*. 2014b). Improved performance at both these time-points has been observed in scientific investigations with elite athletes (Stray-Gundersen *et al*. 2001; Wachsmuth *et al*. 2013), however only a handful of studies include serial performance measures following altitude training to identify the optimal window for competition (Levine and Stray-Gundersen, 1997; Gore *et al*. 1998; Saunders *et al*. 2009b; Wachsmuth *et al*. 2013; Saugy *et al*. 2014; Rodriguez *et al*. 2015).

A repeated measures design in seven elite runners (Saunders *et al*. 2009b) showed the timing of a peak performance post-altitude exhibited large individual variability, with some athletes achieving their best performance during time-frames (three to 13 days post-altitude)

traditionally viewed as sub-optimal for performance (Baumann *et al*. 1994; Millet *et al*. 2010; Chapman *et al*. 2014b). Investigations of elite swimmers report impaired or unchanged performances compared to baseline immediately following altitude training, with peak performances occurring two to four weeks thereafter (Wachsmuth *et al*. 2013; Rodriguez *et al*. 2015). Alternatively, other investigations in swimmers have shown tendencies towards improvements in 200 m swimming time (p = 0.051) at one week following LHTH, with no change observed in a parallel group training at sea-level (Bonne *et al*. 2014). Studies involving runners demonstrate improved performance both immediately following altitude exposure, and after a period of sea-level training (Levine and Stray-Gundersen, 1997; Saunders *et al*. 2009b). In the original study comparing LHTH and LHTL in collegiate runners, performance in the LHTH group did not improve immediately following the altitude sojourn, compared to a 1.5% improvement observed in the LHTL group, suggesting that LHTL may be more effective for performance in the immediate post-altitude window (Levine and Stray-Gundersen, 1997). The inclusion of low altitude or sea-level high intensity workouts during a LHTL program, facilitating the maintenance of training intensity and oxygen flux may account for this observation (Chapman *et al*. 2014b). Furthermore, in middle and long-distance events where aerobic metabolism and oxygen carrying capacity are significant determinants of performance (Gastin, 2001), competing immediately following an altitude sojourn whilst Hb_{mass} is likely to be at its peak would appear advantageous. Alternatively, a period of sea-level training following LHTH or LHTL may be beneficial to performance, particularly if an athlete can gain an additional training response from training at a higher level, facilitated by the additional Hb_{mass} and potential ventilatory adaptations conferred by altitude training (Chapman *et al*. 2014b). Along with the rate of decay of these adaptations, the periodisation and distribution of training intensity completed by the athlete, particularly within the immediately preceding period, would likely also contribute substantially to the timing of a peak performance.

2.4.7 TRAINING

Altitude training and exposure to hypoxic conditions has an influence on training itself – and this aspect has received less attention in the literature despite its obvious and acknowledged importance (Chapman *et al*. 1998; Friedmann-Bette, 2008; Mujika, 2013). Whilst the factors discussed above contribute significantly to an observed response following altitude training, it is almost impossible to assess a competitive performance without placing it within the context of at least the recent training load and overall periodisation of an athlete's preparation (Mujika, 2013). Sound principles exist regarding effective training (e.g. overload, tapering, interval training) and training quantification methods, however they are rarely discussed in the literature when interpreting performances following altitude training. As such, the remainder of this review will explore these principles, and how they may relate to altitude training.

2.5 TRAINING LOAD QUANTIFICATION

2.5.1 IMPORTANCE OF LOAD MONITORING FOR ALTITUDE TRAINING

To maximise the effectiveness of training and achieve peak performance at a desired time, quantifying the training load of an athlete and its relationship to performance outcomes should be a priority (Borresen and Lambert, 2009). An incorrect training load may lead to excessive accumulated fatigue or detraining; an appropriate load should facilitate optimal improvements in performance. To enhance the possible benefits of altitude training or exposure, it is paramount that an effective monitoring system is in place to assess fitness and fatigue responses to training, especially given that the additional stress imposed by the hypoxic environment may lead to an increased risk of illness, maladaptation or overtraining compared to training in normoxic conditions (Baumann *et al*. 1994; Bailey and Davies, 1997; Mazzeo, 2005; Saunders *et al*. 2009a; Buchheit *et al*. 2013; Schmitt *et al*. 2018). Detailed monitoring of an athlete's physical and perceptual responses to training is required; i) in the lead-up period to ensure they are in appropriate physical condition to maximise the benefit of altitude training, ii) during training at altitude to minimise the risk of overtraining which may be increased in a hypoxic environment, and iii) post altitude, as training quality may be much higher as a result of adaptations conferred from altitude, and optimal loads during this period are critical. To achieve this, a longitudinal monitoring approach is necessary. However, this is seldom seen in the altitude training literature, perhaps contributing to the perceived uncertainty regarding performance following altitude training (Bonetti and Hopkins, 2009). Indeed, a summary of LHTH studies, and their inclusion of physiological, performance and training information, is provided in **Table 2.1**.

Table 2.1. Summary of physiology, performance and training data presented in LHTH altitude studies.

Study	Participants	Control group	Altitude exposure	$\dot{V}O_{2max}$	Hb_{mass}	Performance	Training quantification	Acclimatisation
Adams *et al*. 1975	12 highly trained runners - $\dot{V}O_{2max}$ = 73 $mL \cdot kg^{-1} \cdot min^{-1}$	Yes – crossover design	3 weeks @ 2300 m	↓ 3%	-	≈ (2 mile TT)	Yes 19.3 km each day @ 75% $\dot{V}O_{2max}$	-
Bailey *et al*. 1998	24 Elite runners - $\dot{V}O_{2max}$ = 75 $mL \cdot kg^{-1} \cdot min^{-1}$	Yes	4 weeks @ 1500-2000 m or 1640 m	≈	-	↓ 2% (1640 m) ≈ (1500-2000 m) (4 x 1000 m intervals)	No Same relative intensity at altitude as sea-level	-
Bonne *et al*. 2014	10 Danish Olympic swimmers	Yes	1 week @ 3094 m followed by 3 weeks @ 2130 m	≈	↑ 6% in LHTH ≈ in CON	Tendencies for larger improvements ($p < 0.1$) in maximal 200 m and 3000 m swimming in LHTH vs. CON	Yes ↑ training load at altitude	Higher proportion of low intensity training vs. sea-level during week 1 at altitude
Buskirk *et al*. 1967	6 collegiate runners	No	48-63 days at 4000 m	≈	-	↓ 1.5% No athletes improved on pre-altitude performance (preferred event)	Periodisation not reported Duration and intensity of training ↓ 60% for initial 3 weeks Reached 75% of pre-altitude training	↓ duration and intensity compared to sea-level

Study	Participants	Control group	Altitude exposure	$\dot{V}O_{2max}$	Hb_{mass}	Performance	Training quantification	Acclimatisation
Daniels *et al*. 1970	6 elite runners - $\dot{V}O_{2max}$ = 74 $mL \cdot kg^{-1} \cdot min^{-1}$	No	2 x 2 weeks and 2 x 1 week @ 2300 m with 5 to 11 days at sea-level in between	↑ 5%	-	↑ 14 personal best performances in competition	Periodisation not reported Normal sea-level training continued at altitude	Hard training sessions included from outset, time trials conducted on initial 2 days at altitude
Faulkner *et al*. 1967	15 collegiate swimmers	No	2 weeks @ 2300 m	≈	-	≈	No	-
Faulkner *et al*. 1968	5 collegiate runners $\dot{V}O_{2max}$ = 69 $mL \cdot kg^{-1} \cdot min^{-1}$	No	6 weeks @ 2300 m	≈	-	≈ or ↓ (1 to 3 mile TTs)	No Individual variation in intensity and duration of training	-
Frese *et al*. 2010	8 elite runners	Yes	20 days @ 1300 m, 19 days at sea-level, 22 days at 1650 m	-	↑ 5%	-	-	-
Garvican *et al*. 2012	8 elite cyclists $\dot{V}O_{2max}$ = 75 $mL \cdot kg^{-1} \cdot min^{-1}$	Yes	3 weeks @ 2760 m	-	↑ 3.5%	-	Yes Lead-in training not quantified Volume and intensity ↑ during camp	↓ training duration and intensity during first 3 days at altitude

Study	Participants	Control group	Altitude exposure	$\dot{V}O_{2max}$	Hb_{mass}	Performance	Training quantification	Acclimatisation
Gore *et al*. 1997	21 national and international level runners $\dot{V}O_{2max}$ ~ 70 $mL{\cdot}kg^{-1}{\cdot}min^{-1}$	Yes	4 weeks @ 1300 m or 1740 m	↑ 2.6% (1300 m) ↑ 2.1% (1740 m)	≈	↑ 2.4% (1300 m) ↑ 1.7% (1740 m) (3.2 km TT)	Periodisation not reported Identical for all participants	-
Gore *et al*. 1998	8 elite cyclists $\dot{V}O_{2max}$ = 81 $mL{\cdot}kg^{-1}{\cdot}min^{-1}$	No	4 weeks @ 2690 m	↓ 2.2%	≈	↑ 4% (4000 m pursuit)	Yes ↑ training volume at altitude	↑ proportion of low intensity training
Gough *et al*. 2012	17 Australian national team swimmers	Yes (racing control)	3 weeks @ 2130 m or 2320 m	-	↑ 4%	≈ (race performance in preferred event)	Yes, however lead-in training not reported Minimal taper in load	Lowest intensity observed during first week of camp
Jensen *et al*. 1993	9 elite rowers	Yes	3 weeks @ 1822 m	≈	-	≈ (6 min all out rowing)	No	-
Levine *et al*. 1997	13 collegiate runners $\dot{V}O_{2max}$ = 65 $mL{\cdot}kg^{-1}{\cdot}min^{-1}$	Yes	4 weeks @ 2500 m	↑ 5%	↑ 9% (red cell volume)	≈	Yes ↑ volume and intensity vs. prior sea-level training during weeks 2 and 3 at altitude ↓ volume and intensity in week prior to LHTH	Lowest intensity and volume at altitude observed during first week of camp
Mizuno *et al*. 1990	10 XC skiers $\dot{V}O_{2max}$ = 72 $mL{\cdot}kg^{-1}{\cdot}min^{-1}$	No	2 weeks @ 2100 m	≈	-	↑ treadmill time to exhaustion – 17%	No	-

Study	Participants	Control group	Altitude exposure	$\dot{V}O_{2max}$	Hb_{mass}	Performance	Training quantification	Acclimatisation
Pugliese *et al*. 2014	2 Olympic champion athletes	No	3 weeks @ 2090 m	-	-	↑ 1-4% (race performance)	Yes ≈ absolute training intensity at altitude ↑ training volume at altitude vs. sea-level	High intensity sessions from day 2-3 at altitude
Rodriguez *et al*. 2015	31 elite swimmers	Yes	3 or 4 weeks @ 2320 m	≈	↑ 4-6%	↑ but ≠ with control group	Yes, however no volumes reported Little variation in week to week TRIMP	-
Svedenhag *et al*. 1991	5 elite runners $\dot{V}O_{2max}$ = 74 $mL \cdot kg^{-1} \cdot min^{-1}$	Yes	2 weeks @ 2000 m	≈	-	≈ (treadmill time to exhaustion)	-	-
Wachsmuth *et al*. 2013	45 elite swimmers	Yes (within subject design)	3 to 4 weeks @ 1360 or 2320 m	-	↑ 7% (2320 m) ↑ 4% (1360 m)	↓ 0 to 14 days post-altitude ≈ 15 to 25 days ↑ 25 to 35 days	-	-

↓ decrease/impaired, ↑ increase/improved, ≈ unchanged, CON = control group, TRIMP = training impulse, TT = time trial, acclimatisation = first week at altitude

2.5.2 TRAINING QUANTIFICATION METHODS

Several reviews have described the methods and applications of quantifying training load in sports (Borresen and Lambert, 2009; Lambert, 2012), monitoring training with respect to minimising non-functional overreaching, injury and illness (Halson, 2014), and objective and subjective measures of athlete well-being available to guide training and detect any progression towards negative outcomes (Saw *et al*. 2016). Irrespective of the quantification methods used, they can be defined as quantifying either external or internal training load (Impellizzeri *et al*. 2005; Halson, 2014; Saw *et al*. 2016). The external training load is an objective measure of the work an athlete completes either during training or competition (e.g., distance completed, total elevation gain, or running speed). Alternatively, the internal workload assesses the biological stress imposed by the training session and is typically defined by the disturbance in homeostasis of physiological and metabolic processes (Lambert, 2012). In a recent investigation studying the relationship between different training load methods and performance in cyclists (Sanders *et al*. 2017), measures integrating individual physiological characteristics (i.e. measures of internal load) had the strongest dose-response relationships with performance and submaximal aerobic fitness. It is important to emphasise that the external training load does not strictly measure the biological stress imposed by a given training session. In fact, two athletes may undertake an identical external training load but experience quite different internal loads, depending on their fitness, training background and genetic characteristics (Impellizzeri *et al*. 2005; Halson, 2014; Lambert, 2012). In this respect, quantifying external load alone is limited, as it may not be sensitive enough to detect individual responses to training. However, it is necessary to provide context for the physiological stress imposed by training.

Both external and internal load contribute to quantifying an athlete's actual training load, and a combination of both is the key for proper training monitoring (Halson, 2014).

Monitoring daily training load might contribute to optimise athlete development, given better training regulation and earlier detection of overtraining or injuries (Roos *et al*. 2013). Assessing the relationship between external load, internal load, and competition performance should enhance evaluation of stress/recovery balance and adjustment of individual training programs to optimise adaptation. A recent systematic review indicated the combination of subjective and objective data as the most promising approach to evaluate training load and responses to the training. Validated questionnaires or rating of perceived exertion (RPE), combined with physiological parameters such as heart rate, are often used on a daily basis and seem to provide the most reliable training-related information. From the coaches' perspective, training duration and mode, RPE, and personal remarks in the athletes' training diaries were considered to be essential information (Roos *et al*. 2013).

A recent case study described a similar approach of incorporating subjective and objective measures in monitoring elite runners during 21 days of altitude training (Sperlich *et al*. 2016). Training load of the subsequent training session was reduced if two or more of the 11 measured variables (including both objective; SpO_2, resting heart rate, body mass, and subjective measures; body and sleep perception), were outside the athlete's normal individual range. Participants' running speed at lactate threshold improved and no athlete showed any signs of a maladaptive response, indicating this approach may have been effective in modulating training as required under conditions of additional physiological stress.

Data relating to training loads and to athletes' responses and adaptations are of interest to athletes, coaches, and sport scientists. Training data, physiological monitoring and direct observation can have a positive motivational impact on the athlete by heightening awareness of their investment in time and effort, as well as others (e.g. coach and sports scientist), and encouraging a more systematic and goal-oriented approach to training (Hopkins, 1991). A systematic approach to training quantification also facilitates better training prescription, with

coaches able to modify training based on data, physiological measures of stress and direct observation. Sport scientists can undertake descriptive and experimental studies on training effects, performance prediction and enhancement, recovery, and injury prevention to identify outcomes relevant and easily implementable by coaches into their training environments. Mujika (2013) considers the information about training (i.e. training quantification) the most important information in training intervention studies, and lack of a precise description of the training contents, in terms of volume, intensity, and frequency before and during a training intervention to be, a substantial limitation. Therefore, precise information about training quantification is absolutely necessary, as manipulating the training program is the basis of many studies, and interpretation of findings from such research is difficult without it.

2.5.3 TRAINING QUANTIFICATION AND PERIODISATION – MISSING PIECES OF THE ALTITUDE PUZZLE?

Several controlled studies only report basic metrics such as overall training volume or duration (Adams *et al*. 1975; Gore *et al*. 1997; Bailey *et al*. 1998), making it difficult to determine all the factors that influence athletic performance and the timing of a peak-performance. In many cases, the performance outcomes of altitude training research, whether negative (Adams *et al*. 1975; Gough *et al*. 2012; Bejder *et al*. 2017) or positive (Levine and Stray-Gundersen, 1997; Bonne *et al*. 2014), could be explained by the training completed prior to these performances when adequately quantified. As an example, a recent placebo controlled, crossover, double-blinded study investigating the efficacy of six weeks of LHTL or sea-level training on performance in highly trained triathletes reported no differences between groups in power output during an incremental test, a 26 km time-trial test, three minute all out exercise and 30 second repeated sprint ability (Bejder *et al*. 2017). It was observed however that almost half of total training time was allocated to threshold and high intensity training, contravening the polarised model of training generally adopted by elite athletes, both at sea-level and altitude

(Tønnessen *et al*. 2014; Brocherie *et al*. 2017; Solli *et al*. 2017). In this respect, training monitoring and quantification may enhance the interpretation of research findings, allowing practitioners to make informed decisions on implementation of training interventions with their athletes.

With the abundance of conflicting results evident in the altitude training literature (Bonetti and Hopkins, 2009), as well as the lack of training information presented in many studies, it is perhaps not surprising that both coaches and scientists are conflicted regarding the best training strategies to employ during altitude camps (Baumann *et al*. 1994; Wilber, 2004), and the best time to compete after training at altitude (Chapman *et al*. 2014b). Recent studies and reviews concerning the efficacy of altitude training highlight the importance of training quantification and consistent training between parallel groups in explaining any findings, as well as the contribution towards improved performance of individualised and periodised training, maintenance of training intensity at altitude, and well managed training intensity distribution (Chapman *et al*. 1998; Friedmann-Bette, 2008; Robertson *et al*. 2010a; Chapman *et al*. 2014a; Brocherie *et al*. 2017; Robach *et al*. 2018).

2.6 TRAINING PERIODISATION DURING ALTITUDE TRAINING

2.6.1 ACCLIMATISATION STRATEGIES AT ALTITUDE

Common amongst many coaches' strategies is the presence of a low intensity first seven to 10 days of altitude training in order to facilitate the acclimatisation process (Wilber, 2004 – **Table 2.1, Figure 2.6**). The initial acclimatisation period is generally characterised by a reduction in training volume and intensity, relative to normal sea-level training. It is generally thought there should be no anaerobic training sessions during this phase of altitude training to minimise the risk of overreaching early in a camp (Lange, 1986). Additionally, the haematological response to altitude training may be limited by incorrect prescription of training loads (Ploszczyca *et al*. 2018). Increased resting cortisol and decreased testosterone concentrations have been observed in athletes following LHTH, likely due to a combination of hypoxic exposure and increased training load (Gore *et al*. 1998; Wilber *et al*. 2000). A significant reduction in the testosterone to cortisol ratio during altitude training may negatively affect erythropoiesis, especially at the commencement of acclimatisation (Berglund, 1992), however confirmatory research in this area is limited. Hence, the low intensity approach to acclimatisation is often seen in research studies investigating the effects of classic altitude training (Levine and Stray-Gundersen, 1997; Garvican *et al*. 2012), as well as during case reports in some elite athletes (Solli *et al*. 2017). An exception to this paradigm is observed in the study by Daniels and Oldridge (1970). Whilst limited in sample size and lacking a control group, the authors reported substantial performance improvements in five elite runners who completed four altitude sojourns at 2300 m elevation, interspersed with trips to sea-level during which they competed at sanctioned national and international races. Unfortunately, specific characterisation of training was not reported, however it was observed that normal sea-level training was conducted at altitude, and hard training sessions were included from the beginning of the altitude training camp, contravening the general consensus described above. It must be

noted, the participants were elite national team members, accustomed to altitude training. A similar pattern of training was reported in two Olympic champion athletes completing three weeks of LHTH three weeks prior to their gold medal performances, with high intensity sessions commencing on day two of LHTH (Pugliese *et al*. 2014). Interestingly, to prepare for the camp, the training volume of key sessions was reduced in the week preceding LHTH. An advantage of maintaining high intensity training during acclimatisation may be the avoidance of a substantial period of detraining at altitude. Given the importance of maintaining running speed/power output at altitude (Chapman *et al*. 1998), and the typical use of altitude training being for two to four weeks (Friedmann-Bette, 2008), completing a low intensity first seven to 10 days of training would mean a disproportionate part of the camp may be spent detraining. As such, there may be some merit in challenging the status quo regarding load control during the acclimatisation phase of altitude training.

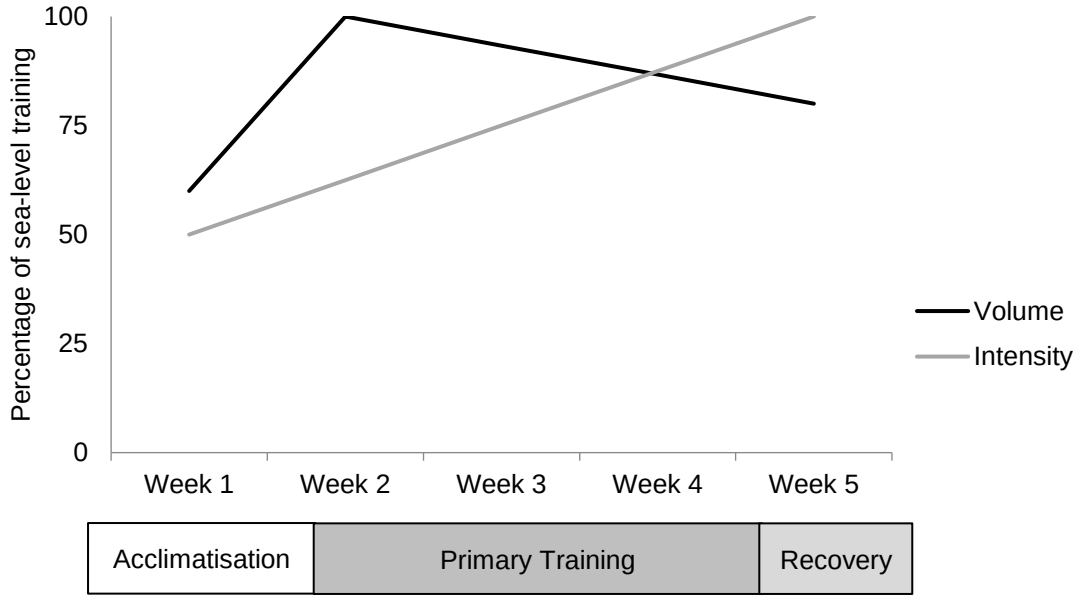


Figure 2.6. Load control during acclimatisation and training at altitude. Training volume and intensity are reduced substantially during the initial period at altitude. Adapted from Wilber, 2004.

2.6.2 OVERLOAD AND TAPER

Periods of intensified training are inherent to elite endurance athletes and intended to stimulate adaptations that may improve performance (Le Meur *et al*. 2014). Strategic periods of overload may occur at different phases throughout the season, including during the general preparation phase or immediately prior to competition to optimally prepare the athlete (Tønnessen *et al*. 2014; Aubry *et al*. 2014; Hellard *et al*. 2017). These training blocks can manifest in the form of specific training camps, and for elite endurance athletes, may occur at altitude.

Several scientific investigations have shown performance super-compensation is achieved when such a period of intensified training at sea-level is combined with a suitable period of taper in skiers, triathletes, kayakers, swimmers and cyclists (Breil *et al*. 2010; Garcia-Pallares *et al*. 2010; Aubry *et al*. 2014; Clark *et al*. 2014, Rønnestad *et al*. 2016, Hellard *et al*. 2017; Rønnestad *et al*. 2017). The duration of these "shock-microcycles" varies between investigations, however interventions as short as seven to 11 days have elicited improved performance (Breil *et al*. 2010; Clark *et al*. 2014; Rønnestad *et al*. 2017), as have other investigations examining longer periods of intensified training (Aubry *et al*. 2014; Rønnestad *et al*. 2016). In highly trained cyclists ($\dot{V}O_{2peak}$ = 63 $mL \cdot kg^{-1} \cdot min^{-1}$), seven days of daily high intensity training sessions, followed by a seven day period of recovery has been shown as superior in improving 20 km time trial performance, when compared to a control group completing their own training (Clark *et al*. 2014). A similar case-study in an elite cyclist ($\dot{V}O_{2max}$ = 89 $mL \cdot kg^{-1} \cdot min^{-1}$) yielded a season's best performance and victory in the national cup, following a seven day overload period and five day step-taper, suggesting even athletes approaching the limits of human performance may benefit from such a strategy (Rønnestad *et al*. 2017). In a longer-term investigation, highly trained cyclists completing three weeks of overload training (130% of regular training volume) experienced substantially greater

improvements in performance following a taper period than those maintaining regular training volumes, when symptoms of functional overreaching (transient reduced performance, high perceived fatigue) were not induced (Aubry *et al*. 2014).

Increases in training load can be achieved by manipulating the volume, frequency and intensity of training; however in running, each of these would result in an increase in the mechanical trauma associated with training, perhaps explaining the lack of related research in runners. Given the weight bearing nature of running, additional mechanical stress may increase the risk of injury which is counterproductive to maximising performance (Hausswirth *et al*. 2014; Raysmith and Drew, 2016). Training under hypoxic stress allows athletes to experience higher physiological loads than those achieved when completing equivalent training in normoxic conditions (Saunders *et al*. 2009a). As such one of the benefits of altitude camps may be their use as an additional physiological stressor to regular training conducted at sea-level, without a concomitant increase in mechanical load. Accordingly, a recent survey of elite British runners revealed athletes perceived one of the benefits of altitude training to be acquiring "a really good base and volume of training without as much stress on the legs" (Turner *et al*. 2018). Therefore, LHTH may be an attractive strategy whereby elite runners close to maximising their physiological potential can achieve the benefits conferred by residence at altitude, including stimulation of the erythropoietic pathway, alongside increases in training load necessary to stimulate adaptation and ultimately improve performance. Certain altitude training studies reporting improved performance upon return to sea-level have also featured athletes completing a period of increased training load during their altitude sojourns, suggesting this strategy may have merit (Levine and Stray-Gundersen, 1997; Bonne *et al*. 2014 – **Figure 2.7**). Collegiate runners completing an increase load of training at altitude compared to a preceding period at sea-level training achieved performance improvements on return to sea-level (Levine and Stray-Gundersen, 1997). Additionally, an Olympic champion cross country

skier increased training volume at altitude by 35% relative to prior sea-level training, with the athlete noting increased time available for rest and recovery during a training camp being an important factor contributing to their positive effects (Solli *et al*. 2017). Moreover, two Olympic champion athletes completed a higher relative intensity and volume of training during three weeks LHTH at 2090 m prior to their gold medal performances, compared to prior training at sea-level (Pugliese *et al*. 2014). However, beyond these observations, the efficacy of an intensified training period at altitude in runners is uncertain, largely due to the lack of training quantification observed across the altitude training literature.

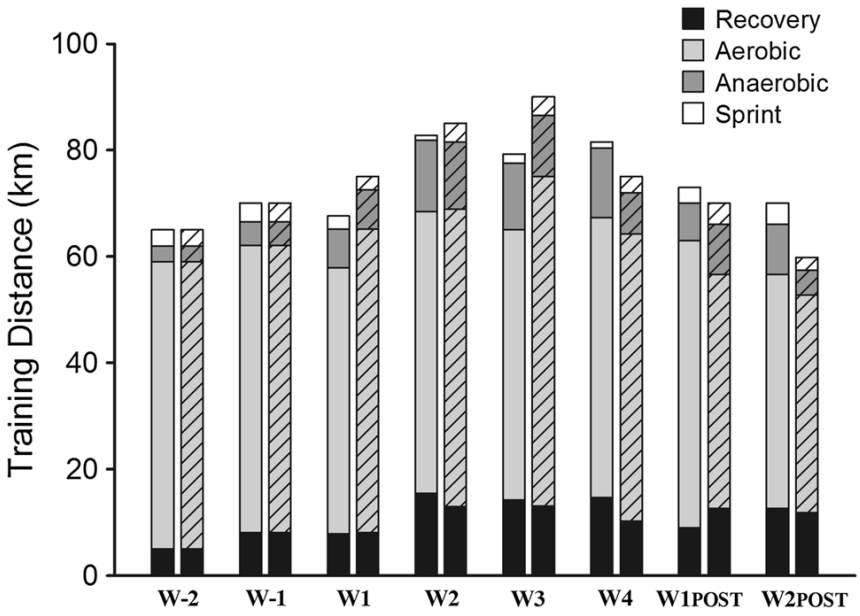


Figure 2.7. Training load and intensity distribution of elite swimmers completing LHTH (crossed bars). Sea-level performance over 200 and 3000 m tended to show greater improvements after LHTH than control group participants (clear bars) training at sea-level. Increases in volume and intensity of training occurred in weeks two and three at altitude. Reproduced from Bonne *et al*. 2014.

The success of short term overload strategies at sea-level (Clark *et al*. 2014; Rønnestad *et al*. 2017) may also translate to altitude training. Case reports of elite athletes (Daniels and Oldridge, 1970; Saunders *et al*. 2009b) have shown the effective use of repeated one to two week blocks of altitude training, interspersed with similar length periods at sea-level for

training and competition. Such short duration stays at altitude are typically viewed as insufficient to induce an increase in Hb_{mass}, nor stimulate other performance enhancing adaptations related to acclimatisation (Wilber *et al*. 2007). However, it may be that short periods at altitude, with the hypoxic stimulus used to intensify training, interspersed with periods of recovery at sea-level, may be an effective tool to stimulate adaptation and improve performance. In particular, the study of Daniels and Oldridge (1970) noted that normal sea-level training was continued at altitude, and hard training completed from the outset, suggesting that training would have been more intense given the altitude. However, the efficacy of this method is yet to be confirmed in controlled investigations, and it has been recommended this approach be restricted to altitude experienced, elite athletes only (Baumann *et al*. 1994; Saunders *et al*. 2009a).

The taper is a reduction in training load in the final days before competition, with the aim of optimising performance through maximising decreases in accumulated fatigue from training and retention or enhancement of physical fitness (Bosquet *et al*. 2007). The mechanisms underpinning performance improvements associated with the taper have been reviewed elsewhere, and include physiological, metabolic, haematological, neuromuscular and psychological changes (Mujika *et al*. 2004).

Various guidelines and strategies exist concerning tapering, with reductions in training load achievable through manipulations in training volume, intensity or frequency. A meta-analysis concluded an optimal tapering strategy should involve a 41-60% reduction in training volume over a period of two weeks (Bosquet *et al*. 2007). A study observing the tapering practices of elite runners (Spilsbury *et al*. 2015) reported a reduction in volume of 30-40% in the week preceding competition. The authors determined that amongst other factors, the nature of the taper was heavily influenced by the content of training undertaken prior (Spilsbury *et al*. 2015). Levels of fatigue preceding the taper also might influence the chosen strategy (Bosquet

et al. 2007), with athletes undertaking a large volume of running perhaps requiring a large reduction in training to alleviate accumulated fatigue (Spilsbury *et al*. 2015). Training completed prior to a taper may also potentiate its effects, with studies modelling tapering strategies suggesting that greater training loads prior to a taper would allow greater performance gains (Thomas and Busso, 2005), a finding confirmed in subsequent experimental studies conducted at sea-level (Aubry *et al*. 2014; Hellard *et al*. 2017).

Tapering would appear to have added relevance for athletes undertaking altitude exposures since training sessions completed in hypoxia evoke a higher physiological load than sessions completed in normoxia at the same absolute intensity (Mazzeo, 2008; Saunders *et al*. 2009a) leading to greater accumulated fatigue than when training at sea-level (Schmitt *et al*. 2018). Investigations observing minimal tapers in volume (5-10%) during three weeks of LHTH at 2300 m in elite swimmers (Gough *et al*. 2012; Rodriguez *et al*. 2015) have reported slower or unchanged performance immediately post altitude training. Such small reductions in volume may not be sufficient to dissipate the accumulated fatigue altitude training imposes and may explain the unclear performance findings. Information pertaining to the tapering practices of elite athletes at altitude is scarce in the literature, however it appears that such a practice, particularly following a period of intensified training often observed with altitude exposures, would result in performance improvements at sea-level.

2.6.3 MODIFICATIONS TO TRAINING SESSIONS

It has been proposed that due to the reduced oxygen availability at altitude and subsequent reduction in oxygen transport and uptake, some elite athletes are unable to maintain the training velocities required for competitive fitness, which may lead to undesirable performance outcomes upon return to sea-level (Chapman *et al*. 1998). Impaired performance in middle-distance and distance events (800 m to marathon), as well as during aerobic interval

training sessions at altitude is frequently reported (Péronnet *et al*. 1991; Levine and Stray-Gundersen 1997; Deb *et al*. 2018). Modifications to the prescription of training sessions are recommended to help facilitate the maintenance of exercise performance. For instance, it is suggested that recovery intervals for high intensity training sessions be increased at altitude to help maintain running velocity and oxygen flux (Baumann *et al*. 1994; Wilber, 2004; Saunders *et al*. 2009a). It has been reported in female cyclists completing repeated sprint interval training with work to rest ratios of 1:3, 1:2 and 1:1 in normoxia and hypoxia, that performance was not compromised by hypoxia with a 1:3 ratio, yet relatively more depressed when shorter recoveries were provided (Brosnan *et al*. 2000). Moreover, to avoid a reduction in race specific fitness, it is recommended athletes should undertake a series of shorter race-pace efforts where velocity is not compromised and for which they have longer recoveries than at sea-level to help maintain speed (Saunders *et al*. 2009a). With acclimatisation and partial restoration of $\dot{V}O_{2max}$ at altitude, the duration of interval efforts can be increased and/or recoveries decreased (Saunders *et al*. 2009a). Additionally, athletes may travel to a lower altitude if feasible to help maintain sea-level training intensity and levels of oxygen flux, with this approach traditionally advocated for all high intensity training sessions (Levine and Stray-Gundersen, 1997).

The reduction in the rate of oxygen uptake to steady state at altitude effectively increases the anaerobic contribution to exercise at all distances (Fulco *et al*. 1998). Whether this increase is sufficient to maintain exercise performance in hypoxia appears to be dependent on duration of exercise. At sprint intensities, the anaerobic contribution to exercise increases sufficiently at altitude to maintain performance in exercise bouts less than 60 seconds in duration (McLellan *et al*. 1990; Weyand *et al*. 1999; Ogawa *et al*. 2007). During exhaustive treadmill exercise at severe intensities (110-120% $\dot{V}O_{2max}$ – similar to 1500 m running race pace) in normoxia and hypoxia, it was observed that the anaerobic contribution to exercise did not increase, however time to exhaustion and accumulated oxygen uptake were significantly

reduced in hypoxia compared to normoxia. (Friedmann *et al*. 2007). The authors concluded that performance impairment during all-out exercise of 2-3 minutes duration was due to the reduction in the aerobic capacity in hypoxia (Friedmann *et al*. 2007). The protocol chosen by the investigators in this study was designed to induce maximal accumulated oxygen deficit (MAOD) in normoxia, therefore the capacity to increase the anaerobic contribution in hypoxia was not present. In contrast, shorter duration (sprint training), or lower intensity (threshold and $\dot{V}O_{2max}$ training) exercise also relevant to middle-distance and distance runners (Billat, 2001; Tjelta, 2016) do not elicit maximal anaerobic contributions in normoxia, and therefore allow for an increased anaerobic contribution and thus maintained performance at altitude (Weyand *et al*. 1999). Therefore, whilst longer duration exercise is clearly impaired at moderate altitudes (Péronnet *et al*. 1991; Deb *et al*. 2018), there may be certain combinations of duration and intensity of exercise that allow performance to remain relatively unimpaired, and further, athletes may gain an advantage by remaining at moderate altitudes to perform these high intensity sessions. Supporting this, an investigation concerning repeated sprint training completed in either normoxia or hypoxia reported improved blood perfusion, better waste metabolite removal and improved anaerobic glycolytic activity following training in hypoxia only (Faiss *et al*. 2013a). However, the efficacy of completing aerobic intervals in moderate hypoxia whilst residing at sea-level (intermittent hypoxic training) with respect to enhancing sea-level performance is unclear (Faiss *et al*. 2013b). Further understanding is therefore required concerning the physiological responses to exercise in hypoxia at the variety of high intensities at which middle-distance and distance athletes are required to train (i.e. threshold, $\dot{V}O_{2max}$, race pace, sprint). Such knowledge would help optimise intensity specific modifications to training sessions (e.g. stay high or descend to lower altitude, increase recoveries, modify pace) during natural altitude camps.

2.6.4 INDIVIDUALISATION OF TRAINING

It is well recognised that individuals may adapt differently to a given dose of endurance training (Vesterinen *et al*. 2016). Factors such as genetics, pre-training autonomic activity, training status, training program characteristics (e.g. high intensity or high volume, recovery time between sessions) and lifestyle factors (diet, sleep, psychological stress, level of habitual activity) are known to contribute to a given individual's response (Mann *et al*. 2014).

A recent study investigating the effect of three different periodisation models (i.e. traditional, reverse and mixed) on performance in well-trained cyclists revealed no significant differences between groups, however large individual differences in the adaptive response were observed (Sylta *et al*. 2016). Other studies have shown trained individuals may be more suited to either high volume or high intensity training (Bonafiglia *et al*. 2016; Vesterinen *et al*. 2016), with nocturnal heart rate variability proposed as a mechanism by which individual suitability to either of these programs may be determined (Vesterinen *et al*. 2016). Several studies have demonstrated alterations to training programs by manipulating either volume or intensity can have favourable outcomes in alleviating a non-response to a training stimulus. In highly trained cross-country skiers exhibiting no improvements in $\dot{V}O_{2max}$, maximal power and competition points following a training program favouring high volume and low intensity training, it was observed that significant improvements occurred in all these parameters after completing a subsequent block of training with a greater emphasis on high intensity training (Gaskill *et al*. 1999). Alternatively, in healthy but sedentary subjects completing one to five endurance training sessions per week during an initial six week program, it was observed that an initial non-response was eliminated by completing a subsequent six week program with two additional sessions per week (Montero and Lundby, 2017).

The issue of response and non-response to altitude training has been a contentious topic in the altitude training literature, with the focus of these discussions largely centred on individual variability in physiological responses induced by hypoxia, namely the increase in EPO concentration, and reduction in $\dot{V}O_{2max}$ at altitude (Chapman *et al*. 1998; Chapman, 2013). Alternatively, whilst previous investigations have been conducted largely in untrained, non-elite populations (Vesterinen *et al*. 2016; Bonafiglia *et al*. 2016; Montero and Lundby, 2017), they provide cause to consider manipulations of the training stimulus as a means of eliminating a "non-response" to altitude training. Just as a certain "hypoxic dose" may not be adequate to stimulate adaptation in an individual, a certain training program, or periodisation strategy may be sub-optimal (**Figure 2.8**), and have an equal, if not greater impact upon performance. Given that endurance athletes already engage in a high volume of training (Saunders *et al*. 2009b; Tønnessen *et al*. 2014; Solli *et al*. 2017), a further increase in volume may present an increased injury risk, and not provide an additional adaptive stimulus. For example, in highly trained swimmers, no improvements in sprinting or endurance performance, nor aerobic capacity were observed after a 10 day period where training volume was doubled, but intensity maintained compared to regular training (Costill *et al*. 1988). Given higher physiological loads can be achieved at altitude for the same mechanical stress and training intensity achieved at sea-level, individually prescribed high intensity training may elicit further adaptation for already highly trained athletes (Buchheit and Laursen, 2013).

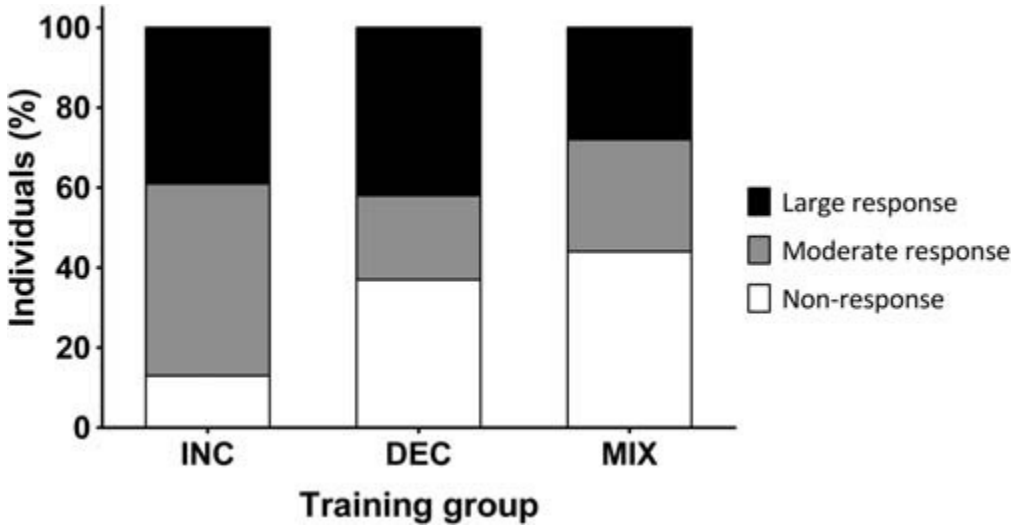


Figure 2.8. Individual responses to three different training periodisation models. Certain individuals may respond exceptionally well (large response) or poorly (non-response) to a given training stimulus. Evidence from experimental studies (Gaskill *et al*. 1999; Montero and Lundby, 2017) suggests a non-response to training may be alleviated by manipulating volume or intensity of training. Reproduced from Sylta *et al*. 2016.

The principle of individualising training is often neglected, with groups of athletes often prescribed the same or similar training programs, a practice evident in the altitude training literature (Adams *et al*. 1975; Gore *et al*. 1997; Heinicke *et al*. 2005; Frese and Friedmann-Bette, 2010; Bonne *et al*. 2014). Additionally, due to the higher relative intensity of exercise for a given workload characteristic of altitude (Mazzeo, 2008), other studies have uniformly decreased the absolute training intensity in all participants to keep the training stimulus consistent and isolate the effects of altitude exposure from training (Dill and Adams, 1971; Adams *et al*. 1975; Bailey *et al*. 1998; Brugniaux *et al*. 2006; McLean *et al*. 2015), despite the inter-individual variability that occurs amongst athletes to a dose of training, as well as in response to hypoxia (Chapman *et al*. 1998; Chapman, 2013; Mann *et al*. 2014; Montero and Lundby, 2017). Recommendations for sea-level performance enhancement utilising various altitude training modalities frequently advocate for the individualised prescription of training (Saunders *et al*. 2009a) and individual adjustment of training intensity at altitude to avoid overtraining or detraining (Friedmann-Bette, 2008), given the individual variation in reduction of aerobic performance capacity observed at altitude (Buskirk *et al*. 1967; Adams *et al*. 1975;

Chapman *et al*. 2011). Indeed, several case reports and controlled investigations of highly trained and elite athletes undertaking altitude training (both LHTH and LHTL) featuring individualised training programs have reported improved performance at sea-level at the group level (Levine and Stray-Gundersen, 1997; Stray-Gundersen *et al*. 2001; Rodriguez *et al*. 2015).

2.6.5 REPEATED EXPOSURES TO ALTITUDE

Whilst well controlled studies in the literature are necessary to establish the independent effects of hypoxia on physiological adaptation and performance, a potential shortcoming of these studies is that programs administered (e.g. training, nature of altitude exposure) may differ substantially from those used by elite athletes in practice (Buskirk *et al*. 1967; Adams *et al*. 1975; Bailey *et al*. 1998; Siebenmann *et al*. 2012; Bejder *et al*. 2017). Several altitude training studies have used single exposures in non-acclimatised altitude novices (Levine and Stray-Gundersen, 1997; Friedmann *et al*. 2005; Robach *et al*. 2018), whereas elite athletes typically utilise altitude training multiple times throughout a season in preparing for competition (Baumann *et al*. 1994; Wilber, 2004; Solli *et al*. 2017). Controlled experimental designs testing the efficacy of such an approach are rare in the literature (Robertson *et al*. 2010b), however several case-reports describing these practices in elite athletes with successful results exist (Daniels and Oldridge, 1970; Saunders *et al*. 2009b; Frese and Friedmann-Bette, 2010; Wachsmuth *et al*. 2013; Solli *et al*. 2017). Indeed, the repeated use of altitude exposures throughout a season is a practice recommended by several coaches and scientists (Baumann *et al*. 1994; Wilber, 2004; Saunders *et al*. 2009a; Millet *et al*. 2010).

In addition to multiple two to four week camps often utilised by athletes throughout different phases of a season, repeated short (five to 14 days) periods at altitude interspersed with similar duration stints at sea-level for training and/or competition may be used by athletes to enhance sea-level performance, with improvements of ~ 2% being reported in elite athletes

(Daniels and Oldridge, 1970; Saunders *et al*. 2009b). Such a strategy may allow athletes to achieve a higher overall dose of altitude exposure required to stimulate erythropoiesis and increase Hb_{mass}, whilst balancing the need to spend time at sea-level to maximise training quality, and eliminate fatigue associated with prolonged periods at altitude. Athletes restricted to low altitude (< 2000 m) training venues may find such a strategy particularly relevant; in elite 400 and 800 m runners, Hb_{mass} increased significantly by 5.1% after two, three week camps at 1300 and 1650 m, interspersed by three weeks at sea-level, with non-significant changes in Hb_{mass} reported after each individual camp (Frese and Friedmann-Bette, 2010).

Athletes frequently undertaking altitude training may experience a more rapid acclimatisation response during subsequent exposures, facilitating increased training quality relative to prior exposures (Millet *et al*. 2010), a particularly relevant point when altitude training is utilised immediately prior to competition, as it has been demonstrated that training quality at altitude is an important factor contributing to the success or failure of subsequent sea-level competition (Levine and Stray-Gundersen, 1997; Chapman *et al*. 1998). Anecdotally, coaches have suggested that experienced athletes who have previously undertaken altitude training tend to adapt faster and are able to achieve sea-level intensity in aerobic and anaerobic workouts (Baumann *et al*. 1994; Wilber, 2004). Moreover, it has been recommended that differences should exist between the training of novice and experienced athletes – specifically, novices should undertake a lower volume and frequency of high intensity training than experienced athletes, with greater reductions in pace compared to sea-level training, as well as having a longer acclimatisation period with no interval training at the commencement of the camp (Baumann *et al*. 1994; Issurin, 2007; Millet *et al*. 2010). Such practices may have implications for inducing the training response pathway of altitude adaptation facilitated by maintenance of near sea-level training intensity (Chapman *et al*. 1998), and therefore affect the performance response.

The physiological and molecular evidence supporting the repeated use of altitude training by elite athletes is emerging, suggesting a faster acclimatisation response is indeed present, via several pathways. An evaluation of pre-acclimatisation strategies utilised to minimise Acute Mountain Sickness (AMS) and improve performance upon acute exposure to 4300 m revealed that those interventions inducing the greatest degree of ventilatory acclimatisation were most successful in reducing AMS and improving exercise performance (Fulco *et al*. 2013). Ventilatory acclimatisation has also been shown as beneficial for high intensity exercise performance at altitude in elite cyclists (Townsend *et al*. 2016). Compared to measures taken at sea-level prior to 14 days altitude acclimatisation at 2700 m, a larger increase in the hypoxic ventilatory response, in combination with a higher exercise ventilation helped to maintain SaO_2 during exercise, and were associated with better performance at altitude (Townsend *et al*. 2016). Studies investigating the acclimatisation responses of native lowlanders upon acute exposure, after chronic exposure, and upon re-exposure following a period at lower altitudes to high altitudes (4300 to 5260 m) have shown that a degree of ventilatory acclimatisation is retained upon re-exposure to high altitude, even after a period of 8-21 days at lower altitude (Beidleman *et al*. 1997; Subudhi *et al*. 2014). In a separate study part of the AltitudeOmics project, it was demonstrated that red blood cells display a "metabolic memory" upon re-ascent to high altitude, consistent with improved physical performances in comparison to the first ascent (D'Alessandro *et al*. 2016). At a molecular level, reduced levels of erythrocyte equilibrative nucleoside transporter 1 (eENT1) resulting from initial hypoxia, established an erythrocyte hypoxic adenosine response for a second hypoxic exposure maintained upon re-ascent in humans or re-exposure to hypoxia in mice (Song *et al*. 2017), suggesting an erythrocyte hypoxic memory exists, which mediates faster and improved acclimatisation upon re-ascent associated with high levels of circulating adenosine (Song *et al*. 2017). Whether these adaptations are similar at moderate altitudes relevant to elite athletes and

remain after more prolonged periods at sea-level is yet to be determined. However, these studies provide experimental evidence supporting anecdotal observations that repeated altitude exposures facilitate a faster acclimatisation response, which may allow elite athletes to better perform necessary high intensity training at altitude to improve competitive fitness and ultimately sea-level performance. Meanwhile, athletes unaccustomed to altitude training (often observed within the literature) may be challenged in performing intensive training under hypoxic stress, and potentially detrain.

2.7 CONCLUSION

The present review has examined: i) the physiological responses to altitude training, ii) the practice of altitude training in elite athletes and scientific literature examining its efficacy in improving sea-level performance, iii) factors affecting the performance response to altitude training, and finally iv) established training practices and how they may relate to altitude training.

Whilst the literature demonstrates a volume of research on classic altitude training, and the physiological and haematological adaptations it may confer, it is lacking regarding optimal training periodisation strategies that may be employed during altitude sojourns to improve performance in subsequent sea-level competition. Such strategies are of particular interest, given the importance of training in contributing to an athlete's performance. Physiological limitations to performance exist at altitude, suppressing some potentially beneficial adaptations that may result in performance impairment upon return to sea-level. Whilst it is well understood the reduced oxygen availability characteristic of hypoxia will adversely affect training intensity, less evident is which intensities (of the many) at which distance/middle-distance athletes are required to train are most affected, in particular high intensities (i.e. threshold, $\dot{V}O_{2max}$ and race pace). Despite scepticism regarding its efficacy within the scientific community, elite athletes continue to frequently and successfully use classic altitude training, which suggests that effective high intensity training can be completed at moderate altitudes, and negative effects may be mitigated. However, scientific understanding underpinning LHTH is lacking with respect to training, and specifically which intensities may require modification at altitude.

Performance results following altitude training have been reported equivocally in the literature, potentially due to the lack of training description observed in certain studies,

removing the context in which any performance results are considered. An absence of sound training periodisation strategies in other investigations may reduce their ecological validity when compared to how elite athletes typically employ altitude training, and reduce their applicability for practitioners. Training quantification and periodisation related aspects are infrequently discussed when assessing performance outcomes following altitude training, resulting in a lack of knowledge concerning how established practices such as intensified training, tapering and interval training apply to athletes undertaking altitude training sojourns. Consequently, further research in this area is necessary. Therefore, this thesis aimed to a) explore the physiological and performance responses during exercise at altitude at a variety of intensities, and b) examine how training periodisation during LHTH at low and moderate altitudes affects sea-level performance. The findings of this thesis will have relevance to coaches and practitioners seeking to optimise the training process during altitude exposure for sea-level performance enhancement.

3.0 STUDY ONE: TRAINING AT 2100 m ALTITUDE AFFECTS RUNNING SPEED AND SESSION RPE AT DIFFERENT INTENSITIES IN ELITE MIDDLE-DISTANCE RUNNERS.

Citation: **Sharma AP**, Saunders PU, Garvican-Lewis LA, Clark B, Stanley J, Robertson EY, Thompson KG. Training at 2100 m altitude affects running speed and session RPE at different intensities in elite middle-distance runners. *Int J Sports Physiol Perform*. 2017;12(Suppl 2):S2-147-S2-152.

3.i FORM E: DECLARATION OF CO-AUTHORED PUBLICATION CHAPTER

Declaration for Thesis Chapter [3]

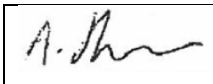
Declaration by candidate

In the case of Chapter [3], the nature and extent of my contribution to the work was the following:

Nature of contribution	**Extent of contribution (%)**
Study design, data collection and analysis, study write-up	70

The following co-authors contributed to the work.

Name	**Nature of contribution**	**Extent of contribution**	**Contributor is also a student at UC Y/N**
Philo Saunders	Assistance with design, data collection, proof reading, editing of drafts	7	N
Laura Garvican-Lewis	Assistance with design, proof reading, editing of drafts	5	N
Brad Clark	Assistance with design, proof reading, editing of drafts	5	N
Jamie Stanley	Assistance with data collection, proof reading, editing of drafts	3	N
Eileen Robertson	Assistance with data collection, proof reading, editing of drafts	3	N
Kevin Thompson	Assistance with design, proof reading, editing of drafts	7	N

Candidate's Signature		**Date** **12.6.2018**

Declaration by co-authors

The undersigned hereby certify that:

(1) The above declaration correctly reflects the nature and extent of the candidate's contribution to this work, and the nature of the contribution of each of the co-authors.
(2) they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least that part of the publication in their field of expertise;
(3) they take public responsibility for their part of the publication, except for the responsible author who accepts overall responsibility for the publication;
(4) there are no other authors of the publication according to these criteria;
(5) potential conflicts of interest have been disclosed to (a) granting bodies, (b) the editor or publisher of journals or other publications, and (c) the head of the responsible academic unit; and
(6) the original data are stored at the following location(s) and will be held for at least five years from the date indicated below:

Location(s)	Australian Institute of Sport Department of Physiology, Bruce, ACT Research Institute for Sport and Exercise, University of Canberra, Bruce ACT

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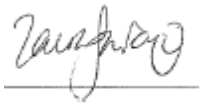
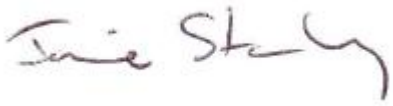
Signature 2		12.6.2018
Signature 3		12.6.2018
Signature 4		12.6.2018
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Signature 6		12.6.2018

3.1 ABSTRACT

Purpose: This investigation sought to determine the effect of training at 2100 m natural altitude on running speed (RS) during training sessions over a range of intensities relevant to middle-distance running performance.

Methods: In an observational study, 19 elite middle-distance runners (mean ± SD; Age, 25 ± 5 years; $\dot{V}O_{2max}$, 71 ± 5 $mL \cdot kg^{-1} \cdot min^{-1}$) completed either four to six weeks of sea-level training (CON, n = 7), or a five week natural altitude training camp living at 2100 m and training at 1400-2700 m (ALT, n = 12) following a period of sea-level training. Each training session was recorded on a GPS watch, and athletes also provided a session rating of perceived exertion (sRPE) score. Training sessions were grouped according to duration and intensity. Running speed ($km \cdot h^{-1}$) and sRPE from matched training sessions completed at sea-level and 2100 m were compared within ALT, with sessions completed at sea-level in CON describing normal variation.

Results: In ALT**,** RS was reduced at altitude compared to sea-level, with the greatest decrements observed during threshold and $\dot{V}O_{2max}$ intensity sessions (5.8 and 3.6% respectively). Velocity of low-intensity, and race pace sessions completed at a lower altitude (1400 m) and/or with additional recovery was maintained in ALT, though at a significantly greater sRPE (p = 0.04 and 0.05 respectively). There was no change in velocity or sRPE at any intensity within CON.

Conclusion: Running speed in elite middle-distance athletes is adversely affected at 2100 m natural altitude, with levels of impairment dependent on the intensity of training. Maintenance of RS at certain intensities whilst training at altitude can result in a higher perceived exertion.

3.2 INTRODUCTION

Live high train high (LHTH) or classic altitude training is used by endurance athletes to facilitate adaptation and improve performance (Daniels and Oldridge, 1970; Gore *et al*. 2007; Saunders *et al*. 2009b; Gore *et al*. 2013; Rodriguez *et al*. 2015). Training sessions completed in hypoxia evoke a higher physiological load than equivalent sessions completed in normoxia (Saunders *et al*. 2009a). However, the reduced partial pressure of oxygen and subsequent reduction in oxygen transport and uptake at race like intensities (Levine and Stray-Gundersen, 1997; Chapman *et al*. 1999; Chapman, 2013) affects both performance and perceived exertion. In Australian Rules footballers undertaking altitude training at 2100 m, rating of perceived exertion (RPE) scores were 13% higher than controls completing similar, predominantly aerobic training at sea-level (McLean *et al*. 2013a). Perceptual training data from endurance athletes at natural altitude is scarce, and perhaps would differ from footballers given that a larger volume of endurance training and higher frequency of altitude training is completed.

Impaired performances in 3000 m time trials have been reported (Chapman *et al*. 2011) for elite runners at natural altitude compared to sea-level, however the trials were performed within 48 hours of arrival at altitude, contravening the usual practice of allowing an acclimatisation period before progressing to intense training (Wilber, 2004). Nevertheless, slower running velocities at altitude have also been reported following acclimatisation (Levine and Stray-Gundersen, 1997), however with an improvement in performance over the course of altitude exposure (Buchheit *et al*. 2015; Chapman *et al*. 2016). Beyond these investigations, few data exist in scientific literature regarding the effect of moderate altitude on running speed (RS) in elite athletes.

It is important to consider the effects altitude may have on performance over the range of intensities at which distance runners train (Billat, 2001; Tønnessen *et al*. 2014; Tjelta, 2016), because some elite athletes may be unable to maintain the training velocities required for competitive fitness (Chapman *et al*. 1998). An investigation in elite cyclists competing in a stage race at altitude demonstrated mean maximal power output for intervals up to 60 seconds duration were minimally affected between 1000 to 2000 m altitude, compared to when completed in a race simulation near sea-level; however above 2000 m, performance was impaired by up to 10 per cent (Garvican-Lewis *et al*. 2015b). Thus, it remains unclear which training intensities may be adversely affected at altitude, and there is a lack of published data from elite athletes undertaking natural altitude training in a real world setting upon which to base recommendations regarding optimal practice.

Therefore, through observing a group of elite middle-distance runners participating in a LHTH altitude camp, this investigation sought to determine the effect of training at 2100 m natural altitude on RS during training sessions covering a range of intensities relevant to middle-distance running performance.

3.3 METHODS

Subjects

Nineteen elite middle-distance (800 to 5000 m) runners (13 males, 6 females; mean ± SD; Age, 25 ± 5 years; $\dot{V}O_{2max}$, 71 ± 5 $mL \cdot kg^{-1} \cdot min^{-1}$) participated in the investigation. Over two thirds were ranked in the top 15 for their event in Australia at the time of writing. Seven athletes (5 males, 2 females; Age, 23 ± 5 years; $\dot{V}O_{2max}$, 72 ± 6 $mL \cdot kg^{-1} \cdot min^{-1}$) completed four to six weeks of training at or near sea-level (CON). Training occurred following the domestic track season, when the athletes were in an endurance phase of training in preparation for the winter cross country season. Their training included low-intensity, threshold and $\dot{V}O_{2max}$ intensity training. Twelve athletes (8 males, 4 females; Age, 26 ± 5 years; $\dot{V}O_{2max}$, 70 ± 5 $mL \cdot kg^{-1} \cdot min^{-1}$) participated in a five week natural altitude training camp (ALT) in Flagstaff, USA (2100 m elevation) after four weeks of sea-level training, also following the domestic track season. Five athletes had previously trained in Flagstaff and the remainder had experienced altitude training at 1600 to 1800 m in Australia. These athletes were preparing for competition preceding and following LHTH, therefore their training also included middle-distance specific race pace sessions. Training was predominantly completed between 2100-2700 m except for a weekly race pace session, usually completed at 1400 m, though occasionally at 2100 m. All procedures and risks were explained to participants before they provided written consent to participate. Ethical approval for the investigation was granted by the institutional ethics committees and all procedures complied with the Declaration of Helsinki.

Design

The study was an observational design examining the training of elite middle-distance runners to compare responses between sea-level and altitude sessions in those athletes

completing LHTH. Participants' training sessions were designed by their coaches and were not manipulated or directly influenced as part of the study.

Procedures

Over the observational period, each athlete recorded their training on a GPS watch (Forerunner, Garmin International, Kansas, USA) to measure distance completed and RS during each training session. Additionally, for each training session, athletes provided a session rating of perceived exertion (sRPE) score on a modified Borg scale (Foster, 1998). Training sessions were arbitrarily categorised according to intensity and duration, as described in **Table 3.1**. In ALT, all training sessions completed within the three weeks prior to altitude training, and repeated at altitude following a week of acclimatisation, were considered for analysis to determine the effect of training at altitude on RS and sRPE. Where repeats of the same session occurred within the sea-level or altitude periods a mean value of those sessions was calculated and used for comparison. In CON, identical training sessions repeated within two weeks were included for analysis to illustrate normal variation in training. Matched training sessions were completed on similar terrain, topography and weather conditions. All race pace training was completed on standardised outdoor 400 m athletics tracks.

Statistical Analyses

Mean speed ($km \cdot h^{-1}$) and sRPE from matched training sessions were analysed within groups (sea-level vs. altitude in ALT, sea-level vs. sea-level in CON). Additionally, an integrated approach was used to present these data with sRPE divided by RS to determine the ratio of exertion to running velocity (i.e. RPE units per $km \cdot h^{-1}$ RS) at each training intensity within groups. Data were compared using paired t-tests, with effect sizes (Cohen's *d*) and percentage changes also calculated along with 90% confidence limits (CL). The individual response expressed as a percent was calculated as the square root of the difference in the

variance in the change scores between the CON and ALT groups (Hopkins, 2015). The standardised mean effect (Cohen's *d*) was also calculated by dividing the change scores and individual responses by the baseline SD (threshold: 1.3 $km \cdot h^{-1}$, $\dot{V}O_{2max}$: 1.4 $km \cdot h^{-1}$, race pace: 1.8 $km \cdot h^{-1}$) (Hopkins, 2015). All other variables are displayed as mean ± standard deviation (SD) unless otherwise stated and alpha was set at $p \leq 0.05$.

Table 3.1. Characterisation and examples of training sessions at different intensities

	Low-intensity	**Threshold**	**$\dot{V}O_{2max}$**	**Race pace**
Description	Continuous low intensity running at 2100-2700 m	Continuous running and fartlek sessions from 10 to 30 min performed around lactate threshold at 2100 m	Longer intervals from 1 to 6 min around velocity at $\dot{V}O_{2max}$ at 2100 m	Intervals 2 min or shorter at middle-distance race pace or faster –completed in Flagstaff at 2100 m or Sedona at 1400 m
Sessions	Continuous running > 70 min	3 km threshold 30 min threshold 4 x 5 min on 2 min jog 1, 2, 3, 4, 3, 2, 1 min on 1 min float – 22 min 20 min "Mona" fartlek – 4 x 90 s, 4 x 60 s, 4 x 30 s, 4 x 15 s with equal float 6 x (20 s hard, 40 s float, 40 s hard, 80 s float) - 18 min	4 to 6 x 1 km on 4:30/5:00 cycle 4 x 1600 m with 2 min recovery 3 x 3, 2, 1 min with equal walk/jog recovery 16 x 400 m on 2 min cycle 1600 m tempo, 5 min recovery, 800 m fast, 5 min recovery, 1600 m tempo (1600s) 3 x 1000 m, 100 jog, 500 m (1000s) 3 x (6 x 200 m on 1 min recovery, 800 m, 3:00 recovery) (800s)	2 x 3 x 400 m with 60 s recovery and 5:00 between sets (1400 m) 1600 m tempo, 5 min recovery, 800 m fast, 5 min recovery, 1600 m tempo (800) (1400 m) 3 x 4 x 400 m with 30 s/45 s/60 s recovery at sea-level and 60 s/75 s/90 s at altitude. 5 min between sets (1400 m) 3 x (6 x 200 m on 1:00 recovery, 800 m, 3:00 recovery) (200s) (2100 m) Taper session - 8 x 200 m with 1:30 recovery (2100 m)

3.4 RESULTS

Change in RS at altitude

The changes in RS across the spectrum of training intensities in this investigation are shown in **Table 3.2**. No differences were observed for RS in CON. In ALT, RS was reduced at altitude overall, [(19.1 ± 3.9 to 18.5 ± 3.9 km·h^{-1}, $p < 0.001$; d = -0.15 (-0.50, 0.20); % change (CL) = 2.9 (-3.8, -2.1)], with the largest reductions occurring in threshold and $\dot{V}O_{2max}$ sessions (**Table 3.2**). RS was reduced by 1.0 ± 0.5, 0.7 ± 0.4 and 0.4 ± 0.6 km·h^{-1} during threshold, $\dot{V}O_{2max}$ and race pace training respectively in ALT, with changes of 0.1 ± 0.3 and 0.2 ± 0.4 km·h^{-1} observed for threshold and $\dot{V}O_{2max}$ respectively in CON. RS during low-intensity training and race pace sessions conducted at 1400 m was maintained within 1% of sea-level values (**Table 3.2**). Individual variation for changes in RS at different intensities is shown in **Figure 3.1**. Change scores expressed with their individual responses at the three high intensities examined are shown in **Table 3.3.**

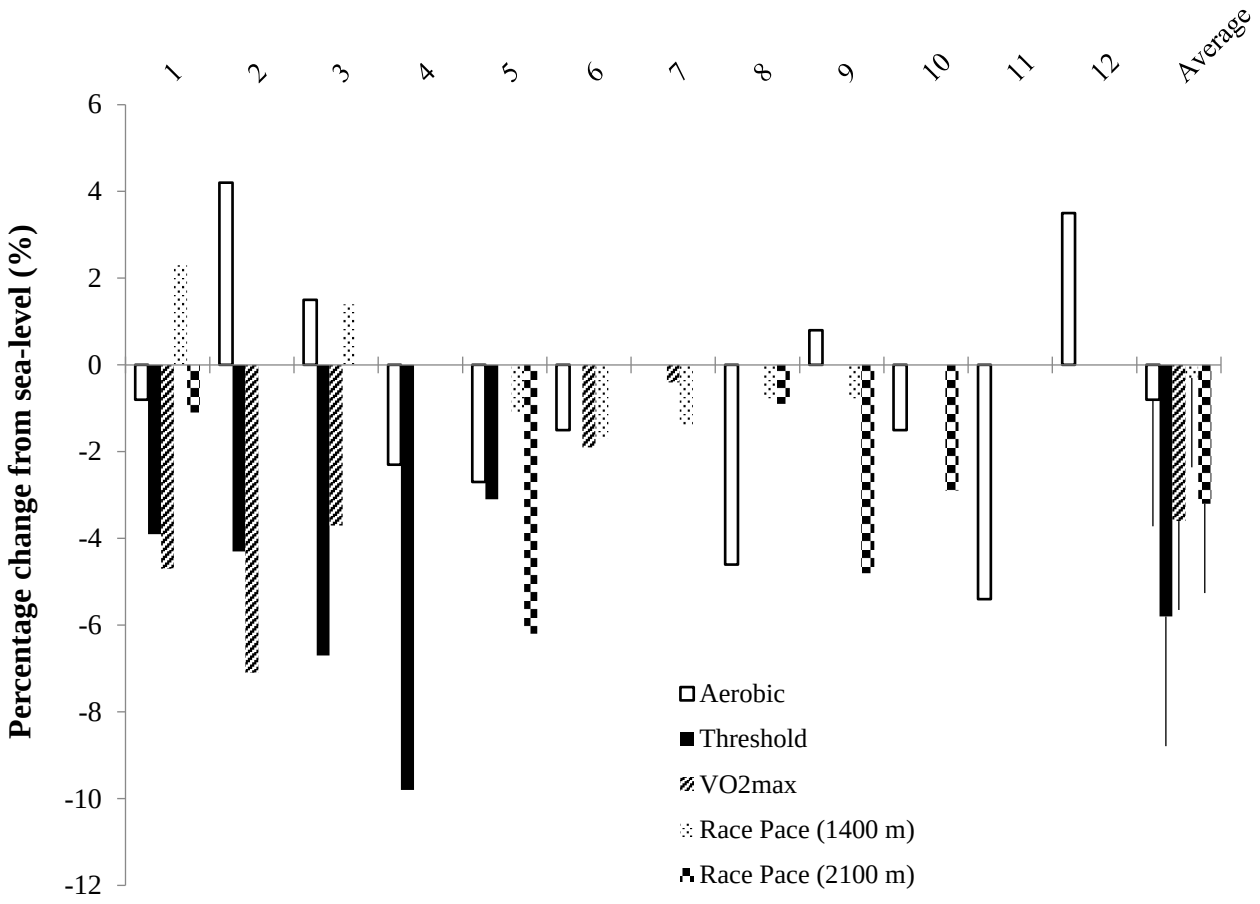


Figure 3.1. Individual changes in running speed at different training intensities. Upward bars indicate running speed at altitude was faster than at sea-level, and vice-versa for downward bars. Each number on the x-axis represents a unique individual; not all individuals completed suitable sessions across all training intensities within the observational period.

Table 3.2. Running speed changes at sea-level and altitude in elite middle-distance runners

	Session Type	*n* (total sessions, pairs)	RS (km·h^{-1}) (top) sRPE (bottom)		*p* value	Cohen's *d* effect size (90% CLs)	% change (90% CLs)	Exertion/velocity ratio		*p* value	Cohen's *d* effect size (90% CLs)	% change (90% CLs)
			Sea-level	Sea-level (CON)/Altitude (ALT)				Sea-level	Sea-level (CON)/Altitude (ALT)			
CON	**Low-intensity**	14, 7	14.1 ± 0.3	14.2 ± 0.6	0.24	0.20 (-0.68, 1.08)	1.2 (-0.4, 2.8)	0.32 ± 0.08	0.31 ± 0.07	0.25	- 0.13 (-1.00, 0.76)	- 4.2 (-10.7, 2.3)
			4.6 ± 1.1	4.4 ± 1.2	0.46	- 0.15 (-1.02, 0.74)	- 3.0 (-10.1, 4.1)					
	Threshold	22, 11	17.8 ± 1.4	17.9 ± 1.5	0.12	0.07 (-0.64, 0.77)	0.7 (-0.1, 1.5)	0.43 ± 0.05	0.43 ± 0.06	0.49	0.00 (-0.70, 0.70)	1.5 (-1.9, 4.9)
			7.6 ± 0.7	7.7 ± 1.0	0.34	0.12 (-0.59, 0.81)	2.3 (-1.6, 6.2)					
	$\dot{V}O_{2max}$	14, 7	19.5 ± 1.3	19.7 ± 1.5	0.33	0.13 (-0.78, 1.05)	0.8 (-1.0, 2.4)	0.37 ± 0.04	0.38 ± 0.05	0.36	0.22 (-0.67, 1.09)	3.4 (-2.4, 9.2)
			7.1 ± 0.8	7.4 ± 0.9	0.17	0.35 (-0.55, 1.22)	4.1 (-0.5, 8.7)					
ALT	**Low-intensity**	66, 11	14.1 ± 0.5	14.0 ± 0.6	0.41	- 0.20 (-0.90, 0.50)	- 0.8 (-2.4, 0.8)	0.31 ± 0.07	0.36 ± 0.07	0.02	0.71 (-0.03, 1.41)	17.6 (5.8, 29.5)
			4.4 ± 1.0	5.0 ± 0.9	0.04	0.66 (-0.08, 1.36)	17.0 (4.4, 29.7)					
	Threshold	24, 12	18.2 ± 1.3	17.1 ± 1.6	0.00002	- 0.68 (-1.37, 0.01)	- 5.8 (-7.3, -4.3)	0.37 ± 0.05	0.40 ± 0.05	0.04	0.60 (-0.11, 1.26)	9.4 (3.2, 15.7)
			6.8 ± 1.2	6.9 ± 1.3	0.50	0.14 (-0.54, 0.80)	3.0 (-3.0, 9.1)					
	$\dot{V}O_{2max}$	18, 9	19.7 ± 1.4	19.0 ± 1.2	0.001	- 0.54 (-1.33, 0.25)	- 3.6 (-4.9, -2.4)	0.37 ± 0.07	0.42 ± 0.05	0.03	0.82 (-0.02, 1.59)	18.6 (5.6, 31.6)
			7.2 ± 1.5	8.0 ± 1.1	0.09	0.61 (-0.21, 1.37)	14.3 (1.6, 27.0)					
	Race pace @ 1400 m	14, 7	23.4 ± 1.9	23.4 ± 2.1	0.74	- 0.02 (-0.90, 0.86)	- 0.3 (-1.3, 0.7)	0.30 ± 0.06	0.37 ± 0.06	0.06	1.17 (0.15, 2.04)	29.8 (5.7, 54.0)
			7.0 ± 1.8	8.6 ± 1.2	0.05	1.04 (0.05, 1.91)	29.3 (5.7, 53.0)					
	Race pace @ 2100 m	10, 5	24.8 ± 1.8	24.0 ± 1.3	0.04	- 0.48 (-1.54, 0.57)	- 3.2 (-5.1, -1.4)	0.28 ± 0.04	0.27 ± 0.06	0.72	- 0.20 (-1.22, 0.87)	- 3.1 (-15.4, 9.2)
			7.0 ± 1.1	6.6 ± 1.6	0.48	- 0.29 (-1.31, 0.78)	- 6.1 (-18.8, 6.6)					

CON = control group, ALT = altitude group, CLs = confidence limits

Change in sRPE at altitude

sRPE data are shown in **Table 3.2**. In ALT, Session RPE was greater at altitude compared to sea-level overall (6.6 ± 1.6 vs. 6.2 ± 1.7, $p = 0.03$; $d = 0.22$ [-0.17, 0.60]; % change = 8.7 [3.1, 14.3]) with the largest increases occurring at low-intensity, $\dot{V}O_{2max}$, and race pace sessions at 1400 m (**Table 3.2**). There were no significant differences in sRPE at any intensity in CON.

Change in 'exertion/velocity' ratio at altitude

Exertion/velocity ratio data are shown in **Table 3.2**. In ALT, the ratio was greater at altitude compared to sea-level overall [0.37 ± 0.07 vs. 0.33 ± 0.07, $p = 0.00003$; $d = 0.57$ (0.21, 0.92); % change = 15.2 (9.3, 21.1)]. Trivial to small differences were observed in CON.

Table 3.3. Individual responses for changes in running speed

	Change score	**Individual response (SD)**	**Standardised individual response (*d*)**
Threshold	- 5.8 % - 1.0 km·h^{-1}	2.6 % 0.4 km·h^{-1}	0.8 ± 0.3
$\dot{V}O_{2max}$	- 3.6 % - 0.7 km·h^{-1}	0.4 % 0.2 km·h^{-1}	0.5 ± 0.1
Race pace*	- 1.5 % - 0.4 km·h^{-1}	1.4 % 0.5 km·h^{-1}	0.2 ± 0.3

* Data from 1400 and 2100 m are pooled. Change scores are presented as a % change between sea-level and altitude (top) and difference in running speed (bottom)

3.5 DISCUSSION

The main finding of this investigation is RS in elite middle-distance runners across a range of training intensities is adversely affected at moderate altitude by up to 6%. The level of performance decrement differed depending on the intensity of the training session. A novel finding of the current study was an approximately 9% increase in sRPE at altitude; which, like RS, was specific to different intensities. Furthermore, training at altitude increased the exertion:velocity ratio by approximately 15%. There were no changes in RS, sRPE or exertion/velocity at any intensity within the control group.

In the current study, substantial individual variation in RS at altitude occurred at various training intensities (**Figure 3.1**), in accordance with previous findings (Chapman *et al*. 1998; Chapman *et al*. 2011). The variability in performance reduction at altitude is greatest when the aerobic contributions are highest (threshold > $\dot{V}O_{2max}$ > race pace, **Table 3.3**), perhaps unsurprising given the well characterised impairment of the aerobic system at altitude (Buskirk, 1967; Daniels and Oldridge, 1970; Dill and Adams, 1971; Fulco *et al*. 1998) as well as the individual variability this exhibits (Chapman *et al*. 1998; Chapman, 2013). Alternatively, the intensity at which "threshold" and "$\dot{V}O_{2max}$" sessions were performed was often prescribed by coaches based on their familiarity with their athletes' ability (i.e. subjectively), opposed to objectively based off lactate threshold or $\dot{V}O_{2max}$ testing in a laboratory. The subjective prescription of training may have led to inconsistencies and variability in the execution of sessions between individuals and thus contributed to the variability in performance observed.

Chapman and colleagues reported those athletes who maintained altitude training speeds during 1000 m intervals close to sea-level speeds were able to improve performance following altitude, however those who suffered large decrements in performance at altitude were slower in post-altitude time trial performance (Chapman *et al*. 1998). Consequently, it

has been suggested the maintenance of interval training speed, and therefore oxygen flux through the system is an important aspect for stimulating adaptation and improving performance (Chapman *et al*. 1998; Saunders *et al*. 2009a). In the current investigation, $\dot{V}O_{2max}$ intervals, and race pace intervals at 1400 m were slower than sea-level by 3.6 and 0.3% respectively, potentially small enough to maintain associated adaptations. However, high intensity intervals completed at 2100 m were more adversely affected than those at 1400 m (**Table 3.2**) which agrees with previous findings in cyclists (Garvican-Lewis *et al*. 2015b). To avoid a reduction in race specific fitness, athletes completed shorter race-pace efforts where velocity is not compromised, in some cases with longer recoveries between efforts than at sea-level (**Table 3.1**), and at a lower altitude to help maintain speed (Saunders *et al*. 2009a). Whilst fewer short intervals were completed at 2100 m than at 1400 m (**Table 3.1**), recoveries were maintained, perhaps accounting for the greater decrement in performance.

In the current investigation, athletes training at altitude were able to maintain sea-level speeds for low-intensity sessions and some race pace sessions (**Table 3.2**). With respect to low intensity aerobic training, submaximal $\dot{V}O_2$ has been shown to remain unchanged at increasing altitudes suggesting if submaximal training intensity can be maintained, oxygen flux through the system will also remain unchanged (Clark *et al*. 2007). However, as a result of maintaining training intensity, sRPE values at altitude were substantially higher. The largest decrement in RS at altitude occurred during threshold intensity training (**Table 3.2**). The reduction in longer duration (10 to 30 minutes) RS at threshold is likely the result of impairment of the aerobic energy system at higher altitudes. An increase in altitude is associated with arterial oxyhaemoglobin desaturation and impaired oxygen delivery during exercise which leads to a decrement in $\dot{V}O_{2max}$ in hypoxia, increasing linearly with altitude (Wehrlin and Hallén, 2006), particularly in highly trained endurance athletes (Chapman *et al*. 1999). Notably, the threshold sessions were performed in an identical manner to sea-level, whereas certain other sessions

were modified to allow for faster running. However, sRPE values were similar to those obtained when equivalent sessions were completed at sea-level, suggesting equivalent work rates can be achieved at altitude, but require higher levels of exertion. Whilst the maintenance of training speed and therefore oxygen flux at altitude is beneficial in terms of facilitating physiological adaptation (Chapman *et al*. 1998; Saunders *et al*. 2009a), the implications of higher perceptual ratings on training loads and athlete wellness is an important consideration.

Novel to the current investigation is the integration of exertion and velocity data when considering training at altitude (i.e. exertion:velocity ratio). The ratio describes the relationship between exertion (in this case on a 10 point RPE scale) and running speed in $km \cdot h^{-1}$. An increase in this ratio would suggest a greater level of exertion is required to maintain a certain running speed, a decrease would entail the exertion is easier. In a majority of training intensities completed at altitude (except race pace sessions @ 2100 m where athletes performed taper sessions at a lower perceived exertion than sea-level), the exertion/velocity ratio increased by 9 to 30%, suggesting athletes would perceive a much greater level of exertion for similar training sessions at altitude compared with sea-level. Large increases in the ratio were seen in $\dot{V}O_{2max}$ and race pace sessions (**Table 3.2**) indicating training at high intensities (i.e. race like intensity) is greatly affected at altitude due to changes in oxygen consumption (Chapman *et al*. 1999; Wehrlin and Hallén, 2006; Saunders *et al*. 2009a).

3.6 PRACTICAL APPLICATIONS

- Running speed during threshold and $\dot{V}O_{2max}$ intensity training sessions are adversely affected at 2100 m altitude by 6 and 4 % respectively, equating to 13 and 6 seconds per kilometre at the speeds at which elite middle-distance runners train
- Elite runners maintain intensity of long runs at altitude, but with a higher perceived exertion
- Intensity of race pace intervals can be maintained by allowing for additional/less intense recovery, or completing sessions at a lower moderate altitude (e.g. 1400 m) to allow for higher physiological loads and perceived exertion conferred by training in hypoxia
- Performing a session at altitude at the same running speed as when completed at sea-level can be up to 30% harder

3.7 CONCLUSION

Running speed in elite middle-distance athletes is adversely affected at 2100 m altitude, with levels of impairment dependent on the intensity of training. However, perceived exertion is increased compared to sea-level training at equivalent running speeds. Therefore, balancing the need to maintain intensity along with managing athlete training loads and fatigue is critical when planning periods of altitude training, and considering potential modifications to training to facilitate an optimal response.

4.0 STUDY TWO: NORMOBARIC HYPOXIA REDUCES $\dot{V}O_2$ AT DIFFERENT INTENSITIES IN HIGHLY TRAINED RUNNERS

Citation: **Sharma AP**, Saunders PU, Garvican-Lewis LA, Clark B, Gore CJ, Thompson KG, Périard JD. Normobaric hypoxia reduces $\dot{V}O_2$ at different intensities in highly trained runners. *Med Sports Sci Exerc.* 2018 Aug 7. doi: 10.1249/MSS.0000000000001745

4.i FORM E: DECLARATION OF CO-AUTHORED PUBLICATION CHAPTER

Declaration for Thesis Chapter [4]

Declaration by candidate

In the case of Chapter [4], the nature and extent of my contribution to the work was the following:

Nature of contribution	**Extent of contribution (%)**
Study design, data collection and analysis, study write-up	65

The following co-authors contributed to the work.

Name	**Nature of contribution**	**Extent of contribution**	**Contributor is also a student at UC Y/N**
Philo Saunders	Assistance with design, proof reading, editing of drafts	7	N
Laura Garvican-Lewis	Assistance with design, proof reading, editing of drafts	7	N
Brad Clark	Assistance with data collection, proof reading, editing of drafts	5	N
Christopher Gore	Assistance with design, proof reading, editing of drafts	5	N
Kevin Thompson	Assistance with design, proof reading, editing of drafts	5	N
Julien Périard	Assistance with data analysis, proof reading, editing of drafts	6	N

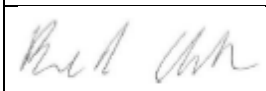
Candidate's Signature		**Date** **12.6.2018**

Declaration by co-authors

The undersigned hereby certify that:

(1) The above declaration correctly reflects the nature and extent of the candidate's contribution to this work, and the nature of the contribution of each of the co-authors.
(2) they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least that part of the publication in their field of expertise;
(3) they take public responsibility for their part of the publication, except for the responsible author who accepts overall responsibility for the publication;
(4) there are no other authors of the publication according to these criteria;
(5) potential conflicts of interest have been disclosed to (a) granting bodies, (b) the editor or publisher of journals or other publications, and (c) the head of the responsible academic unit; and
(6) the original data are stored at the following location(s) and will be held for at least five years from the date indicated below:

Location(s)	Australian Institute of Sport Department of Physiology, Bruce, ACT Research Institute for Sport and Exercise, University of Canberra, Bruce ACT

Signature 1		**Date** 12.6.2018
Signature 2		12.6.2018
Signature 3		12.6.2018
Signature 4		12.6.2018
Signature 5		12.6.2018
Signature 6		12.6.2018

4.1 ABSTRACT

Introduction: We sought to determine the effect of low and moderate normobaric hypoxia on oxygen consumption and anaerobic contribution during interval running at different exercise intensities.

Methods: Eight runners (age: 25 ± 7 years, $\dot{V}O_{2max}$: 72.1 ± 5.6 $mL \cdot kg^{-1} \cdot min^{-1}$) completed three separate interval sessions at threshold (4 x 5 min, 2 min recovery), $\dot{V}O_{2max}$ (8 x 90 s, 90 s recovery), and race pace (10 x 45 s, 1 min 45 s recovery) in each of; normoxia (elevation: 580 m, fraction of inspired oxygen [FiO_2]: 0.21, low (1400 m, 0.195) or moderate (2100 m, 0.18) normobaric hypoxia. The absolute running speed for each intensity was kept the same at each altitude to evaluate the effect of FiO_2 on physiological responses. Expired gas was collected throughout each session, with total $\dot{V}O_2$ and accumulated oxygen deficit calculated. Data were compared using repeated measures ANOVA.

Results: There were significant differences between training sessions for peak and total $\dot{V}O_2$, and anaerobic contribution ($p < 0.001$, $p = 0.01$ respectively), with race pace sessions eliciting the lowest and highest responses respectively. Compared to 580 m, total $\dot{V}O_2$ at 2100 m was significantly lower ($p < 0.05$), and anaerobic contribution significantly higher ($p < 0.05$) during both threshold and $\dot{V}O_{2max}$ sessions. No significant differences were observed between altitudes for race pace sessions.

Conclusion: To maintain oxygen flux, completing threshold and $\dot{V}O_{2max}$ training sessions at 1400 m simulated altitude appears more beneficial compared with 2100 m. However, remaining at moderate altitude is a suitable option when increasing the anaerobic contribution to exercise is the desired outcome.

4.2 INTRODUCTION

Elite endurance athletes frequently reside and/or train at moderate altitudes to improve sea-level performance via adaptations acquired from the hypoxic stimulus (Saunders *et al*. 2009a). General recommendations regarding training at altitude suggest a reduction in absolute running speed, especially during the initial phase of a camp, to minimise the risk of overtraining and facilitate the acclimatisation process (Baumann *et al*. 1994; Wilber, 2004). Previous research has shown however, that the reduction in running speed at altitude is coupled with a lower oxygen flux, resulting in a potential deconditioning effect impairing subsequent sea-level performance (Levine and Stray-Gundersen, 1997; Chapman *et al*. 1998). As such, the maintenance of absolute exercise intensity as well as oxygen flux are likely to be important factors contributing to improved sea-level performance following altitude training (Brosnan *et al*. 2000).

The physiological responses associated with submaximal and maximal aerobic exercise in hypoxia are well described (Fulco *et al*. 1998; Mazzeo, 2008). Due to the reduced partial pressure of oxygen at natural altitude, or reduced fraction of inspired oxygen in simulated altitude environments, maximal oxygen consumption ($\dot{V}O_{2max}$) is reduced (Wehrlin and Hallén, 2006; MacInnis *et al*. 2015). The decline of $\dot{V}O_{2max}$ (~ 6% per 1000 m) increases with altitude (Wehrlin and Hallén, 2006) and has been observed in elite athletes at altitudes as low as 580 m (Gore *et al*. 1996).

Greater physiological and metabolic adjustments are required to maintain homeostasis and performance for a given absolute workload when exercise is performed at altitude compared with sea-level (Mazzeo, 2008). Due to the reduction in $\dot{V}O_{2max}$, training sessions in hypoxia at submaximal intensities are completed at a higher relative intensity than equivalent sessions in normoxia. Whilst submaximal $\dot{V}O_2$ has been shown to remain unchanged at higher

altitudes (Clark *et al*. 2007), this is at an increased overall physiological cost, as oxygen transport to the working muscles is maintained by increasing cardiac output (as a function of increased heart rate) and muscle blood flow, compensating for the reduction in arterial oxygen content (Fulco *et al*. 1998; Mazzeo, 2008).

At severe exercise intensities, to compensate for the reduced oxygen availability in hypoxia, the anaerobic contribution to exercise increases to maintain performance during sprint exercise (McLellan *et al*. 1990; Weyand *et al*. 1999; Ogawa *et al*. 2007). We have recently demonstrated (Sharma *et al*. 2017) that running speed during self-paced intervals (lactate threshold, $\dot{V}O_{2max}$ and middle-distance race pace) at 2100 m was reduced to different degrees. The greatest reductions were observed in those with the greatest aerobic contributions (i.e. threshold and $\dot{V}O_{2max}$), confirming previous findings in elite cyclists (Brosnan *et al*. 2000), suggesting that physiological responses attempting to compensate for the reduced oxygen availability at altitude are insufficient to maintain performance at certain intensities more so than others. However to our knowledge, studies investigating these responses at multiple training intensities having not been conducted. Furthermore, whilst the physiological responses to single bouts of exercise in hypoxia are generally well understood, studies investigating physiological responses during interval training (i.e. repeated bouts) are limited to severe exercise intensities (Ogawa *et al*. 2007; Feriche *et al*. 2007).

Training with higher levels of oxygen flux characteristic of lower altitudes is typically viewed as beneficial in facilitating adaptation and improved performance (and a key reason for the recommendation of "Live High Train Low" over "Live High Train High") (Levine and Stray-Gundersen, 1997; Chapman *et al*. 1998). However, remaining at moderate altitudes for high intensity training may result in greater levels of muscle deoxygenation, which has been proposed to stimulate muscular adaptations (e.g. improved muscle pH regulation, buffer capacity and anaerobic glycolytic activity, and increased muscle blood perfusion,

mitochondrial volume and capillary density) (Mizuno *et al*. 1990; Vogt *et al*. 2001; Gore *et al*. 2007; Lundby *et al*. 2009; Faiss *et al*. 2013a). Such adaptations may also enhance competitive performance during middle-distance and distance events, where both aerobic and anaerobic contributions to performance are relevant (Gastin, 2001). In practical terms, knowledge of how physiological responses differ when training at different intensities would inform intensity specific modifications to training sessions (e.g. stay high or descend to lower altitude, increase recoveries, modify pace) designed to maintain exercise intensity and oxygen flux, or alternatively amplify the anaerobic contribution and lower oxygen flux during interval training at altitude.

We therefore sought to determine the effect of low (1400 m) and moderate (2100 m) normobaric hypoxia on $\dot{V}O_2$, anaerobic contribution and other physiological parameters during interval training sessions at three different intensities in highly trained runners. To ensure any physiological differences observed were due to hypoxia, and not a lower self-paced intensity of exercise (Brosnan *et al*. 2000; Sharma *et al*. 2017), constant work load intervals were prescribed to athletes at the same absolute running speed for each training intensity. Compared to exercise in normoxia (580 m), we hypothesised that $\dot{V}O_2$ would be lower, and anaerobic contribution higher across all training intensities at simulated altitudes of 1400 m and 2100 m.

4.3 METHODS

Subjects

Eight highly trained male runners and triathletes (age: 25 ± 7 years, body mass: 71 ± 5 kilograms) participated in the investigation. The investigation took place during the pre-competition phase of the season, with participants regularly engaged in training consisting of continuous and interval running five to seven days a week, and habituated to running on a motorised treadmill. None of the participants had prolonged exposure to altitude in the 12 months prior to participating in the investigation. All procedures and risks were explained to participants before they provided written informed consent to participate. Ethical approval for the investigation was granted by the institutional ethics committee (University of Canberra – Human Research Ethics Committee ref. no. 16-233) and all procedures complied with the Declaration of Helsinki.

Study design

To assess the effect of low and moderate hypoxia on the physiological responses to interval running at three different intensities compared to normoxia, an unblinded, randomised, repeated measures design was employed. The simulated altitudes chosen for training sessions are typically used by elite endurance athletes during both live high train high (LHTH) and live high train low (LHTL) altitude training (Carr *et al*. 2015; Sharma *et al*. 2017). The altitude for each training session was known to participants to preserve ecological validity, as elite athletes engaged in altitude training are aware of the altitude at which they are exercising. Furthermore, it was determined that the physiological impact of blinding would be inconsequential as the running speeds were constant across altitudes and based on the incremental exercise test in normoxia (580 m).

The investigation took place in Canberra, Australia (elevation: 580 m), and participants were required to complete 12 exercise sessions on a motorised treadmill (pulsar 3p, h/p/cosmos, Germany) over a five week period, with all sessions taking place in an environmental chamber (ATS-1000BLHP, Altitude Training Systems, Lidcombe, Australia). The first week involved completing three incremental exercise tests in normoxia (elevation: 580 m, FiO_2: 0.21), as well as low (1400 m, FiO_2: 0.195) and moderate (2100 m, FiO_2: 0.18) normobaric hypoxia, to characterise $\dot{V}O_{2max}$, velocity at $\dot{V}O_{2max}$ ($v\dot{V}O_{2max}$), 4 mM lactate threshold (LT), and to prescribe running speeds for the subsequent interval sessions. Over the following three to four weeks participants completed three different interval training sessions - threshold (4 x 5 min with 2 min recovery), $\dot{V}O_{2max}$ (8 x 90 s with 90 s recovery), and race pace (10 x 45 s with 1 min 45 s recovery) at each of the altitudes, for a total of nine sessions. These sessions were completed in a randomised order determined individually for each participant. Participants stood at rest during all recovery periods. Running speeds for these sessions were calculated from the incremental exercise test completed at 580 m; threshold, $\dot{V}O_{2max}$ and race pace sessions were completed at 4 mM lactate threshold speed, $v\dot{V}O_{2max}$, and 110% of the $v\dot{V}O_{2max}$ respectively. Participants maintained regular training commitments during the investigation but were instructed to refrain from strenuous exercise for the 12 hours preceding each trial. For each participant, testing sessions were conducted at a similar time of day, with trials separated by at least 48 hours.

Baseline trials – Incremental exercise testing

Each incremental exercise test to exhaustion comprised of four submaximal stages completed at 12, 14, 16 and 18 $km \cdot h^{-1}$ (0% gradient) for determination of $\dot{V}O_2$ and capillary blood lactate concentration ([BLa]), immediately followed by an incremental ramp to exhaustion to determine $\dot{V}O_{2max}$. The initial submaximal stage at 12 $km \cdot h^{-1}$ was four minutes in length, with the subsequent three stages each being three minutes in length. Three minute

stages have previously been shown to provide reliable and valid (in comparison to 10 minute stages) estimates of speed at running speed at 4 mM [BLa] (Weltman et al. 1990). Immediately following each stage a small capillary blood sample was taken from the fingertip, to measure [BLa] (Lactate Pro, Arkray, Kyoto, Japan). Following the completion of the fourth submaximal stage, the gradient of the treadmill was increased by 0.5% every 30 s, until the participant reached volitional exhaustion. A final capillary blood sample was taken 1 min after cessation of exercise to determine maximal [BLa]. Heart rate (Polar Electro, Kempele, Finland) was measured throughout the test. Expired gas was collected throughout both the submaximal and maximal portions of the test for determination of ventilation ($\dot{V}_E$), $\dot{V}O_2$, $\dot{V}CO_2$ and respiratory exchange ratio (RER), using a metabolic cart (True One 2400, ParvoMedics, USA). Prior to each test, the ParvoMedics system was calibrated with normoxic gas (20.93% O_2 and 0.04% CO_2) and a gas of known concentration (16.01% O_2 and 4.00% CO_2). A 3-liter syringe was used to calibrate flow. Submaximal $\dot{V}O_2$ for each stage was indicated by mean $\dot{V}O_2$ during the final minute, and $\dot{V}O_{2max}$ ($mL \cdot kg^{-1} \cdot min^{-1}$) as the highest 30 s value achieved during the incremental ramp portion of the test. We acknowledge that this may be taken to represent $\dot{V}O_{2peak}$ (Poole and Jones, 2017), as evidenced by the RER values obtained (**Table 4.1**), however we will refer to this as $\dot{V}O_{2max}$ throughout the manuscript for consistency and clarity in differentiating between peak $\dot{V}O_2$ obtained during the interval training sessions. Individual running speed at 4 mM [BLa] was calculated using freely available software (Newell *et al.* 2007), and $v\dot{V}O_{2max}$ was calculated from the running speed:$\dot{V}O_2$ relationship using the four submaximal speeds.

Experimental trials

Participants completed three different interval training sessions at each of the three altitudes, for a total of nine sessions. Prior to each trial, participants completed a 15 min standardised warm-up in normoxia including 10 min of low-intensity continuous running, some

stretches and mobility exercises, and strides. Participants then entered the environmental chamber for 10 min before commencing the interval session, and were fitted with a heart rate monitor, safety harness, nose-clip and Hans-Rudolph mouthpiece for collection of expired gas. To start the interval session, participants straddled the treadmill belt whilst it was brought up to speed, with the first interval starting with them lowering themselves onto the moving treadmill belt. At the end of each interval, participants lifted themselves clear of the treadmill belt where they remained straddling the moving belt for the duration of the recovery period. For safety purposes, the safety harness remained fitted throughout the session. $\dot{V}O_{2peak}$ (highest 15 s value), total $\dot{V}O_2$ and accumulated oxygen deficit (AOD) were measured for each interval, which was then averaged across the session. RER was taken as the mean value from the last 15 s of each interval for all intensities. Heart rate was measured throughout the test, and rating of perceived exertion (RPE) was obtained following each interval (Borg, 1970). After each interval during the threshold sessions, and every second interval during the $\dot{V}O_{2max}$ and race pace sessions, a capillary blood sample was taken from the fingertip to measure [BLa]. Additionally, RPE for each interval was divided by running speed to determine the ratio of perceived exertion to running velocity (i.e. RPE units per $km \cdot h^{-1}$ running speed) (Sharma *et al*. 2017).

Normobaric hypoxia

All trials were conducted in the same 100 m^3 environmental chamber. The low and moderate simulated altitudes were achieved through nitrogen injection (flow rate 1000 $L \cdot min^{-1}$, 89% nitrogen, 11% oxygen), creating a normobaric hypoxic environment. The room had an in-built barometric pressure compensation, with the percentage oxygen in the chamber adjusted to account for the ~ 10% lower barometric pressure compared to sea-level of Canberra (altitude, 580 m). The percentage of inspired oxygen for 580, 1400 and 2100 m were 20.94 ± 0.05%,

19.45 ± 0.06% and 18.00 ± 0.08% respectively. Mean temperature, pressure and relative humidity for the trials was 21.4 ± 1.5ºC, 708.4 ± 4.1 mmHg and 53.6 ± 11.4% respectively.

Calculation of accumulated oxygen deficit

The AOD (Medbo *et al*. 1988) arising from each interval was calculated as the difference between estimated oxygen requirements of the work achieved (derived from the running speed:$\dot{V}O_2$ regression for each individual athlete) and the total $\dot{V}O_2$ consumed during each interval. AOD and total $\dot{V}O_2$ for each interval completed during a session were summated to give total AOD and $\dot{V}O_2$ for the session. The relative aerobic and anaerobic contributions of each interval were calculated as the percentage of measured $\dot{V}O_2$ compared with the predicted $\dot{V}O_2$ and then averaged to give a value for the session.

Statistical analysis

All statistical calculations were performed using performed using the SPSS statistical package version 23 (IBM, New York, USA). Differences between altitudes for measures obtained during incremental exercise testing ($\dot{V}O_{2max}$, v$\dot{V}O_{2max}$, and 4 mM LT) were quantified using one-way ANOVA. To test changes in measured variables ($\dot{V}O_{2peak}$, V_E, RER, $\dot{V}O_{2peak}$ as % of altitude specific $\dot{V}O_{2max}$ [% $\dot{V}O_{2max}$], total $\dot{V}O_2$, AOD, aerobic contribution, anaerobic contribution, heart rate, RPE, exertion/velocity ratio and [BLa]) within training sessions (threshold, $\dot{V}O_{2max}$ and race pace) between altitudes (580 m, 1400 m and 2100 m) and over time (4, 8 and 10 intervals for threshold, $\dot{V}O_{2max}$ and race pace respectively), two-way (altitude x time) repeated measures ANOVA were performed. Data from submaximal speeds (12, 14, 16 and 18 $km \cdot h^{-1}$) during the incremental exercise test ($\dot{V}O_2$, V_E, RER, heart rate, [BLa]) were similarly compared using two-way (altitude x speed) repeated measures ANOVA. To compare between the three training sessions across the three altitudes, two-way (altitude x session) repeated measures ANOVA was also performed. ANOVA assumptions were verified

preceding all statistical procedures; however, none of the data violated the assumption of sphericity. Where significant effects were established, pairwise differences were identified using the Bonferroni post hoc analysis procedure adjusted for multiple comparisons. *P* values less than 0.05 were considered statistically significant. Effect size was measured using partial eta-squared (η^2) values with $\eta^2 > 0.06$ representing a moderate effect and $\eta^2 > 0.14$ a large effect. All values are expressed as means ± SD.

4.4 RESULTS

Incremental exercise testing

Summary data from the incremental exercise tests in each altitude are shown in **Table 4.1**. There were significant reductions in $\dot{V}O_{2max}$ at 1400 m (3.4 ± 2.5%, $p = 0.02$) and 2100 m (7.3 ± 2.4%, $p < 0.001$) compared with 580 m, with a significant reduction observed from 1400 m to 2100 m (4.0 ± 3.2%, $p = 0.046$). $\dot{V}O_2$ at each of the submaximal workloads (12, 14, 16 and 18 $km \cdot h^{-1}$) was not significantly different between altitudes ($p > 0.05$, **Figure 4.1A**). No significant differences were observed for running speed at 4 mM LT between altitudes, however $v\dot{V}O_{2max}$ was significantly lower at 2100 m compared to both 580 m and 1400 m (p = 0.003 and 0.004 respectively).

Table 4.1. Summary data from the maximal incremental exercise tests.

	580 m	**1400 m**	**2100 m**
$\dot{V}O_{2max}$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	72.1 ± 5.9	69.6 ± 5.8*	66.8 ± 5.3$^{*\#}$
RER	1.08 ± 0.03	1.08 ± 0.03	1.08 ± 0.07
4 mM lactate threshold ($km \cdot h^{-1}$)	17.5 ± 0.9	17.4 ± 0.7	17.0 ± 1.3
$v\dot{V}O_{2max}$ ($km \cdot h^{-1}$)	20.1 ± 1.3	19.9 ± 1.1	19.0 ± 1.0$^{*\#}$
110% $v\dot{V}O_{2max}$ ($km \cdot h^{-1}$)	22.1 ± 1.4	21.8 ± 1.2	20.9 ± 1.1$^{*\#}$

* $p < 0.05$ vs. 580 m, $^{\#}$ $p < 0.05$ vs. 1400 m. Data reported as mean ± SD

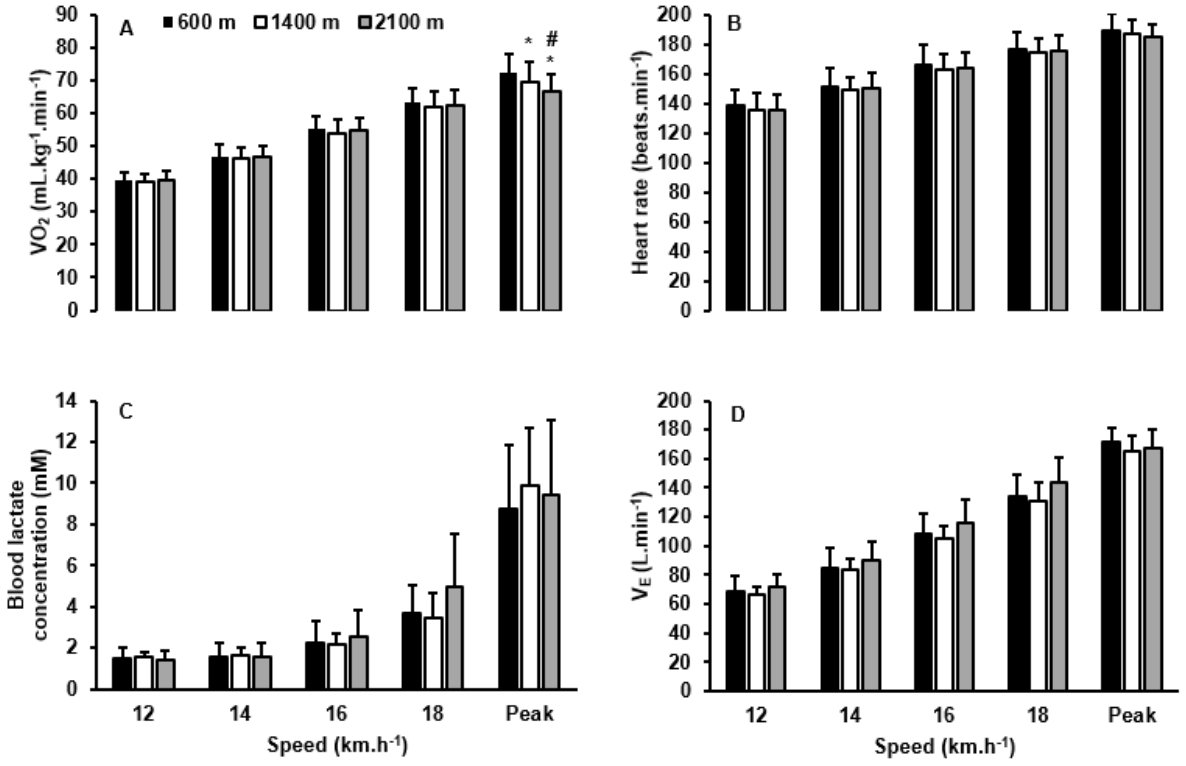


Figure 4.1. Changes in A) $\dot{V}O_2$, B) heart rate, C) [BLa], D) minute ventilation at submaximal and maximal speeds during incremental exercise testing in normoxia (black), low (white) and moderate (grey) moderate hypoxia. * significantly different to 580 m; # significantly different to 1400 m, $p < 0.05$.

Threshold (4 x 5 min) sessions

Threshold intervals were completed at 17.5 ± 0.9 km·h^{-1} across all altitudes. A main effect of altitude was observed ($p = 0.017$, $\eta^2 = 0.44$) for $\%\dot{V}O_{2max}$, with intervals completed at 2100 m (89.9 ± 5.7%) significantly higher ($p = 0.04$, % change = 4.8 ± 4.0%) than 580 m (85.9 ± 5.7%). There were no significant differences ($p = 0.69$) between 580 m and 1400 m (87.2 ± 6.0%). A significant altitude effect ($p = 0.002$, $\eta^2 = 0.59$) was observed for total $\dot{V}O_2$ (averaged over the four intervals), being significantly lower at 2100 m versus 580 m (270.9 ± 14.6 mL·kg^{-1} vs. 283.4 ± 11.8 mL·kg^{-1}, $p = 0.003$, % change = 4.4 ± 2.1%), with the difference between 1400 m (278.1 ± 15.7 mL·kg^{-1}) and 2100 m trending towards significance ($p = 0.065$). A significant time effect ($p < 0.001$, $\eta^2 = 0.84$) was also observed (**Figure 4.2A**), with $\dot{V}O_2$

significantly higher during intervals two, three and four compared to interval one ($p < 0.01$) across all altitudes. An altitude effect ($p = 0.001$, $\eta^2 = 0.48$) was observed for AOD, with higher values being observed at 1400 m (27.0 ± 17.4 $mL \cdot kg^{-1}$) and 2100 m (35.8 ± 15.8 $mL \cdot kg^{-1}$) compared to 580 m (23.7 ± 17.3 $mL \cdot kg^{-1}$; p vs. 580 m = 0.38 and 0.001 respectively). A main effect for time ($p < 0.001$, $\eta^2 = 0.84$) was also observed, with the AOD during interval one being significantly higher than all other intervals across altitudes ($p < 0.01$). A significant altitude effect was observed for aerobic contribution ($p = 0.01$, $\eta^2 = 0.48$), being significantly lower at 2100 m (88.6 ± 4.0%) compared to 580 m (92.5 ± 4.7%; $p = 0.002$), but not significantly different between 580 m and 1400 m (91.4 ± 4.8%; $p = 0.37$). Consequently, anaerobic contribution (**Figure 4.2B**) was higher at 1400 m (8.6 ± 4.8%) and 2100 m (11.4 ± 4.0%) compared to 580 m (7.5 ± 4.7%), with this difference being significant at 2100 m ($p = 0.002$). There was a significant altitude by interval interaction ($p = 0.05$, $\eta^2 = 0.25$) for [BLa]. [BLa] remained stable during all four intervals at 580 m and 1400 m ($p > 0.05$) but was significantly higher after the fourth interval compared to the first at 2100 m ($p = 0.038$). At 2100 m, [BLa] was significantly higher than all intervals at 580 m ($p < 0.05$) and after intervals three and four at 1400 m ($p < 0.05$). Significant effects for time were observed for $\dot{V}O_{2peak}$ ($p = 0.001$, $\eta^2 = 0.67$), V_E ($p = 0.001$, $\eta^2 = 0.54$), heart rate ($p < 0.001$, $\eta^2 = 0.96$), RPE ($p < 0.001$, $\eta^2 = 0.71$) and RPE.$Speed^{-1}$ ($p < 0.001$, $\eta^2 = 0.72$), with values increasing with each interval.

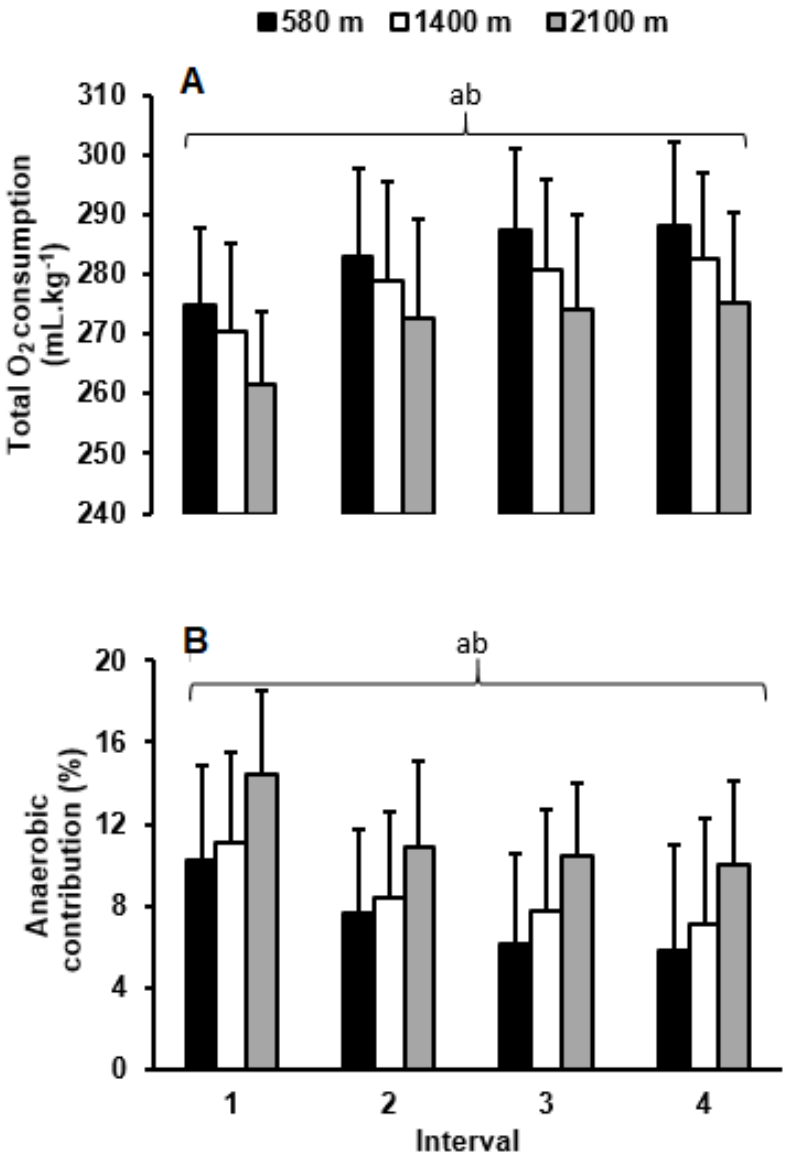


Figure 4.2. Changes in A) total O_2 consumption and B) anaerobic contribution during threshold training (4 x 5 min at 4 mM with 2 min recovery) at 580, 1400 and 2100 m. a: Significant altitude effect ($p < 0.05$), b: Significant time effect ($p < 0.05$). *Significantly different to 580 m, #Significantly different to 1400 m, +Significantly different to interval one within altitude.

$\dot{V}O_{2max}$ (8 x 90 s) sessions

$\dot{V}O_{2max}$ intervals were completed at 20.1 ± 1.3 km·h^{-1} across all altitudes, which was 1.0 and 5.8% greater than the v$\dot{V}O_{2peak}$ at 1400 m and 2100 m respectively. The %$\dot{V}O_{2max}$ at each altitude was 90.9 ± 4.5% (580 m), 90.3 ± 4.4% (1400 m) and 92.5 ± 3.7% (2100 m), with no

significant effect for altitude observed (p = 0.511, η^2 = 0.09). A main effect of altitude (p = 0.003, η^2 = 0.57) was observed for $\dot{V}O_{2peak}$, which compared to 580 m (65.4 ± 4.2 $mL \cdot kg^{-1} \cdot min^{-1}$) was significantly lower at 2100 m (61.6 ± 3.1 $mL \cdot kg^{-1} \cdot min^{-1}$; p = 0.013) but not 1400 m (62.8 ± 4.1 $mL \cdot kg^{-1} \cdot min^{-1}$; p = 0.121). Compared to 580 m (82.6 ± 6.9 $mL \cdot kg^{-1}$) total $\dot{V}O_2$ (averaged over eight intervals) was lower at both 1400 m (79.7 ± 6.4 $mL \cdot kg^{-1}$, % change = -3.6 ± 4.0%) and 2100 m (78.7 ± 5.1 $mL \cdot kg^{-1}$, % change = -4.7 ± 3.8%), with altitude (p = 0.07, η^2 = 0.31) and interaction (p = 0.06, η^2 = 0.197) effects both approaching significance (**Figure 4.3A**). A time effect (p < 0.001, η^2 = 0.85) was also observed, with $\dot{V}O_2$ significantly higher during intervals two to eight compared to interval one (p < 0.01) across all altitudes. Significant altitude effects were observed for AOD (p = 0.047, η^2 = 0.35), aerobic contribution (p = 0.03, η^2 = 0.396) and anaerobic contribution (p = 0.03, η^2 = 0.396). AOD at 1400 m (25.1 ± 6.0 $mL \cdot kg^{-1}$) and 2100 m (26.3 ± 5.5 $mL \cdot kg^{-1}$) was higher than 580 m (22.5.1 ± 4.6 $mL \cdot kg^{-1}$), with this difference approaching significance at 2100 m (p = 0.06). A main effect of time (p < 0.001, η^2 = 0.84) was observed for AOD, with the oxygen deficit during interval one being significantly higher than all other intervals across altitudes (p < 0.05). The aerobic contribution was significantly lower at 2100 m (75.2 ± 3.1%) compared to 580 m (78.7 ± 3.4%; p = 0.047), but not significantly different between 580 m and 1400 m (76.2 ± 4.2%; p = 0.37). Consequently, anaerobic contribution at 2100 m (24.8 ± 3.1%) was significantly higher compared to 580 m (21.3 ± 3.4%; p = 0.047), however there was no significant difference with 1400 m (23.8 ± 4.2%; **Figure 4.3B**). A significant interaction effect was observed for RER (p < 0.001, η^2 = 0.36). During intervals one and two, RER was higher at 2100 m than both 580 m and 1400 m (p < 0.05), with no significant difference between altitudes for the remaining intervals. Significant effects of time were observed for $\dot{V}O_{2peak}$ (p < 0.001, η^2 = 0.77), V_E (p < 0.001, η^2 = 0.58), [BLa] (p < 0.001, η^2 = 0.62), heart rate (p < 0.001, η^2 = 0.89), RPE (p <

0.001, η^2 = 0.74) and RPE·km·h^{-1} (p < 0.001, η^2 = 0.75), with values increasing with each interval.

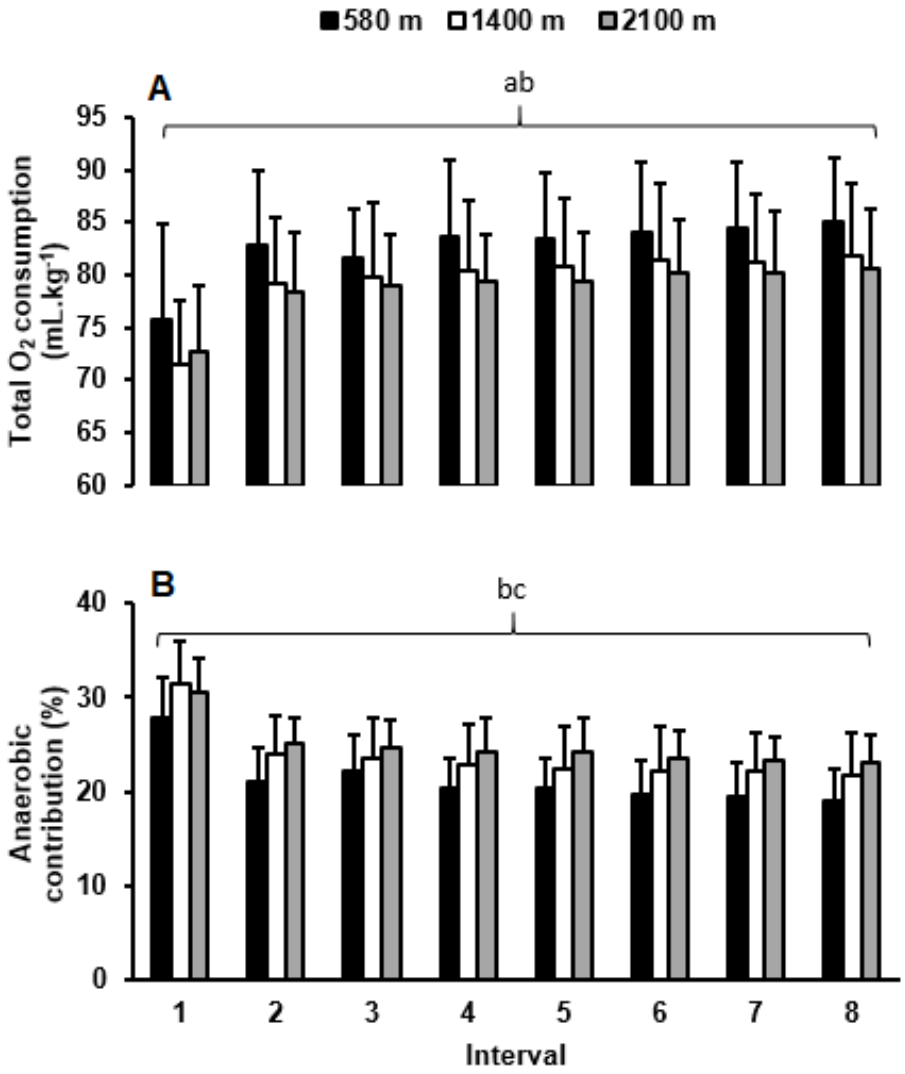


Figure 4.3. Changes in physiological parameters during $\dot{V}O_2$ max (8 x 90 s @ vV̇O2 peak with 90 s recovery) training at 580, 1400 and 2100 m. A) Total O_2 consumption. B) Anaerobic contribution. a = altitude effect, p < 0.1, b = significant time effect, c = significant altitude effect. Significance = p < 0.05.

Race pace (10 x 45 s) sessions

Race pace intervals were completed at 22.1 ± 1.4 km·h^{-1} across all altitudes, which was 1.4 and 5.7% greater than 110% of the v$\dot{V}O_{2peak}$ at 1400 m and 2100 m respectively. The %$\dot{V}O_{2max}$ at each altitude was 83.7 ± 3.0% (580 m), 85.7 ± 5.2% (1400 m) and 86.9 ± 3.4% (2100 m), with no significant effect of altitude (p = 0.182, η^2 = 0.216). A main effect of altitude (p = 0.019, η^2 = 0.43) was observed for $\dot{V}O_{2peak}$, whereby it was lower at 2100 m (58.0 ± 3.8 mL·kg^{-1}·min^{-1}; p = 0.043) but not 1400 m (59.6 ± 4.9 mL·kg^{-1}·min^{-1}; p = 0.304) compared to 580 m (60.3 ± 4.2 mL·kg^{-1}·min^{-1}). No significant altitude effects were found for total $\dot{V}O_2$ (p = 0.273, η^2 = 0.169), AOD (p = 0.56, η^2 = 0.08), aerobic and anaerobic contribution (p = 0.47, η^2 = 0.10) and [BLa] (p = 0.18, η^2 = 0.22) (**Figure 4.4**). A significant altitude effect was observed for heart rate, with the difference between each of 1400 m (163 ± 5 bpm, p = 0.061) and 2100 m (165 ± 7 bpm, p = 0.054), and 580 m (167 ± 7 bpm) approaching significance. There was a significant altitude by interval interaction (p = 0.021, η^2 = 0.214) for RPE·km·h^{-1}. RPE·km·h^{-1} remained relatively stable at 1400 m and with no significant differences between any intervals (p > 0.05); however, at 580 m RPE·km·h^{-1} for interval one was significantly lower compared to intervals six, seven and eight (p = 0.03). At 2100 m, RPE·km·h^{-1} for intervals seven, nine and ten was significantly higher than intervals one through five (p < 0.05). Significant effects for time were observed for $\dot{V}O_{2peak}$ (p < 0.001, η^2 = 0.62), V_E (p < 0.001, η^2 = 0.69), [BLa] (p < 0.001, η^2 = 0.58), heart rate (p < 0.001, η^2 = 0.82) and RPE (p < 0.001, η^2 = 0.77), with values increasing over the course of the session.

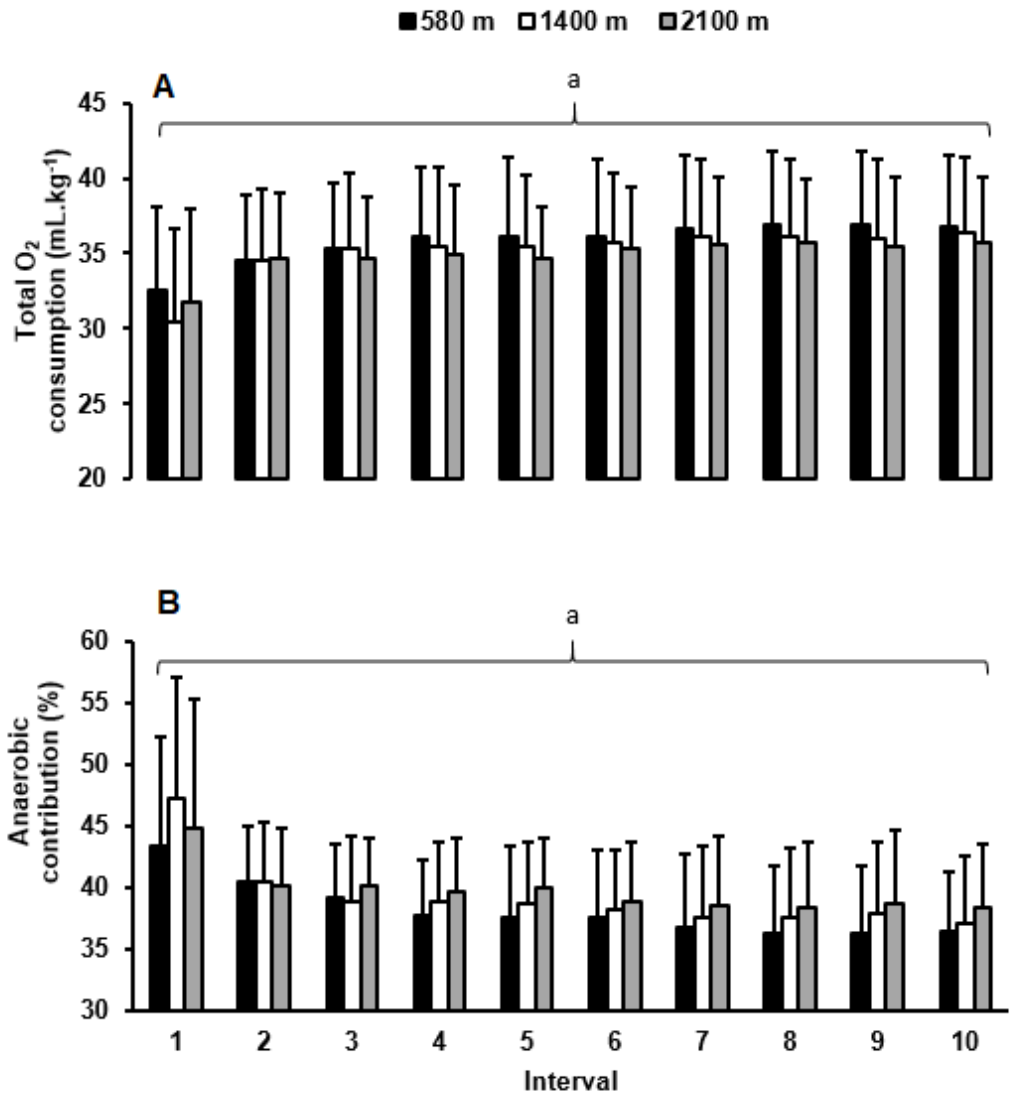


Figure 4.4. Changes in physiological parameters during race pace (10 x 45 s @ 110% v$\dot{V}O_2$ peak with 105 s recovery) training at 580, 1400 and 2100 m. A) Total O_2 consumption. B) Anaerobic contribution. a = significant time effect. Significance = $p < 0.05$.

Between session comparisons

A comparison of the mean values for each session at the different altitudes is presented in **Table 4.2**. A significant interaction effect ($p < 0.001$, $\eta^2 = 0.74$) was observed for mean relative intensity of exercise (running speed relative to % of altitude specific v$\dot{V}O_{2max}$). Relative intensity at 2100 m (92 ± 2%, 106 ± 2% and 116 ± 3% for threshold, $\dot{V}O_{2max}$ and race pace sessions respectively) was significantly higher than both 1400 m (89 ± 2%, 101 ± 3% and 111

± 3%) and 580 m (88 ± 3%, 100 ± 0% and 110 ± 0%) for all three sessions ($p < 0.01$). However, there were no differences between 580 m and 1400 m ($p > 0.80$). Significant differences between sessions ($p < 0.001$, $\eta^2 = 0.95$) were observed for anaerobic contribution, with race pace sessions (mean anaerobic contribution across altitudes = 39 ± 5%) significantly higher ($p < 0.001$) than both threshold (9 ± 4%) and $\dot{V}O_{2max}$ (23 ± 4%) sessions (threshold vs. $\dot{V}O_{2max}$, $p < 0.001$). A significant effect between sessions was also found for total $\dot{V}O_2$ (normalized to $mL \cdot kg^{-1} \cdot min^{-1}$), with race pace sessions (mean total $\dot{V}O_2$ across altitudes = 47.0 ± 6.5 $mL \cdot kg^{-1} \cdot min^{-1}$) significantly lower ($p < 0.01$) than both threshold (55.5 ± 2.8 $mL \cdot kg^{-1} \cdot min^{-1}$) and $\dot{V}O_{2max}$ (53.6 ± 4.1 $mL \cdot kg^{-1} \cdot min^{-1}$) sessions.

Table 4.2. Mean values per interval for the threshold, $\dot{V}O_{2max}$ and race pace sessions in normoxic (580 m), low (1400 m) and moderate (2100 m) hypoxic conditions.

	Threshold (4 x 5 min)			$\dot{V}O_2$ max (8 x 90 s)			Race pace (10 x 45 s)			Main effect
	580 m	1400 m	2100 m	580 m	1400 m	2100 m	580 m	1400 m	2100 m	
$\dot{V}O_{2peak}$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	61.7 ± 2.7	60.5 ± 2.2	59.9 ± 3.1	65.4 ± 4.2	62.8 ± 4.1	61.6 ± 3.1	60.3 ± 4.2	59.6 ± 4.9	58.0 ± 3.8	*†
Total $\dot{V}O_2$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	56.7 ± 2.4	55.6 ± 3.1	54.2 ± 2.9	55.2 ± 4.6	53.2 ± 4.2	52.6 ± 3.4	47.8 ± 6.6	46.9 ± 7.0	46.5 ± 6.1	*†
Accumulated O_2 deficit ($mL \cdot kg^{-1}$)	23.7 ± 17.3	27.0 ± 17.4	35.8 ± 15.8	22.5 ± 4.6	25.1 ± 6.0	26.3 ± 5.5	22.1 ± 3.1	22.6 ± 3.6	23.0 ± 3.5	†
Aerobic/Anaerobic contribution (%)	93/7 ± 5	91/9 ± 5	89/11 ± 4	79/21 ± 3	76/24 ± 4	75/25 ± 3	62/38 ± 5	61/39 ± 5	60/40 ± 5	*†
[BLa] (mM)	4.2 ± 2.1	4.7 ± 1.9	6.2 ± 2.1	6.4 ± 1.6	6.9 ± 2.1	7.9 ± 2.6	7.0 ± 2.7	6.4 ± 2.3	7.3 ± 2.8	*†
V_E ($L \cdot min^{-1}$)	132 ± 17	137 ± 18	145 ± 22	144 ± 15	148 ± 15	150 ± 16	140 ± 21	139 ± 21	142 ± 24	†
RER	0.94 ± 0.05	0.96 ± 0.03	0.98 ± 0.05	0.94 ± 0.03	0.94 ± 0.05	0.98 ± 0.04	0.89 ± 0.05	0.89 ± 0.05	0.88 ± 0.05	*
HR (bpm)	172 ± 8	174 ± 9	174 ± 9	175 ± 9	174 ± 10	174 ± 9	167 ± 7	163 ± 5	165 ± 7	*
RPE	14.2 ± 1.6	14.3 ± 0.9	14.8 ± 0.9	15.3 ± 1.1	15.1 ± 1.0	15.5 ± 0.6	14.8 ± 0.7	14.9 ± 0.9	15.6 ± 0.7	*†
RPE·km·h^{-1}	0.81 ± 0.08	0.81 ± 0.08	0.84 ± 0.05	0.77 ± 0.04	0.76 ± 0.03	0.78 ± 0.03	0.68 ± 0.03	0.69 ± 0.04	0.72 ± 0.06	*†

* Significant main effect of session, $p < 0.05$, † Significant main effect of condition (altitude), $p < 0.05$

4.5 DISCUSSION

We sought to determine the effect of low and moderate simulated normobaric hypoxia on total $\dot{V}O_2$ and anaerobic contribution to interval running at different intensities, but at the same absolute running speed across altitudes, in highly trained athletes. We confirm the results of previous research (Wehrlin and Hallén, 2006; Friedmann *et al*. 2007; Black *et al*. 2017) suggesting that for high intensity exercise completed at the same absolute work rate, total $\dot{V}O_2$ is reduced at moderate simulated altitudes compared to sea-level. Consequently, AOD and anaerobic contribution were higher in hypoxia, further corroborating previous findings (McLellan *et al*. 1990; Weyand *et al*. 1999; Ogawa *et al*. 2007). However, we extend these findings by showing for the first time that the magnitude of these differences is dependent on exercise intensity, with larger changes observed in training sessions with a greater aerobic contribution (i.e. threshold and $\dot{V}O_{2max}$), with no significant differences observed for race pace sessions between FiO_2 conditions, in contrast to our hypothesis. Moreover, our data reveal that threshold and $\dot{V}O_{2max}$ sessions at a simulated altitude of 2100 m, but not 1400 m, induced significant physiological differences (i.e. higher [BLa], anaerobic contribution) compared to interval exercise in normoxia. Finally, we add to previous literature showing altered total $\dot{V}O_2$ and AOD during single interval bouts of high intensity exercise in hypoxia by demonstrating similar responses during repeated interval training sessions specific to endurance athletes. It has previously been suggested that athletes may not be able to sustain adequate oxygen flux during aerobic exercise at altitude (Levine and Stray-Gundersen, 1997; Chapman *et al*. 1998); our findings confirm these assertions for certain training intensities and therefore have implications for the prescription of interval sessions for athletes completing altitude training.

Total $\dot{V}O_2$ during interval training at simulated altitude

Previous research examining $\dot{V}O_2$ responses during single bout, heavy intensity exercise has shown that $\dot{V}O_2$ and time to exhaustion are reduced at moderate altitudes when completed at equivalent work rates to normoxia (Wehrlin and Hallén, 2006; Friedmann *et al*. 2007; Black *et al*. 2017). We sought to extend upon these findings by investigating $\dot{V}O_2$ responses during interval training sessions at intensities frequently used by elite endurance athletes (Billat, 2001; Tjelta, 2016; Sharma *et al*. 2017). We observed total $\dot{V}O_2$ during threshold and $\dot{V}O_{2max}$ intensity (determined in normoxia) intervals was 4.4 and 4.7% lower at 2100 m compared to 580 m, with no significant differences for these two intensities between 580 m and 1400 m. Furthermore, due to the hypoxia induced reduction in $\dot{V}O_{2max}$ (**Figure 4.1A**), higher altitude specific $\%\dot{V}O_{2max}$ were achieved at simulated moderate altitudes compared to in normoxia, confirming previous findings (Black *et al*. 2017).

No differences in total $\dot{V}O_2$ were observed for race pace intervals at simulated altitudes of either 1400 or 2100 m compared to normoxia (**Figure 4.4A**). Similarly, in a study of physical education students completing 5 x 400 m intervals at 90% of their maximal 400 m speed at 690 and 2320 m, no differences were observed in $\dot{V}O_2$ (Feriche *et al*. 2007). Alternatively, in highly trained middle-distance runners, $\dot{V}O_2$ during intermittent 20 s shuttle runs (increasing in speed from 13.5 to 25 $km{\cdot}h^{-1}$) interspersed with 100 s recovery was significantly reduced from 18 to 25 $km{\cdot}h^{-1}$ at 2500 m hypobaric hypoxia compared sea-level (Ogawa *et al*. 2007). Previous research has shown that whilst the amplitude and time constant of the $\dot{V}O_2$ slow component are unaffected in hypoxia, the time constant of the primary rise in $\dot{V}O_2$ (i.e. onset of exercise) is slower in hypoxia, with no significant change in amplitude (Engelen *et al*. 1996). In comparison to our study, where 45 s intervals were selected for race pace sessions, the relatively short interval length of previous research (Ogawa *et al*. 2007) may have accounted for the lower $\dot{V}O_2$ observed, with insufficient time to reach to amplitude of the initial $\dot{V}O_2$

response. Interestingly, in the study of Ogawa and colleagues (2007), accumulated $\dot{V}O_2$ when all intervals were summated was not significantly different between normoxia and hypoxia, which is similar to our findings. However, accumulated $\dot{V}O_2$ during recovery intervals was significantly lower at each intensity in hypoxia, leading to total accumulated $\dot{V}O_2$ (run + recovery) being lower in hypoxia (Ogawa *et al*. 2007). Although $\dot{V}O_2$ during recovery between intervals was not measured in the current investigation, we speculate that the previously reported impairment of self-paced intermittent exercise in hypoxia (Brosnan *et al*. 2000; Sharma *et al*. 2017; Deb *et al*. 2018) may be driven to an extent by a lower accumulated $\dot{V}O_2$ during recovery (and thus greater AOD overall), along with the lower $\dot{V}O_2$ during exercise intervals at certain intensities. As such, the extension of recovery interval length during exercise in hypoxia could be an important strategy to maintain $\dot{V}O_2$ and performance during high intensity exercise in hypoxia (Brosnan *et al*. 2000; Saunders *et al*. 2009a), however future investigations measuring $\dot{V}O_2$ during recovery intervals of varying length would be required to confirm these assertions.

Accumulated oxygen deficit and anaerobic contribution

The reduction in the rate of oxygen uptake to steady state at altitude effectively increases the anaerobic contribution to exercise at all distances (Fulco *et al*. 1998). Accordingly, we observed the AOD at a simulated altitude of 2100 m to be 51, 17 and 4% higher than at 580 m for threshold, $\dot{V}O_{2max}$ and race pace sessions respectively, and 14, 12 and 3% higher at a simulated altitude of 1400 m. Previous research investigating the importance of aerobic metabolism to a single all-out running sprint over durations from 15 to 180 s showed that under hypoxic conditions (FiO_2 = 0.13, 3500 m), anaerobic energy release was higher than in normoxia at all durations, with the largest differences (up to 18%) being observed at sprint durations of 60 to 90 s (Weyand *et al*. 1999). Additionally, anaerobic energy release during 40 s Wingate tests has been reported to be 9% higher at a simulated altitude of 2000 m compared

to normoxia (Ogura *et al*. 2006). Alternatively, Friedmann and colleagues (2007) demonstrated no significant differences in maximal AOD (MAOD) between sea-level and a simulated altitude of 2500 m during exhaustive exercise at 110-120% of $\dot{V}O_{2max}$, lasting 2 to 3 min, designed to induce MAOD (Medbo *et al*. 1998). They suggested the discrepancy between their findings and those previous (Weyand *et al*. 1999; Ogura *et al*. 2006) was due to the protocol length selected, as Wingate tests and shorter duration sprints are insufficient in length to reach MAOD in normoxia; therefore, the capacity to increase anaerobic contribution is present in hypoxia. In the current study, the greatest increases to anaerobic contribution in hypoxia were observed in the sessions featuring the greatest aerobic contribution to exercise (i.e. threshold and $\dot{V}O_{2max}$), as the combination of duration and intensity prescribed were unlikely to elicit MAOD. Additionally, blood lactate levels were higher for these two intensities at simulated 2100 m. Meanwhile, we observed no significant changes to AOD during the race pace sessions, perhaps unsurprising given it was performed at the highest intensity, with the highest anaerobic contribution (**Table 4.2**). Similarly, Feriche and colleagues (2007) reported no significant differences in AOD when athletes completed 5 x 400 m intervals at 90% maximum 400 m speed in normoxia or hypoxia.

Limitations and future directions

A limitation of the current investigation is the absence of arterial oxyhaemoglobin saturation (SaO_2) measured during exercise – either directly or estimated via pulse oximetry (SpO_2). Following extensive piloting it was determined that the movement induced by running prevented the SaO_2 device being secured sufficiently well on the fingertip and thus invalidated the collection of data. Measurement of SpO_2 during exercise can be unreliable and inaccurate, especially when using many forms of commercially available pulse oximetry equipment which may not perform well during exercise, given increased blood flow, movement and vibration (Yamaya *et al*. 2002). Desaturation of arterial blood during exercise in hypoxia is well

quantified in the literature (Chapman *et al*. 1999; Brosnan *et al*. 2000; Chapman *et al*. 2011), with elite athletes displaying reduced measures of SaO_2 during maximal exercise in normoxia, and both submaximal and maximal exercise in hypoxia (Gore *et al*. 1996). Furthermore, there is a tight coupling between SaO_2 and $\dot{V}O_2$ during exercise, as well as a significant relationship between the degree of SaO_2 decline at maximal exercise, and the decline in both $\dot{V}O_{2max}$ and performance during aerobically dominant events in moderate hypoxia (Brosnan *et al*. 2000; Chapman *et al*. 2011). The responses may also vary depending on the intensity of a training session. For example, in elite female cyclists exercising at 2100 m, SaO_2 was lower during longer self-paced intervals (3 x 10 min) but remained unchanged during shorter repeated sprinting (3 x 6 x 15 s), relative to the same exercise completed in normoxia (Brosnan *et al*. 2000). Our findings showed unchanged $\dot{V}O_2$ during race pace exercise at simulated altitudes of both 1400 and 2100 m, however $\dot{V}O_2$ during both threshold and $\dot{V}O_{2max}$ training was lower at both altitudes compared to normoxia. We thus speculate that SaO_2 measures would have mirrored these findings, however confirmatory data monitoring SaO_2 during interval training are required.

When interpreting and practically applying the findings of the current investigation, it is important to acknowledge the physiological and performance differences that may occur between exercise in normobaric versus hypobaric hypoxia, a topic of contention in the literature (Millet *et al*. 2012; Mounier *et al*. 2012). A recent systematic review revealed a lower minute $\dot{V}_E$ and elevated symptoms of acute mountain sickness during exposure to hypobaric hypoxia compared to normobaric hypoxia (Coppel *et al*. 2015). Additionally, time trial performance is impaired to a greater extent when cycling in hypobaric than normobaric hypoxia, relative to sea-level (Beidleman *et al*. 2014; Saugy *et al*. 2016). However, the mechanisms for this impairment are unclear with SaO_2 reported as significantly lower in hypobaric hypoxia (Saugy *et al*. 2016) and unchanged between hypoxic conditions (Beidleman *et al*. 2014), though the

latter investigation used a more severe level of hypoxia (4300 m vs. 3450 m). Based on our previous findings of impaired performance during self-paced intervals at 2100 m hypobaric hypoxia (Sharma *et al*. 2017), one may expect performance to be similarly impaired in the current investigation, however athletes were able to complete all intervals at the prescribed normoxic training intensities, likely due to an increased anaerobic contribution, particularly during threshold and $\dot{V}O_{2max}$ sessions. Together, these observations suggest the physiological responses to training in hypobaric hypoxia merit further interrogation, with important implications for prescription of training during natural altitude camps.

4.6 PRACTICAL RECOMMENDATIONS

Previous recommendations for altitude training have suggested a live high train low (LHTL) method (Levine and Stray-Gundersen, 1997), or live high train low and high paradigm (LHTLH), whereby high intensity training is completed at a lower altitude, with low intensity training remaining at the residential altitude, usually between 2000 and 2500 m (Stray-Gundersen *et al*. 2001). Based on our current and previous findings (Sharma *et al*. 2017), we would suggest some modifications to this frequently used strategy in athletes habituated to altitude training. For threshold and maximal aerobic sessions, descending to 1400 m would be beneficial in helping to defend oxygen flux. However, if the desired outcome was to increase the anaerobic contribution to exercise, which is relevant for middle-distance performance (Gastin, 2001), especially in the absence of an altitude induced increase in haemoglobin mass (Garvican *et al*. 2011), remaining at moderate altitude and increasing the length of recovery intervals to help maintain running speed would be suitable. Middle-distance race-pace sessions may be completed at moderate altitude with little change in physiological stimulus compared to sea-level training. These updated recommendations are comparable to the recently proposed live high train low and high approach proposed for team sport athletes (Brocherie *et al*. 2015), involving some high intensity exercise completed at moderate altitudes. However, the ergogenic potential of such a strategy for sea-level performance has yet to be confirmed in elite endurance athletes.

4.7 CONCLUSION

The results of the investigation show completing high intensity interval running at a simulated altitude of 2100 m, but not 1400 m, is likely to induce a lower $\dot{V}O_2$ and greater anaerobic contribution to exercise when compared to training at 580 m, with the greatest effects observed for threshold and maximal aerobic sessions.

5.0 STUDY THREE: TRAINING PERIODISATION DURING LIVE HIGH TRAIN HIGH AT 2100 m IMPROVES SEA-LEVEL PERFORMANCE IN ELITE RUNNERS

Citation: **Sharma AP**, Saunders PU, Garvican-Lewis LA, Périard JD, Clark B, Gore CJ, Raysmith BP, Stanley J, Robertson EY, Thompson KG. Training periodisation during Live High Train High at 2100 m improves sea-level performance in elite runners. *J Sports Sci Med.* 2018;17(4):607-616.

5.i FORM E: DECLARATION OF CO-AUTHORED PUBLICATION CHAPTER

Declaration for Thesis Chapter [5]

Declaration by candidate

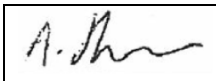
In the case of Chapter [5], the nature and extent of my contribution to the work was the following:

Nature of contribution	**Extent of contribution (%)**
Study design, data collection and analysis, study write-up	60

The following co-authors contributed to the work.

Name	**Nature of contribution**	**Extent of contribution**	**Contributor is also a student at UC Y/N**
Philo Saunders	Assistance with design, data collection, proof reading, editing of drafts	10	N
Laura Garvican-Lewis	Assistance with design, proof reading, editing of drafts	3	N
Julien Périard	Assistance with design, proof reading, editing of drafts	3	N
Brad Clark	Assistance with data collection, data analysis, proof reading, editing of drafts	5	N
Christopher Gore	Assistance with design, proof reading, editing of drafts	3	N
Ben Raysmith	Assistance with data collection, proof reading, editing of drafts	3	N
Jamie Stanley	Assistance with data collection, proof reading, editing of drafts	3	N

Eileen Robertson	Assistance with data collection, proof reading, editing of drafts	3	N
Kevin Thompson	Assistance with design, proof reading, editing of drafts	7	N

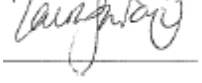
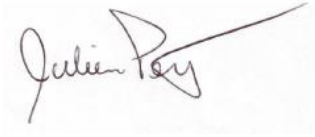
Candidate's Signature		**Date** **12.6.2018**

Declaration by co-authors

The undersigned hereby certify that:

(1) The above declaration correctly reflects the nature and extent of the candidate's contribution to this work, and the nature of the contribution of each of the co-authors.
(2) they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least that part of the publication in their field of expertise;
(3) they take public responsibility for their part of the publication, except for the responsible author who accepts overall responsibility for the publication;
(4) there are no other authors of the publication according to these criteria;
(5) potential conflicts of interest have been disclosed to (a) granting bodies, (b) the editor or publisher of journals or other publications, and (c) the head of the responsible academic unit; and
(6) the original data are stored at the following location(s) and will be held for at least five years from the date indicated below:

Location(s)	Australian Institute of Sport Department of Physiology, Bruce, ACT Research Institute for Sport and Exercise, University of Canberra, Bruce ACT

Signature 1		**Date** 12.6.2018
Signature 2		12.6.2018
Signature 3		12.6.2018
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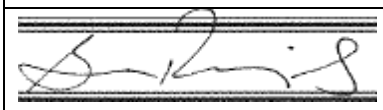
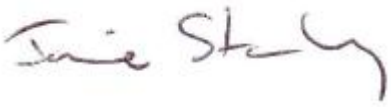
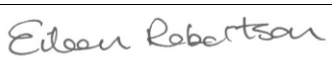
Signature 6		12.6.2018
Signature 7		12.6.2018
Signature 8		12.6.2018
Signature 9		12.6.2018

5.1 ABSTRACT

Purpose: The questionable efficacy of live high train high altitude training (LHTH) is compounded by minimal training quantification in many studies. We sought to quantify the training load (TL) periodisation in a cohort of elite runners completing LHTH immediately prior to sea-level competition.

Methods: Eight elite runners (6 males, 2 females) with a $\dot{V}O_{2peak}$ of 70 ± 4 $mL.kg^{-1}.min^{-1}$ were monitored during 4 weeks of sea-level training, then 3-4 weeks LHTH in preparation for sea-level races following descent to sea-level. TL was calculated using the session rating of perceived exertion (sRPE) method, whereby duration of each training session was multiplied by its sRPE, then summated to give weekly TL. Performance was assessed in competition at sea-level before, and within 8 days of completing LHTH, with runners competing in 800 m (n = 1, 1500 m/mile (n = 6) and half-marathon (n = 1). Haemoglobin mass (Hb_{mass}) via CO rebreathing and running economy (RE) were assessed pre and post LHTH.

Results: Weekly TL during the first 2 weeks at altitude increased by 75% from preceding sea-level training (p = 0.0004, d = 1.65). During the final week at altitude, TL was reduced by 43% compared to the previous weeks (p = 0.002; d = 1.85). The ratio of weekly TL to weekly training volume increased by 17% at altitude (p = 0.009; d = 0.91) compared to prior sea-level training. Hb_{mass} increased by 5% from pre- to post-LHTH (p = 0.006, d = 0.20). Seven athletes achieved lifetime personal best performances within 8 days post-altitude (overall improvement 1.1 ± 0.7%, p = 0.2, d = 0.05).

Conclusions: Specific periodisation of training, including large increases in training load upon arrival to altitude (due to increased training volume and greater stress of training in hypoxia) and tapering, were observed during LHTH in elite runners prior to personal best performances. Periodisation should be individualised and align with timing of competition post-altitude.

5.2 INTRODUCTION

Live high train high (LHTH) refers to athletes living and training at natural altitude for a short period of time (usually two to four weeks) to prepare for competitions at altitude, or to improve sea-level performance subsequent to adaptations gained through acclimatisation and/or associated training in hypoxia (Saunders *et al*. 2009a). The potential benefit of LHTH over other forms of altitude training such as live high train low (LHTL) is the provision of an additional hypoxic training stress which may increase the relative intensity of training (Pugliese *et al*. 2014), in addition to the acclimatisation benefits of altitude residence, including accelerated erythropoiesis (Friedmann-Bette, 2008).

The general consensus from coaches and athletes is that LHTH improves physiological capacities and performance in competition during endurance events, a notion evidenced by its frequent and continued use by elite athletes (Pugliese *et al*. 2014; Solli *et al*. 2017) and supported by several studies in the literature (Daniels and Oldridge, 1970; Gore *et al*. 1998; Bonne *et al*. 2014). Accordingly, a meta-analysis reported a 1.6 ± 2.7% improvement in performance for elite athletes following LHTH (Bonetti and Hopkins, 2009). However, the large variability in these results (exemplified by a standard deviation approaching twice the mean effect) is confirmed by studies reporting no change or a decrement in performance following LHTH (Adams *et al*. 1975; Jensen *et al*. 1993; Levine and Stray-Gundersen, 1997; Bailey *et al*. 1998; Gough *et al*. 2012). Factors which might explain these equivocal findings include the altitude at which athletes lived and trained (Bailey *et al*. 1998), relative intensity of training sessions (Lundby *et al*. 2012), athlete iron status or supplementation protocol (Stray-Gundersen *et al*. 1992), and a reduction in training quality mediated by lower oxygen availability (Chapman *et al*. 1998).

To analyse and establish causal relationships between the training performed and the resultant physiological and performance adaptations, it is imperative to precisely and reliably quantify training load (TL) (Mujika, 2013). A limitation of many LHTH studies is that only basic metrics such as overall training volume or duration have been reported (Adams *et al*. 1975; Gore *et al*. 1997; Bailey *et al*. 1998). Without appropriate quantification, it is no surprise that both the literature and anecdotal evidence from coaches is conflicting regarding the best training strategies to employ during altitude camps, and the optimal time to compete thereafter (Chapman *et al*. 2014b). Longitudinal TL data from LHTH training sojourns, combined with related athlete performance data may assist coaches and scientists to identify training periodisation strategies that may be employed by elite athletes during LHTH to improve sea-level performance.

As such, in a cohort of elite runners, we sought to firstly; quantify the training load (TL) periodisation during LHTH, and secondly; describe the physiological and performance changes following LHTH at 2100 m.

5.3 METHODS

Subjects

Eight athletes (6 males, 2 females; age, 25 ± 6 years; $\dot{V}O_{2peak}$, 70 ± 4 mL.kg^{-1}.min^{-1}; Season's Best as % of World Lead, 90% ± 5%;) participated in 3-4 weeks of LHTH in Flagstaff, USA (elevation 2100 m) after 4 weeks of quantified near sea-level training (three weeks LHTH, *n* = 3; four weeks LHTH, *n* = 5). Of these athletes, 5 represented Australia internationally at the 2016 Olympic/Paralympic Games and/or 2015 IAAF World Championships. Three athletes (participants 4, 7 and 8) had previously trained in Flagstaff and four of the remaining athletes (participants 1, 2, 3 and 6) had experienced LHTH at 1600 to 1800 m in Australia. All of these athletes had participated in at least 1 LHTH camp in Australia within the 4 months preceding the investigation. Additionally, 4 of these athletes (participants 2, 4, 7 and 8) had an extensive history of altitude training utilising both natural LHTH and simulated LHTL over the preceding 3 to 5 years (2-3 camps annually). One athlete (participant 5) had never engaged in altitude training previously. All procedures and risks were explained to participants before they provided written informed consent to participate. Ethical approval for the investigation was granted by the institutional ethics committees (University of Canberra – HREC ref. no. 15-45 and Australian Institute of Sport – approval no. 20150613) and all procedures complied with the Declaration of Helsinki.

Study design

The investigation was an observational cohort case study examining the training of elite middle-distance runners during an in-season training intervention. Participants' training sessions were individually tailored and designed by their coaches, and were not manipulated or directly influenced by researchers involved in the study.

The investigation took place immediately following the Australian domestic track season, and before the American/European summer season, in April-June. The training of each athlete was monitored for 7-8 weeks in total, and was divided into 2 phases. The first phase involved athletes completing 4 weeks of their own, coach-prescribed training at or close to sea-level (i.e. Lead-in phase). Six athletes completed the first 2-3 weeks of this phase in their home environment in Australia, then travelled overseas, in most cases for competition (n = 2 in Nassau, Bahamas, n = 2 in San Francisco, USA. During the final week of this lead-in phase, these 6 athletes convened in San Francisco, where they resided for the remaining 4-7 days of this period, thus acclimating to the same time-zone as Flagstaff and minimising any effects of jet-lag (Fowler *et al*. 2017) occurring simultaneously to altitude adjustment. The remaining 2 athletes completed this entire phase in Australia. Immediately following, participants travelled to Flagstaff (2100 m elevation) to complete 3-4 weeks (hypoxic dose = 1109-1512 $km.h^{-1}$; Garvican-Lewis *et al*. 2016) of LHTH (i.e. Altitude phase). All participants were supplemented with oral iron (Ferro-Grad C, Abbott Laboratories, Australia, 105 g elemental iron) daily for at least 1 week prior to and for the duration of LHTH to ensure erythropoietic adaptations were not compromised by insufficient iron availability (Stray-Gundersen *et al*. 1992). Athletes competed in competitive races within a week of completing LHTH. Laboratory testing of running economy and haemoglobin mass occurred at the commencement and conclusion of the altitude phase (within 24-48 hours of arrival and departure to/from Flagstaff).

Training

The structure of a typical training week for all athletes is shown in **Table 5.1**. Whilst generally adhering to this similar structure, training was individualised for each athlete based on previous altitude training experience (**Table 5.2**), preferred event (**Table 5.3**), physiological characteristics, and anecdotal results regarding the best training strategy for the current altitudes; therefore, not all athletes were on identical training programs. For example, race pace

and some $\dot{V}O_{2max}$ intensity sessions were modified to include additional recoveries between intervals to maintain running speed, whereas the structure of threshold and low-intensity training sessions were not modified (i.e. same interval/recovery length), but performed at a reduced speed and/or higher perceived effort compared to sea-level (Sharma *et al*. 2017). All athletes completed a taper during the final week of the camp in preparation for upcoming races occurring immediately following LHTH, following either 2 or 3 weeks of full training depending on their total camp duration. Training was completed between 2100 to 2700 m with the exception of one race pace session (~ 90 min) completed at 1400 m (Sedona, USA) towards the end of the training camp.

Table 5.1. General structure of a training week at sea-level and altitude.

	MON	**TUES**	**WED**	**THURS**	**FRI**	**SAT**	**SUN**
AM	6-10 km @ low intensity	Intervals + hills @ $\dot{V}O_{2max}$ intensity	10-20 km @ low to moderate intensity	Race pace	Strength training	Threshold	16-30 km @ low to moderate intensity
PM	Strength training + 6-8 km @ low intensity	30-40 min run/swim @ low intensity		30-40 min run/swim @ low intensity		6-8 km @ low intensity or rest	

Table 5.2. Training structure of initial week at altitude depending on prior experience.

	Day 1 (arrive)	**2**	**3**	**4**	**5**	**6**	**7**
New to altitude	Easy run	2 easy runs	2 runs or 1 medium length run	Threshold to $\dot{V}O_{2max}$ + easy run	Strength training	Race pace	Long run
Previous altitude experience	Easy run	Easy run AM threshold run PM	2 runs or 1 medium run	$\dot{V}O_{2max}$ + race pace strides + easy run	Strength training	Race pace	Long run

Table 5.3. Representative full training and taper weeks at altitude for distance and middle-distance athletes

Full training	**Monday**	**Tuesday**	**Wednesday**	**Thursday**	**Friday**	**Saturday**	**Sunday**
Distance	AM – 50 min PM – 30 min	AM – 8 x 1 km/600 m @ HM pace - 5s/HM pace +20s PM – 30 min	AM – 90 min + gym PM – 20 min + drills/strides	AM – 8 x 400 m with 200 m float + 6 min threshold PM – 30 min	AM - 60 min	AM – 10 min threshold + 6 x 200 m hills + 10 min threshold PM – 30 min	145 min
Middle-distance	AM – 45 min PM – Gym + 30 min	AM – 36 min fartlek PM – 35 min	AM – 60 min PM – 25 min	AM – 4 x 400 m, 4 x 300 m, 6 x 200 m PM – 35 min	AM - Gym	AM – 3 km threshold, 6 x 300 m hills	105 min
Taper	**- 6 days**	**- 5 days**	**- 4 days**	**- 3 days**	**- 2 days**	**- 1 days**	**Race day**
Distance	AM – 60 min + Gym	AM – 3 km @ HM pace, 2 x 1 km @ 5 km/10 km pace PM – 30 min	AM – 60 min	AM – 4 x 2 min @ HM pace, 2 x 1 min @ 5 km pace	AM – 40 min PM – travel from altitude	AM – 30 min	AM – ½ marathon race
Middle-distance	80 min	AM – 35 min PM – Gym + 25 min	AM – 2 x (1200 m, 3 x 300 m)	AM – 45 min	Rest	AM – 5 km + strides PM – travel from altitude	AM – 4 km PM – mile race

HM: Half-marathon

Training monitoring

Each athlete recorded all their running training on a GPS watch (Forerunner, Garmin International, Kansas, USA), including total distance (km) and duration (min). Duration was also recorded for cross training or strength training sessions. A session rating of perceived exertion (sRPE) score on a modified Borg scale was provided for all training sessions (Foster, 1998). Training volume (TV) was calculated as total running distance completed each week in kilometres. Daily TL was calculated as the duration of each training session multiplied by sRPE, then summated to give weekly TL. To assess the relationship between weekly TL and TV at sea-level and altitude, weekly TL was divided by weekly TV to give a load/volume ratio.

Performance

This research project was arranged around domestic and international track and field competitions, and the race times of athletes were collated as a record of performance. Running performance was recorded before and after LHTH. The season's best time achieved during the track season preceding the investigation was used as the pre-altitude measure. For 7 of 8 athletes, post-altitude races were completed within 8 days of descending from altitude. These races took place in the USA (Boston, n = 3; Nashville, n = 2; San Diego, n = 1) and Europe (Oslo, n = 1). One participant began competing after 4 weeks at sea-level (Participant 8, who in consultation with his coach, elected to race following a period of sea-level training due to timing of season objectives and personal preference). All races were completed at or near to sea-level (0 to 600 m) on standard 400 m athletics tracks. Of the athletes, 1 competed in the half-marathon, 5 in the 1500 m, 1 in the mile (1609 m), and 1 in the 800 m. Athletes were free to employ their own preparations and use of legal ergogenic aids such as caffeine, but were asked to keep this consistent between races.

$\dot{V}O_{2peak}$

An incremental running test was completed at sea-level prior to departure to altitude on a custom built motorised treadmill (Australian Institute of Sport, Australia) to determine $\dot{V}O_{2peak}$. The test consisted of a self-selected warm-up followed by an incremental protocol commencing at a speed of 14-16 $km \cdot h^{-1}$, depending on the athlete, and increasing by 0.5 $km \cdot h^{-1}$ every 30 s until an increase of 4 $km \cdot h^{-1}$ from the starting speed. After 30 s at terminal speed, the gradient was increased from 0% every 30 s by 0.5% until volitional exhaustion. Heart rate was measured continuously via telemetry (Polar Electro, Kempele, Finland). Expired gases were collected throughout the test for determination of ventilation, $\dot{V}O_2$, $\dot{V}CO_2$ and respiratory exchange ratio, using a metabolic cart (custom-built, open-circuit, indirect calorimetry system described previously [Saunders *et al*. 2004]). $\dot{V}O_{2peak}$ was taken as the highest 30 s value.

Running Economy

Submaximal running economy was measured on a motorised treadmill (Pro, Woodway, Germany). This testing was completed at altitude in Flagstaff and occurred at the commencement and conclusion of the altitude phase (within 24-48 hours of arrival and departure to/from Flagstaff). Participants completed the same self-selected warm-up before each test, followed by 4 min at 16 $km.h^{-1}$. Heart rate and expired gases (True One 2400, ParvoMedics, USA) were collected throughout the test. Prior to each test, the ParvoMedics system was calibrated to room air and a gas of known concentration (16% O_2 and 4% CO_2). A 3-liter syringe was used to calibrate flow. Submaximal running economy and ventilation were respectively taken as mean $\dot{V}O_2$ and $\dot{V}_E$ measured during the final minute at 16 $km.h^{-1}$.

Haemoglobin mass

Total Hb_{mass} was measured at the same time-points as running economy (i.e. at altitude in Flagstaff) using the 2 min CO rebreathing method with some modifications (Schmidt and

Prommer, 2005). Briefly, participants rebreathed a CO bolus equivalent to 1.3 ml.kg^{-1} of body mass for a period of 2 min. Capillary blood samples were drawn at the start of the test and 7 min post administration of the CO dose for determination of the percentage of bound carboxyhaemoglobin (%HbCO). Blood samples were measured a minimum of 5 times for %HbCO using an OSM3 hemoximeter (Radiometer, Copenhagen, Denmark). Expired CO was determined using a Draeger Pac 7000 (Lubeck, Germany) CO sensor. Hb_{mass} was calculated from the mean change in %HbCO before and after rebreathing. Due to the nature of the investigation involving elite athletes on specific training schedules, Hb_{mass} could only be assessed a single time both pre and post-intervention. The typical error of measurement for Hb_{mass} of the investigator administering the tests (obtained from repeated measures within 48 hours on 6 participants taken within 6 weeks of departure to Flagstaff) was 1.1% (90% confidence limits = 0.7, 2.3%).

Statistical Analysis

The magnitude of changes in weekly TL and TV were quantified using standardised mean difference (Cohen's *d* effect sizes), whereby the change in parameter values were divided by the pooled standard deviation (SD). Changes for laboratory measures and performance were analysed using paired t-tests, with effect sizes (Cohen's *d*) and percentage changes also calculated along with 90% confidence limits (CL). Effects sizes were interpreted using effect thresholds of <0.2, 0.2, 0.5, and 0.8 for trivial, small, moderate, and large effects respectively, and expressed with 90% CL to denote the imprecision of the estimate. Data are displayed as mean ± SD unless otherwise stated and alpha was set at $p \leq 0.05$. Testing was performed using the SPSS statistical package (IBM, New York, USA).

5.4 RESULTS

Training

Training load changes are shown in **Figure 5.1A**. There was a very large increase in weekly TL between the lead-in period and first three weeks (first two weeks for $n = 3$ athletes completing three weeks LHTH) at altitude (1193 ± 371 to 1903 ± 455, $p = 0.0002$, $d = 1.71$ [0.67, 2.56], % change [90% CL] = 77 [31, 123]). All athletes increased TL during LHTH relative to lead-in (range 24 to 256%). TL during each of weeks one and two of LHTH were significantly greater ($p < 0.01$, $d = 1.46$-2.06) than each of weeks two, three and four of the lead-in period.

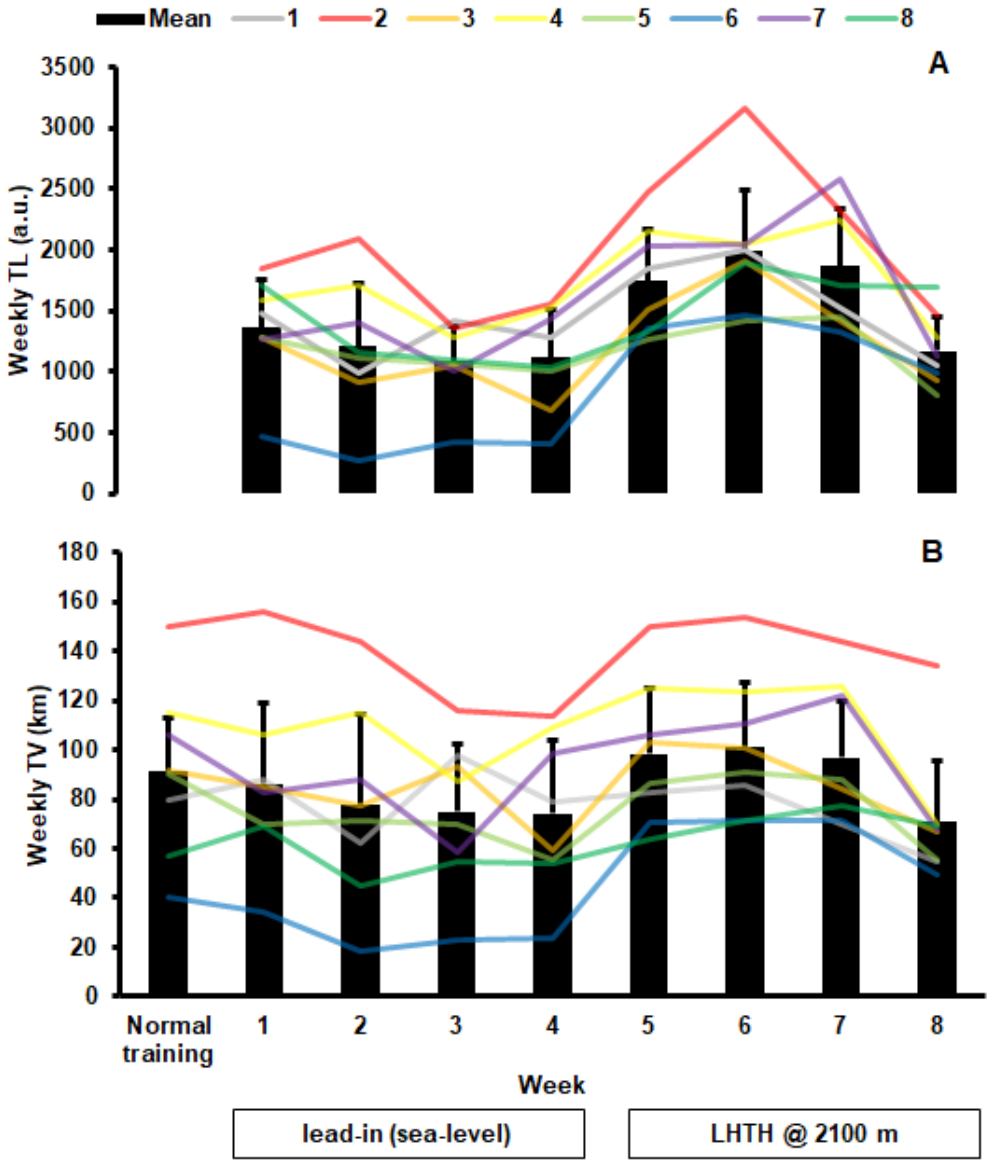


Figure 5.1. A) Quantification of weekly TL and B) weekly TV during lead-in and intervention periods. Histograms represent the mean response, and coloured lines the individuals indicated in the legend. Week 7 represents the 3rd week for athletes completing 4 weeks of LHTH (n = 5), with week 8 being the final week for all (n = 8). Normal training is the 4 weeks prior to the commencement of the study, TV was habitually recorded by participants during this period, however TL was not quantified.

In the four weeks of normal training near sea-level prior to the pre-camp lead-in period mean TV was 91 ± 22 km. During the lead-in period and first three weeks of LHTH (first two weeks for n = 3 athletes completing three weeks LHTH), athletes completed 78 ± 30 and 101 ± 26 km respectively. Weekly TV increased from lead-in to weeks 1 and 2 of the intervention period (p = 0.02, d = 0.83 [-0.07, 1.64], % change = 44 [8, 80]). When a participant was excluded (participant 6) due to illness during the lead-in period (187% increase in TV), the mean increase was 23% (16, 31%).

Athletes scheduled to compete within 8 days following LHTH (7 of 8) recorded a reduced TL (1166 ± 276) and TV (71 ± 25 km) during the final week of LHTH, which was significantly lower (43%, range 28 to 49%; p = 0.002; and 33%; range 12 to 44%; p = 0.002 respectively) than weekly TL and TV of the first 3 weeks of LHTH (first 2 weeks for n = 3 athletes completing 3 weeks LHTH). The load:volume ratio (**Figure 5.2**) increased significantly during LHTH compared to lead-in (15.8 ± 2.9 to 18.5 ± 2.7, p = 0.008, d = 0.96 [0.05, 1.77]), with 7 of 8 athletes recording a higher value during LHTH. Representative individual examples of daily and weekly periodisation of TL and TV are shown in **Figure 5.3** for distance (**Figure 5.3A**) and middle-distance (**Figure 5.3B**) athletes.

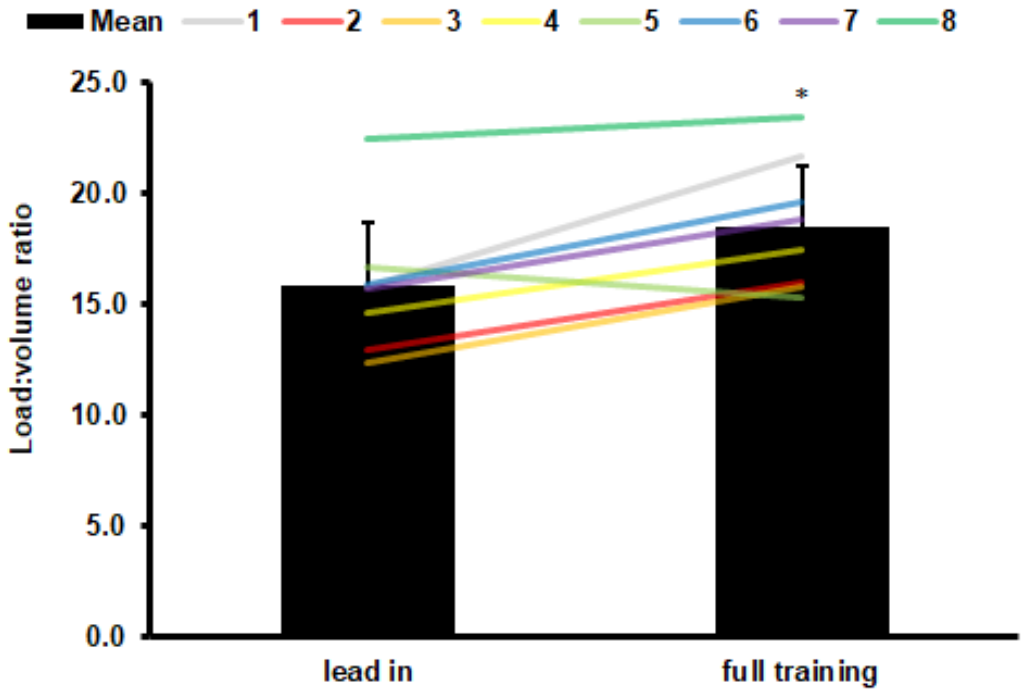


Figure 5.2. Changes in load:volume ratio from sea-level to altitude with individual responses. This value was calculated by divided weekly training load by training volume. Lead in refers to the initial 4 weeks of sea-level training. Full training is the first 3 weeks of the altitude period (first 2 weeks for n = 3 completing 3 weeks of LHTH). Histograms represent the group mean, coloured lines are individuals indicated in the legend. * significantly different to lead in, $p < 0.05$.

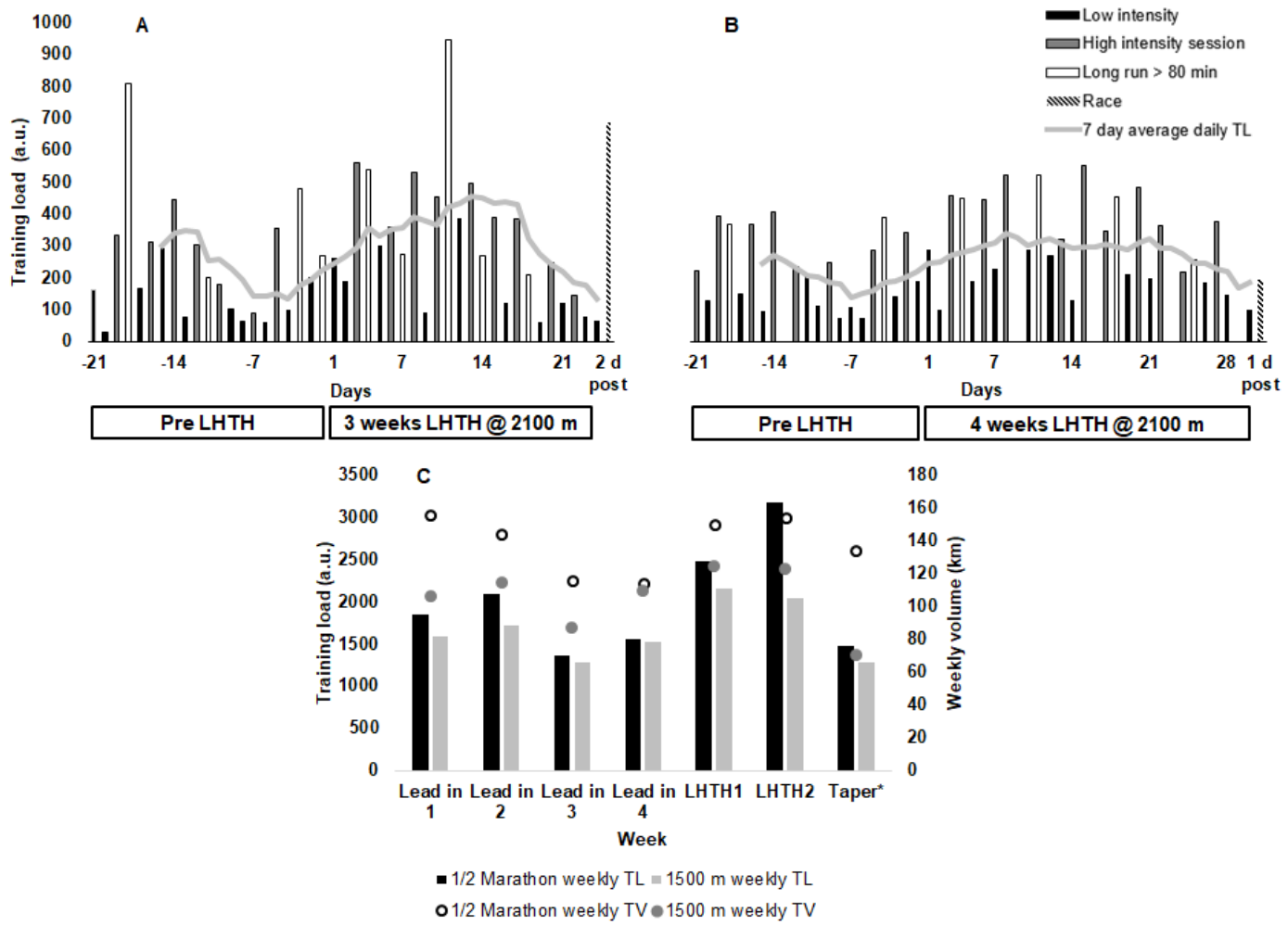


Figure 5.3. Representative individual examples of daily periodisation of running TL for A) distance (10000 m/half-marathon – participant 2) and B) middle-distance (800/1500 m – participant 4) athletes. C) Representative examples of Weekly TL and TV for distance and middle-distance athletes. * Taper is the final week of the intervention period for both athletes - week 3 for the distance athlete and week 4 for middle-distance; the latter completed 126 km and 2242 a.u. TL during their week 3.

Performance

Of the eight athletes, seven competed in races with the first eight days following LHTH, and all seven achieved personal bests, an average of 3.7 days post-altitude (range one to eight days; **Table 5.4**). Participant 8 began competing ~ four weeks following LHTH and achieved a personal record on day 57 post-altitude. The overall improvement of athletes compared to their prior season's best time was 1.1 ± 0.7% (d = 0.05, p = 0.2).

Table 5.4. Individual performance changes in following LHTH

Athlete	Age	Sex	Performance level	Event	Performance time – pre (mm:ss.00)	Performance time – post (mm:ss.00)	% change	Days post LHTH
1	23	M	Provincial	1500 m	04:13.26	04:13.22	- 0.0	1
2	27	F	Top 10 IAAF World Championships, Olympian	Half-marathon	71:51.00	71:07.00	- 1.0	2
3	25	F	IAAF World Indoor Championships finalist	1500 m	04:09.41	04:05.56	- 1.5	6
4	38	M	National finalist	Mile	04:06.20	04:01.81	- 1.8	1
5	19	M	IPC World Championship and Paralympic medallist, WR holder	1500 m	04:06.60	04:05.39	- 0.5	1
6	18	M	National junior medallist	1500 m	03:50.50	03:46.33	- 1.8	7
7	27	M	IPC World Championship and Paralympic medallist, WR holder	1500 m	03:50.61	03:48.55	- 0.9	8
8	23	M	IAAF World Championships representative	800 m	01:47.52	01:45.79	- 1.6	57
Mean	25.0						- 1.1	
SD	6.2						0.7	

Laboratory measures

Laboratory measures were conducted on 7 of 8 participants, with 1 participant (participant 6) being excluded from post-testing due to illness. Total Hb_{mass} increased significantly following LHTH (785 ± 203 to 826 ± 210 g, p = 0.006, d = 0.20 [-0.71, 1.07], % change = 5.3 [3.3, 7.4]; **Figure 5.4A**). Relative Hb_{mass} also increased significantly following LHTH (12.5 ± 1.9 to 13.2 ± 1.9 $g.kg^{-1}$, p = 0.006, d = 0.37 [-0.54, 1.23], % change = 5.2 [2.5, 8.0]). All athletes achieved increases in Hb_{mass} (range 1.2 to 9.7 %); athletes completed 3 weeks of LHTH had a mean change of 4.9 ± 1.0%, with those completing 4 weeks increasing Hb_{mass} by 5.6 ± 3.6%. Tested athletes who were < 24 years of age (n = 3) increased Hb_{mass} by 8.0 ± 1.7%, with athletes > 26 years (n = 3) increasing Hb_{mass} by 2.6 ± 1.0%.

Changes for submaximal ventilation ($\dot{V}_E$) are shown in **Figure 5.4B**. RE at 16 $km{\cdot}h^{-1}$ remained unchanged following the intervention period (pre: 3.5 ± 0.5 $L{\cdot}min^{-1}$, post: 3.4 ± 0.5; p = 0.12, d = 0.17 respectively). $\dot{V}_E$ at 16 $km.h^{-1}$ tended towards increasing after LHTH (p = 0.11, d = 0.30 [-0.58, 1.19], % change = 7.6 [1.5, 13.8]).

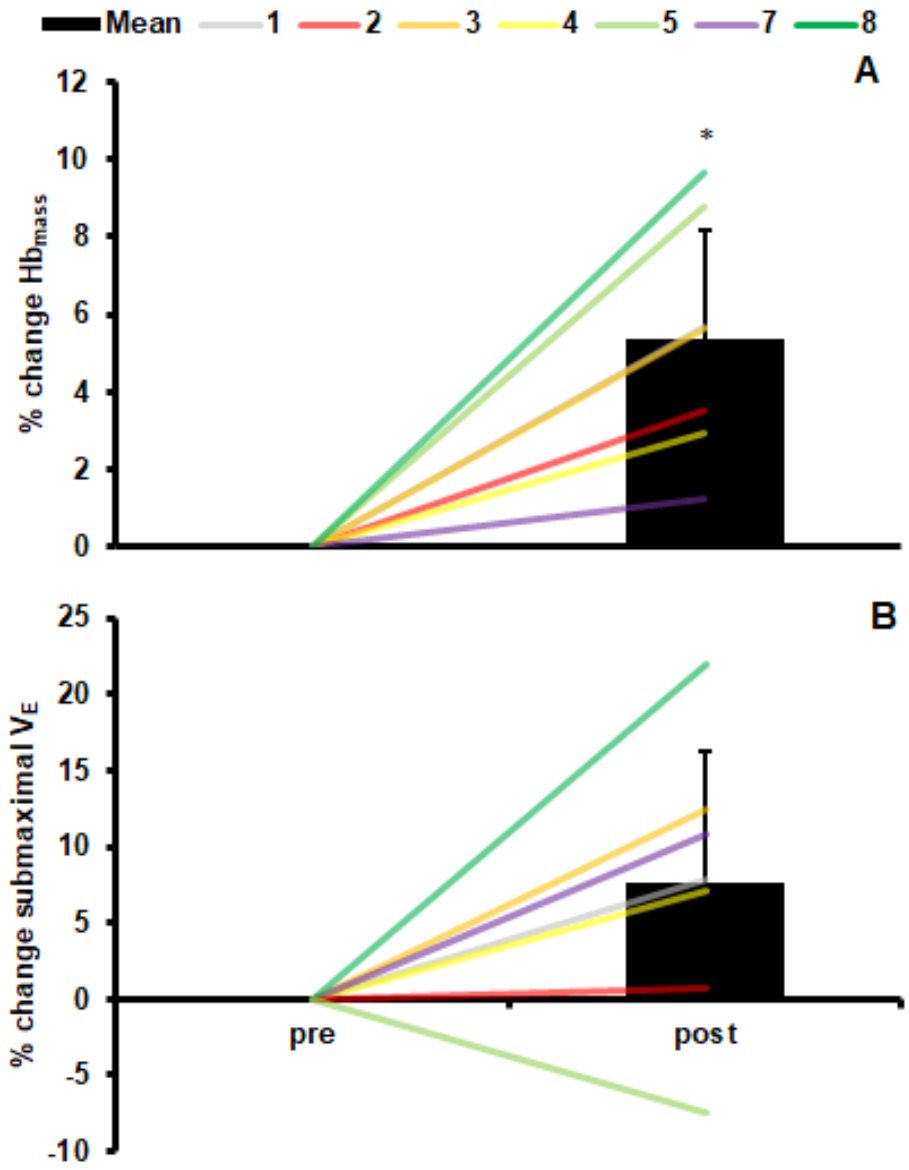


Figure 5.4. Percentage change in Hb_{mass} (A) and submaximal V_E (B) following 3-4 weeks of LHTH for n = 7 (Participant 6 excluded due to illness during post testing). Coloured lines are individuals indicated in the legend, with histograms showing the group response. * $p < 0.01$.

5.5 DISCUSSION

The main findings of this case study were as follows:

1. Compared to sea-level lead-in training, TL and TV were increased by 77% and 44%, respectively, during LHTH.
2. The increase in TL occurred immediately upon arrival to altitude, with athletes foregoing the traditional acclimatisation period of low intensity training.
3. Compared to peak weekly values at altitude, there was a large reduction in TL (43%) and TV (30%) in the final week of LHTH for athletes competing within 8 days following LHTH.
4. All 7 athletes competing within 8 days of completing LHTH achieved personal record performances.

It must be noted our observational case study design of athletes completing individualised training does not allow us to isolate the relative contributions of intensified training and altitude exposure in improving performance. However, these data provide important information regarding training periodisation strategies which may be employed by elite runners engaging in LHTH prior to competition, and are therefore of interest to coaches and scientists prescribing altitude training.

Performance

A combination of intensified training, tapering and 3-4 weeks of LHTH at 2100 m was observed immediately prior to athletes achieving a 1.1% improvement in competitive race performance (**Table 5.4**). This degree of improvement is in accordance with previous uncontrolled (Daniels and Oldridge, 1970; Gore *et al*. 1998) and controlled (Bonne *et al*. 2014;

Rodriguez *et al*. 2015) investigations examining the effect of LHTH on performance involving elite populations, with improvements ranging from ~ 1 to 4% reported.

The question of the optimal time to compete following altitude training has received considerable attention, with the focus being on the decay of physiological adaptations conferred by altitude training (Chapman *et al*. 2014b), including the increase in Hb_{mass} and ventilatory acclimatisation, as well as changes in neuromuscular factors (e.g. changes in stride mechanics, ground contact time, muscle recruitment). A ~ 5% increase in Hb_{mass} was observed following LHTH in the current study (**Figure 5.4A**), an expected finding in accordance with previous studies (Gore *et al*. 2013; Rodriguez *et al*. 2015) and the hypoxic dose experienced by the athletes (Garvican-Lewis *et al*. 2016). However, the range of observed increases (1 to 9%) was quite large. It has been previously noted that certain individuals, based on genetic predisposition, may well have a beneficial response to lower "doses" of altitude, whilst others may require longer exposure to altitude to induce adaptations (Levine and Stray-Gundersen, 2006). Additionally, age may have played a role, with older athletes (> 26 years) increasing Hb_{mass} by only ~ 3% compared to their younger counterparts (8% increase). Whilst experienced, elite athletes are frequently observed to increase Hb_{mass} following altitude training (Millet *et al*. 2017), the lower relative training age and potentially greater room for adaptation in the younger athletes may have contributed to their superior response.

Given the largely aerobic nature of middle distance events (Gastin, 2001), the increase in Hb_{mass} would theoretically be advantageous to performance. However, if altitude induced increases in Hb_{mass} are quickly negated upon return to sea-level, this could affect the optimal timing of competition. In elite Kenyan runners, Hb_{mass} remained stable for the first 14 days at sea-level but declined by 6% after five weeks (Prommer *et al*. 2010). Therefore, acquired Hb_{mass} was likely still at or close to its peak during the 8 day post-altitude window during which the majority of participants competed in the current study. Alternatively, training with

additional Hb_{mass} is also beneficial, and may explain increased performance several weeks following altitude training, regardless of whether Hb_{mass} has returned to baseline levels (Chapman *et al*. 2014b). Accordingly, participant 8 commenced competition 4 weeks following LHTH, and achieved a personal best on day 57 post-altitude.

Submaximal pulmonary $\dot{V}_E$ was increased (albeit not significantly) by ~ 8% following LHTH (**Figure 4B**), with 7 of 8 athletes recording increases, again consistent with previous literature (Daniels and Oldridge, 1970; Levine and Stray-Gundersen, 1997). Although we measured $\dot{V}_E$ in this study while athletes were still living in hypoxia, previous research has shown this increase persists upon return to sea-level (Daniels and Oldridge, 1970) for at least four days (Levine and Stray-Gundersen, 1997). Interestingly, 4 athletes that raced at sea-level within this time-frame recorded a personal record, with 3 being over 800 or 1500 m; another 3 athletes racing over 1500 m on days 6, 7 and 8 post-altitude respectively, also recorded personal bests. Ventilatory adaptations may be worthy of further investigation to determine the time course of decay following altitude training. Such knowledge may be important in scheduling races (dependent on event distance) or targeting a specific anaerobic training block post-altitude.

Training

Periods of intensified training are inherent to elite endurance athletes and intended to stimulate adaptations that may improve performance (Le Meur *et al*. 2014). These training blocks can manifest in the form of specific training camps, and for elite endurance athletes, often occur at altitude. In accordance with previous studies (Levine and Stray-Gundersen, 1997; Bonne *et al*. 2014), there was a significant increase in TL at altitude compared to sea-level reported in the current investigation (**Figure 5.1A**).

A moderate increase in TV at altitude (but not higher than completed during normal training at sea-level [**Figure 5.1B**]) compared to the preceding 4 week period of training at sea-level was observed. However, the increase in TL for from lead-in to LHTH exceeded the magnitude of change in TV over the same period, with a 44% increase in TV contributing to a 77% increase in TL (23 and 51% for TV and TL respectively, if participant 6 who experienced illness during the lead-in is excluded). The increased load:volume ratio at altitude (**Figure 5.2**), suggests that for each kilometre of running completed, the training load would be 17% higher at altitude than at sea-level. The higher "per kilometre load" observed here may therefore be attributable to increased overall fatigue by virtue of increasing TV, or increased perception of effort due to the added stress of hypoxia, however our uncontrolled study design does not allow the separation of these two variables.

In addition to an overall increase in load during LHTH, an interesting aspect was the timing of the increase immediately upon arrival to altitude. Further analysis of athlete training diaries revealed that in most cases, athletes performed their first intense training sessions (around lactate threshold) on day 4 of LHTH, and completed some race pace efforts towards the conclusion of the first week (**Table 5.2**). Even more aggressive were those athletes with an extensive history of altitude training of several years, who completed a progression run at threshold intensity on day 2, and some short race pace efforts on day 4 (**Table 5.2**). Such a practice contrasts with general recommendations regarding altitude training (Millet *et al*. 2010), and typically, the initial acclimatisation period to altitude (first 1-2 weeks) is restricted to low intensity training (Lange, 1986; Levine and Stray-Gundersen, 1997; Wilber, 2004; Bonne *et al*. 2014; Rodriguez *et al*. 2015). It has been proposed that elite athletes accustomed to frequent use of altitude training may be able to maintain the absolute intensity of exercise in moderate hypoxia, as well as commence high intensity training within a couple of days of ascent to altitude (Daniels and Oldridge, 1970; Pugliese *et al*. 2014).

Frequent users of altitude may experience a faster acclimatisation response (Millet *et al*. 2010). Anecdotally, coaches have suggested that experienced athletes who have previously undertaken altitude training tend to adapt faster and are able to achieve sea-level intensity in aerobic and anaerobic workouts (Baumann *et al*. 1994; Wilber, 2004). The physiological and molecular evidence supporting the repeated use of altitude training by elite athletes is emerging. Ventilatory acclimatisation has been shown as beneficial for high intensity exercise performance at altitude in elite cyclists (Townsend *et al*. 2017). Studies have demonstrated that a degree of ventilatory acclimatisation is retained upon re-exposure to high altitude (Subudhi *et al*. 2014; Song *et al*. 2017), which may allow athletes to commence high intensity exercise sooner than previous exposures. Recent studies show that in sea-level natives, plasma adenosine levels are rapidly induced by initial ascent to high altitude, and achieved even higher levels upon re-ascent, a feature that is positively associated with quicker acclimatization (Song *et al*. 2017). In other words, erythrocytes possess a "hypoxic memory" that could facilitate a faster acclimatisation to high altitude when participants have previous altitude exposure experience (Song *et al*. 2017).

Whilst the early increase in TL was large during LHTH, athletes were simply returning to the TV they were accustomed to regularly completing (**Figure 5.1B**). Due to factors such as international travel to Flagstaff during the last week of the lead-in phase, tapering for competition immediately preceding LHTH in the case of 3 athletes, and wanting to arrive at altitude in a fatigue free state conducive to adaptation, TV during this period was reduced compared to normal. It is important to clarify the return to normal TV observed in these athletes – due to the hypoxic environment already placing limitations on training, it would be unwise to increase TV further to previously unaccustomed levels, which may appear the case. Furthermore, in lesser trained athletes, or those without prior experience with LHTH, it would perhaps be ill-advised to immediately increase training intensity at altitude to the magnitude

seen in this study, irrespective of whether they arrived in a fresh state. Finally, international travel of the athletes in this study created an automatic reduction in TL, with training sessions missed due to long haul travel, adjusting to a time zone shift, and recovering from jet lag. It may be optimal for athletes completing altitude training domestically (i.e. without extensive travel required) to actively decrease TL during the lead-in to a camp to facilitate commencing altitude exposure in a fresh, fatigue-free state.

Tapering

The athletes in the current investigation included a substantial taper as a part of their altitude training, with reductions of 33 and 43% in TV and TL respectively during the final week of LHTH, a reduction in line with a meta-analysis by Bosquet and colleagues (2007), which suggested an optimal tapering strategy should involve a 41 to 60% reduction in TV over a period of two weeks. When they considered running separately (n = 9 studies), it was found a reduction in volume of 21 to 40% led to the greatest improvements. A study observing the tapering practices of elite British middle-distance athletes (Spilsbury *et al*. 2015) reported a reduction in TV of 30 to 40% in the week preceding competition. The authors determined that amongst other factors, the nature of the taper was heavily influenced by the content of training undertaken prior (Spilsbury *et al*. 2015). Levels of fatigue preceding the taper also might influence the chosen strategy (Bosquet *et al*. 2007), with athletes undertaking a large volume of running perhaps requiring a large reduction in training to alleviate accumulated fatigue (Spilsbury *et al*. 2015). Such considerations are particularly important at altitude, as training sessions completed in hypoxia evoke a higher physiological load than equivalent sessions completed in normoxia (Mazzeo, 2008; Saunders *et al*. 2009a) leading to greater accumulated fatigue than when training at sea-level (Schmitt *et al*. 2018). Supporting this notion, a distance runner participating in this study completed a higher volume of training than the middle-distance runners, and consequently undertook a more severe taper in training load to prepare

for competition (**Figure 5.3C**). Investigations observing minimal tapers in volume (5-10%) during three weeks of LHTH at 2300 m in elite swimmers (Gough *et al*. 2012; Rodriguez *et al*. 2015) have reported slower or unchanged performance immediately post altitude training. Such small reductions in TV may not be sufficient to dissipate the accumulated fatigue altitude training imposes and may explain the unclear performance findings. Alternatively, altitude training studies reporting larger magnitude tapers also reported performance improvements of ~ 1% (Rodriguez *et al*. 2015). Consequently, tapering would appear to have added relevance for athletes undertaking altitude training with the intention of competing soon thereafter, with a substantial taper in volume (~ 40%) likely necessary for peak performance. Tapering to this extent would likely not be necessary for those athletes using LHTH differently (i.e. in early season general preparation, or choosing to race a few weeks after completion). However, it could be argued that a short sharp taper would freshen up the athlete for the subsequent period of hard training, effectively taking advantage of the beneficial adaptations conferred by altitude training.

Limitations

A limitation of the current investigation is the absence of a control group. It is therefore beyond our scope to definitively establish LHTH as superior with respect to improving subsequent performance to reciprocal sea-level training in elite runners. However, our aim was not to answer this complicated question, but rather to quantify the training periodisation of elite athletes undertaking LHTH immediately prior to competition. Increased TL and hypoxic exposure are inextricably linked, and with this observational case study design involving elite athletes completing individualised training, we further acknowledge it is difficult to isolate which is the dominant of the two variables when explaining our performance results. Finally, the participants were aware they were under observation as part of a scientific investigation, which in addition to the "training camp effect", may have contributed to behavioural changes

leading to improvement in competition performance following altitude training. We would argue however, the athletes were motivated primarily by achieving the fastest times possible in post-altitude competition (e.g. to gain qualification into major competition), and participation in a scientific observation was unlikely to affect the manner in which they trained in attempting to achieve this goal. However, we acknowledge that our unblinded and uncontrolled study design restricts us from parsing out the true effect of altitude adaptation on sea-level endurance exercise performance from any of these confounders.

5.6 CONCLUSIONS

In this cohort of elite runners, training load was intensified from the very beginning of LHTH through a combination of an increased volume from the lead in period and training under hypoxic stress, a strategy differing from current recommendations that advocate reduced/conservative training for the initial 1-2 weeks at altitude. A 1 week taper, and finally a hypoxia induced increase in Hb_{mass} were also observed prior to athletes achieving personal best competition performances.

6.0 STUDY FOUR: INCREASED TRAINING LOAD AT 1600 AND 1800 m IMPROVES PERFORMANCE IN NATIONAL LEVEL RUNNERS.

Citation: **Sharma AP**, Saunders PU, Garvican-Lewis LA, Clark B, Welvaert M, Gore CJ Thompson KG. Increased training load at 1600 and 1800 m improves performance in national level runners. *Int J Sports Physiol Perform.* 2018 Aug 6. doi: 10.1123/ijspp.2018-0104.

6.i FORM E: DECLARATION OF CO-AUTHORED PUBLICATION CHAPTER

Declaration for Thesis Chapter [6]

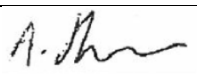
Declaration by candidate

In the case of Chapter [6], the nature and extent of my contribution to the work was the following:

Nature of contribution	**Extent of contribution (%)**
Study design, data collection and analysis, study write-up	70

The following co-authors contributed to the work.

Name	**Nature of contribution**	**Extent of contribution**	**Contributor is also a student at UC Y/N**
Philo Saunders	Assistance with design, proof reading, editing of drafts	7	N
Laura Garvican-Lewis	Assistance with design, proof reading, editing of drafts	5	N
Brad Clark	Assistance with data collection, data analysis, proof reading, editing of drafts	5	N
Marijke Welvaert	Assistance with data analysis, proof reading, editing of drafts	3	N
Christopher Gore	Assistance with data collection, proof reading, editing of drafts	5	N
Kevin Thompson	Assistance with design, proof reading, editing of drafts	5	N

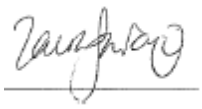
Candidate's Signature		**Date** **12.6.2018**

Declaration by co-authors

The undersigned hereby certify that:

(1) The above declaration correctly reflects the nature and extent of the candidate's contribution to this work, and the nature of the contribution of each of the co-authors.
(2) they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least that part of the publication in their field of expertise;
(3) they take public responsibility for their part of the publication, except for the responsible author who accepts overall responsibility for the publication;
(4) there are no other authors of the publication according to these criteria;
(5) potential conflicts of interest have been disclosed to (a) granting bodies, (b) the editor or publisher of journals or other publications, and (c) the head of the responsible academic unit; and
(6) the original data are stored at the following location(s) and will be held for at least five years from the date indicated below:

Location(s)	Australian Institute of Sport Department of Physiology, Bruce, ACT Research Institute for Sport and Exercise, University of Canberra, Bruce ACT

Signature 1		**Date** 12.6.2018
Signature 2		12.6.2018
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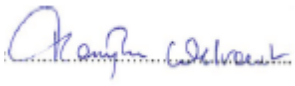
Signature 4		12.6.2018
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6.1 ABSTRACT

Purpose: We aimed to determine the effect of altitude training at 1600 and 1800 m on sea-level performance in national-level runners.

Methods: After three weeks of sea-level training, 24 runners completed a three week sojourn at 1600 m (ALT1600, *n* = 8), 1800 m (ALT1800, *n* = 9) or sea-level (CON, *n* = 7), followed by up to 11 weeks of sea-level racing. Race performance was measured at sea-level during the lead-in period and repeatedly post-intervention. Training volume (km) and load (session RPE) were calculated for all sessions. Haemoglobin mass (Hb_{mass}) was measured via CO rebreathing. Between-group differences were evaluated using effect sizes (Hedges' *g*).

Results: Performance improved within both ALT1600 (mean ± SD; 1.5 ± 0.9%) and ALT1800, (1.6 ± 1.3%) compared to CON (0.4 ± 1.7%); *g* = 0.83 (90% confidence limits; - 0.10, 1.66) and 0.81 (- 0.09, 1.62), respectively. Season best performances occurred 5-71 days post-altitude in ALT1600 and ALT1800. There were large increases in training load from lead-in to intervention within ALT1600 (48 ± 32%) and ALT1800 (60 ± 31%) compared to CON (18 ± 20%); *g* = 1.24 (0.24, 2.08) and 1.69 (0.65, 2.55) respectively. Hb_{mass} increased in ALT1600 and ALT1800 (~ 4%), but not CON.

Conclusions: Larger improvements in performance after altitude training may be due to the greater overall load of training in hypoxia compared to normoxia, combined with a hypoxia mediated increase in Hb_{mass}. A wide timeframe for peak performances suggests the optimal window to race post-altitude is individual, and factors other than altitude exposure *per se* may be important.

6.2 INTRODUCTION

Altitude training is used by athletes to improve sea-level performance subsequent to adaptations acquired during altitude acclimatisation and/or associated training in hypoxia (Saunders *et al*. 2009a). Two questions consistently debated regarding the use of altitude training are 1) how high to live/train, and 2) when to compete following altitude training for optimal performance (Baumann *et al*. 1994; Wilber *et al*. 2007; Chapman *et al*. 2014a; Chapman *et al*. 2014b).

Recommendations for altitude training typically suggest living at 2000-2500 m for three to four weeks to elicit haematological adaptations purported to improve endurance performance (Wilber *et al*. 2007). A dose-response model of altitude training has been proposed, based on the belief that a hypoxia-induced increase in erythropoietin (EPO) is the primary physiological pathway enhancing post-altitude sea-level performance (Wilber *et al*. 2007). Recent reviews of the literature suggest that haemoglobin mass (Hb_{mass}), a key measurable outcome of the erythropoietic cascade, increases in a dose dependent manner following altitude training (Garvican-Lewis *et al*. 2016). Accordingly, several investigations of elite athletes completing altitude training in line with these recommendations report concurrent improvements in Hb_{mass} and sea-level performance (Stray-Gundersen *et al*. 2001; Bonne *et al*. 2014; Rodriguez *et al*. 2015).

Topographical (e.g. Australia) and seasonal (e.g. Northern Hemisphere winter) limitations may restrict the athletes' ability to access altitude training venues > 2000 m. Athletes therefore undertake "low" altitude (1000-2000 m) training in the belief it aids competition performance. While low altitudes are favourable in maintaining training intensity, and therefore promote peripheral adaptations advantageous to performance (Gore *et al*. 2007), the hypoxic dose likely falls at the lower end of what is sufficient to induce haematological adaptations. Performance improvements independent of changes in Hb_{mass} following altitude

training are reported in world-class cyclists (Gore *et al*. 1998), suggesting other mechanisms which may not be as dose dependent are relevant (Gore *et al*. 2007). Improvements in haematological, physiological and performance measures are observed in athletes following low altitude training (Roels *et al*. 2006; Saunders *et al*. 2009b; Frese and Friedmann-Bette, 2010; Wachsmuth *et al*. 2013; Garvican-Lewis *et al*. 2015a), however these improvements are not as consistently observed in comparison to adaptations induced by moderate altitudes > 2000 m (Wilber *et al*. 2007; Chapman *et al*. 2014a). Indeed, other investigations report impaired or unchanged performance, Hb_{mass} and maximal oxygen uptake ($\dot{V}O_{2max}$) following altitude training between 1619-1822 m (Gore *et al*. 1997; Bailey *et al*. 1998; Chapman *et al*. 2014a). These conflicting results provide the impetus for further inquiry into the efficacy of low altitude training for sea-level performance enhancement in elite athletes.

The optimal time to compete following altitude training is an important issue for athletes, coaches and scientists. Anecdotal evidence suggests competing within 48-72 hours upon descending from altitude, or after two to three weeks of re-acclimatisation to sea-level is optimal (Baumann *et al*. 1994; Millet *et al*. 2010; Chapman *et al*. 2014b). Improved performance at both these time-points has been observed in scientific investigations with elite athletes (Stray-Gundersen *et al*. 2001; Wachsmuth *et al*. 2013), however only a handful of studies include serial performance measures following altitude training to identify the optimal window for competition (Levine and Stray-Gundersen, 1997; Gore *et al*. 1998; Saunders *et al*. 2009b; Wachsmuth *et al*. 2013; Rodriguez *et al*. 2015). Investigations of elite swimmers report impaired or unchanged performances compared to baseline immediately following altitude training, with peak performances occurring two to four weeks thereafter (Wachsmuth *et al*. 2013; Rodriguez *et al*. 2015). Studies involving runners demonstrate improved performance both immediately following altitude exposure, and after a period of sea-level training (Levine and Stray-Gundersen, 1997; Saunders *et al*. 2009b).

A confounding factor when evaluating performances following altitude exposure is the nature of training completed. Many studies are limited in only reporting overall training volume or duration (Bailey *et al*. 1998; Saunders *et al*. 2009b), excluding most information regarding periodisation and distribution of training intensity, which with the decay of physiological adaptations conferred by altitude training (Chapman *et al*. 2014b), strongly influences the timing of a peak performance. Adequate quantification of training in low altitude training studies may provide the context required for evaluation of performances and assist in determining the optimal window/s for competition.

We therefore aimed to determine the effect of an in-season, pre-competition block of living and training at 580, 1600 or 1800 m in national-level runners on Hb_{mass} and sea-level performance during the subsequent competition period. Training volume and load were also monitored to help contextualise any changes in performance.

6.3 METHODS

Subjects

Twenty-four runners participated in the investigation, and characteristics at baseline are presented in **Table 6.1**. Nineteen athletes competed at the national championships (Open or Junior) and 13 had represented Australia internationally at Olympic, Paralympic, IAAF, Commonwealth or Oceania competitions. After completing three weeks of quantified training in their home environment at or close to sea-level, athletes completed three weeks of living/training at either 580 m (CON), 1600 m (ALT1600) or 1800 m (ALT1800). Within the two altitude training groups, 7 of 8 participants in ALT1600 and 7 of 9 in ALT1800 had previously experienced living/training at altitude. All procedures and risks were explained to participants before they provided written consent to participate. Parental consent was also obtained for participants under the age of 18 (n = 6). Ethical approval was granted by the Australian Institute of Sport ethics committee and all procedures complied with the Declaration of Helsinki.

Table 6.1. Participant characteristics

Group	Participant	Sex	Age (y)	Height (cm)	Body mass (kg)	Predicted VO_{2max} ($mL \cdot kg^{-1} \cdot min^{-1}$)	Preferred event	2-year best (mm:ss)
ALT1600	1	F	17	161	51	52	1500 m	04:18
	2	F	17	162	51	49	800 m	02:07
	3	F	16	173	56	51	800 m	02:11
	4	M	16	175	68	77	1500 m	03:52
	5	M	32	183	76	71	1500 m	03:42
	6	F	17	171	54	56	800 m	02:16
	7	M	25	178	68	68	3000 m	08:07
	8	M	23	179	69	72	800 m	01:49
Mean			21	174	62	62		
SD			5	8	9	11		
ALT1800	9	M	24	184	58	54	3000 m	09:11
	10	M	39	186	73	75	1500 m	03:46
	11	M	25	176	66	69	1500 m	04:12
	12	M	22	182	72	71	1500 m	03:40
	13	M	24	177	67	68	5000 m	14:11
	14	M	19	190	69	68	1500 m	03:55
	15	M	20	179	72	63	1500 m	04:05
	16	M	20	191	71	70	1500 m	04:09
	17	M	16	170	55	67	3000 m	08:49
Mean			23	182	67	67		
SD			6	7	6	5		
CON	18	M	35	177	60	72	5000 m	14:43
	19	M	28	179	60	73	1500 m	03:48
	20	M	23	184	73	71	3000 m	08:18
	21	M	35	191	91	72	5000 m	16:11
	22	F	24	169	51	60	800 m	02:07
	23	M	20	175	60	70	800 m	01:53
	24	M	25	176	68	68	5000 m	16:00
Mean			27	179	66	70		
SD			5	7	12	4		

2-year best = best time in competition in 2 seasons prior to study commencing

Study design

The investigation was a non-randomised, multi-centre, parallel group study observing the training and competition performance of elite middle-distance runners during and after an in-season training intervention. Training sessions and competition schedules were designed by the participants' coaches and were not manipulated or directly influenced as part of the study.

Athletes' training was monitored for nine to 17 weeks and divided into three phases. The "lead-in phase" was similar for all groups in that they completed three weeks of their own, coach-prescribed training in their home environment at or close to sea-level (Canberra, 580 m elevation or Sydney, 19 m elevation, Australia). Immediately following, CON athletes continued training in their home environment for an additional three weeks (Canberra), whereas ALT1600 (all Sydney based) and ALT1800 athletes (all Canberra based) travelled to Australian venues of Falls Creek (1600 m elevation) and Perisher (1800 m elevation) respectively, to complete three weeks of altitude training ("intervention phase"). Participants then completed a competition period (one to eight races) of up to 11 weeks ("competition phase") during which they raced in sanctioned events at or close to sea-level (0 to 580 m). All participants in ALT1600 and ALT1800 supplemented with oral iron (Ferro-Grad C, Abbott Laboratories, Australia, 105 g elemental iron) daily for at least one week prior to and for the duration of the altitude training camp to help ensure erythropoietic adaptations were not compromised by insufficient iron availability, however we were unable to confirm this via collection of serum ferritin measurements due to logistical constraints. Hb_{mass} testing occurred immediately pre and post the intervention phase at or close to sea-level (ALT1600: Sydney, CON and ALT1800: Canberra). Each participant completed their pre and post testing on identical laboratory equipment (OSM3 hemoximeter, Radiometer, Copenhagen, Denmark), although due to the multi-centre nature of the investigation, the hemoximeter used was different

between groups (hemoximeter A, Sydney, ALT1600; hemoximeter B, Canberra, CON and ALT1800). The same researcher performed all Hb_{mass} tests.

Training

Athletes completed six to eleven running, and one to two strength training sessions per week. Each week typically consisted of; one to three high intensity running sessions, a long run (70-120 min) and lower-intensity aerobic continuous running (30-70 min). Within this structure, training intensity and duration of sessions was individualised by coaches for each athlete based on preferred event (800-5000 m), competition schedule and physiological characteristics.

The lead-in and intervention periods of the investigation occurred immediately prior to the Australian domestic athletics season in late January to April. Major competitions occurred later during the season (> six weeks following the intervention period, e.g. National Championships), and thus training for most athletes was focused around peaking for these races. However, all athletes also competed within three weeks of the intervention period, and therefore retained certain aspects of training (race pace sessions, shorter tapers) to specifically prepare for these races. The 19 athletes competing at the national championships focused training around peaking for this event, with the remaining five athletes (ALT1800, $n = 2$; CON, $n = 3$) targeting state level races within the first four weeks post intervention as their primary focus. During the intervention period, training was completed at the living altitude, except for participants in ALT1800, who completed two to four sessions over the three weeks on a 400 m athletics track at 1000 m altitude (all other training in ALT1800 completed at 1700-2200 m).

Training monitoring

Each athlete recorded all their running training on a GPS watch (Forerunner, Garmin International, Kansas, USA). For all running sessions, athletes recorded the total distance

(kilometres) and duration (minutes). Duration was also recorded for cross training or strength training sessions. Additionally, athletes provided a session rating of perceived exertion (sRPE) score (0-10) on a modified Borg scale for all training sessions (Foster, 1998). Weekly training volume (TV) was defined as the total running distance completed each week in kilometres. Daily training load (TL) was calculated using the session RPE method (duration of each training session multiplied by the sRPE score) and summated to give weekly TL. Training Stress Balance (TSB) was calculated as the ratio of acute TL (7-day rolling average) to chronic TL (28-day rolling average) and expressed as a percentage.

Performance

Race times of athletes were collated as a record of performance. Of the 24 athletes, 6 were 800 m runners (ALT1600, n = 4; CON, n = 2), 10 were 1500 m runners (ALT1600, n = 3; ALT1800, n = 6; CON, n = 1) and 8 were 3000 or 5000 m runners (ALT1600, n = 1; ALT1800, n = 3; CON, n = 4). Some also competed in other events (800 m, n = 11; 1500m, n = 16; 3000 m, n = 11; 5000m, n = 7). Performances were recorded before and after the intervention period. The season's best time achieved in competition within the two months prior to the beginning of the intervention period was used as the pre-altitude measure. In cases where athletes did not compete in a certain event within this period (n = 3), their best time achieved in the previous two seasons was used as the pre-altitude measure. Participants' pre-intervention times in their preferred events as a percentage of their best time achieved in the previous two seasons was 101.0 ± 1.1%, 100.6 ± 0.8% and 101.0 ± 1.6% for ALT1600, ALT1800 and CON respectively. Race times achieved in competition post altitude were compared to pre-intervention values with percentage differences calculated and used to indicate change in race performance. Post altitude races were completed between 5 and 82 days following the intervention period according to individualised racing schedules. All races were completed near to sea-level (0-580 m) on standard 400 m athletics tracks. Athletes were free

to employ their own preparations and use of ergogenic aids such as caffeine, but were asked to keep this consistent between races.

Haemoglobin mass

Total Hb_{mass} was measured using the two-minute CO rebreathing method (Schmidt and Prommer, 2005) with some modifications. Participants rebreathed a CO bolus equivalent to 1.2 $mL \cdot kg^{-1}$ of body weight for a period of two minutes. Capillary blood samples were drawn at the start of the test as well as at seven minutes post administration of the CO dose for determination of the percentage of bound carboxyhaemoglobin (%HbCO). Blood samples were measured five times for %HbCO using a CO-oximeter (OSM3, Radiometer, Copenhagen, Denmark). Expired CO was determined using a Draeger Pac 7000 (Lubeck, Germany) CO sensor. Hb_{mass} was calculated from the mean change in %HbCO before and after rebreathing. The typical error of measurement for Hb_{mass} of the investigator administering all tests was 1.5%. Predicted $\dot{V}O_{2max}$ was calculated based on Hb_{mass} using a previously described regression equation (Saunders *et al*. 2013).

Statistical Analyses

The magnitude of changes within groups and differences between groups for weekly TL, TV, TSB, Hb_{mass} and performance were quantified using standardised mean difference (Hedges' *g* effect sizes), whereby the change in parameter values were divided by the pooled standard deviation. Additionally, percentage changes were calculated and expressed with standard deviation (SD). Effects sizes were interpreted using effect thresholds of < 0.2, 0.2, 0.5, and 0.8 for trivial, small, moderate, and large effects respectively and expressed with 90% confidence limits (CL) to denote the imprecision of the estimate. Data are displayed as mean ± SD unless otherwise stated.

6.4 RESULTS

Performance

Individual season best performance changes are shown in **Figure 6.1**. There were large improvements in race time within ALT1600 (-1.5 ± 0.9%) and ALT1800, (-1.6 ± 1.3%) when compared to CON (-0.4 ± 1.7%); g = 0.83 (-0.10, 1.66) and 0.81 (-0.09, 1.62) respectively. Four of seven, seven of eight and all nine participants within CON, ALT1600 and ALT1800, respectively, achieved performance improvements compared with their season's best times prior to the intervention. The season's best performance (denoted by largest improvement [n = 20] or smallest impairment [n = 4] compared to pre-intervention time) for all participants except two athletes (one each in ALT1600 and CON) occurred in their preferred event. Season's best performances occurred between 5 and 71 days post-altitude in ALT1600 and ALT1800 (**Figure 6.2B**), and TSB on the day of these performances ranged from 50 to 151 (**Figure 6.2C**).

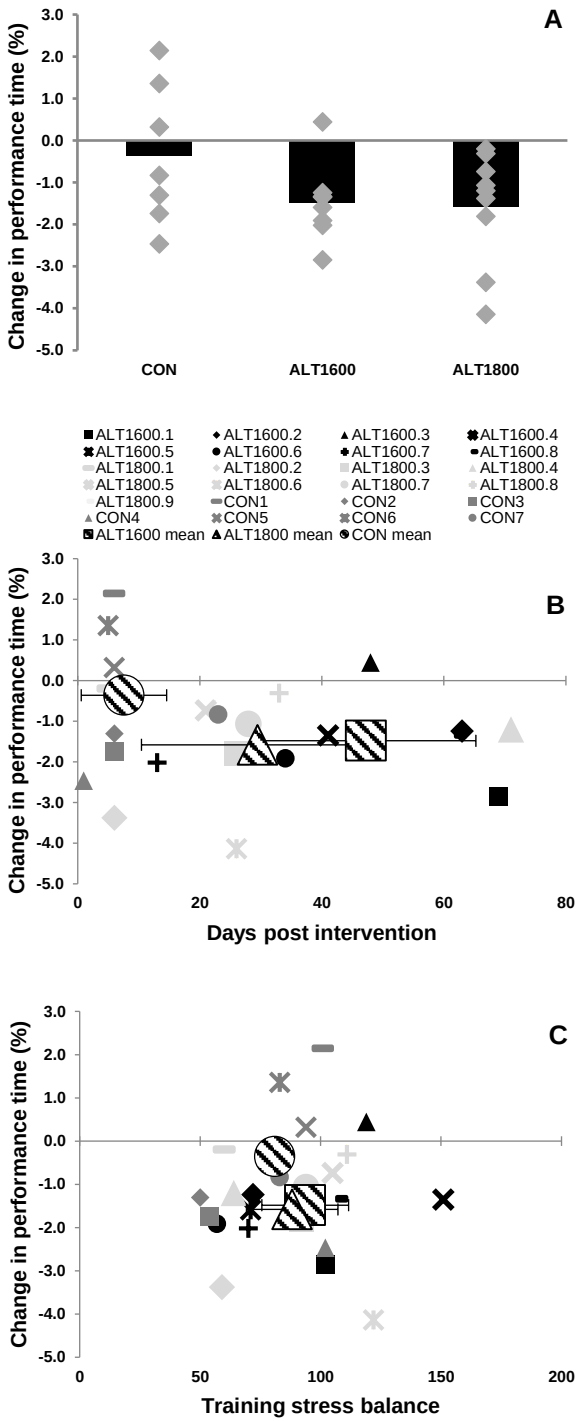

Figure 6.1. Performance changes following three weeks of living and training at 580 m (CON), 1600 m (ALT1600) and 1800 m (ALT1800). A) Group and individual performance changes. Negative values indicate faster times relative to pre-intervention season's best performances. Group means are indicated by histograms and diamonds denote individual responses. B and C) Individual post-intervention season's best performances vs. days post intervention (B) and training stress balance (C) when performance was achieved. Performance changes are relative to pre-intervention season's best and negative values indicate faster times. Legend applies to both figures 6.1B and 6.1C.

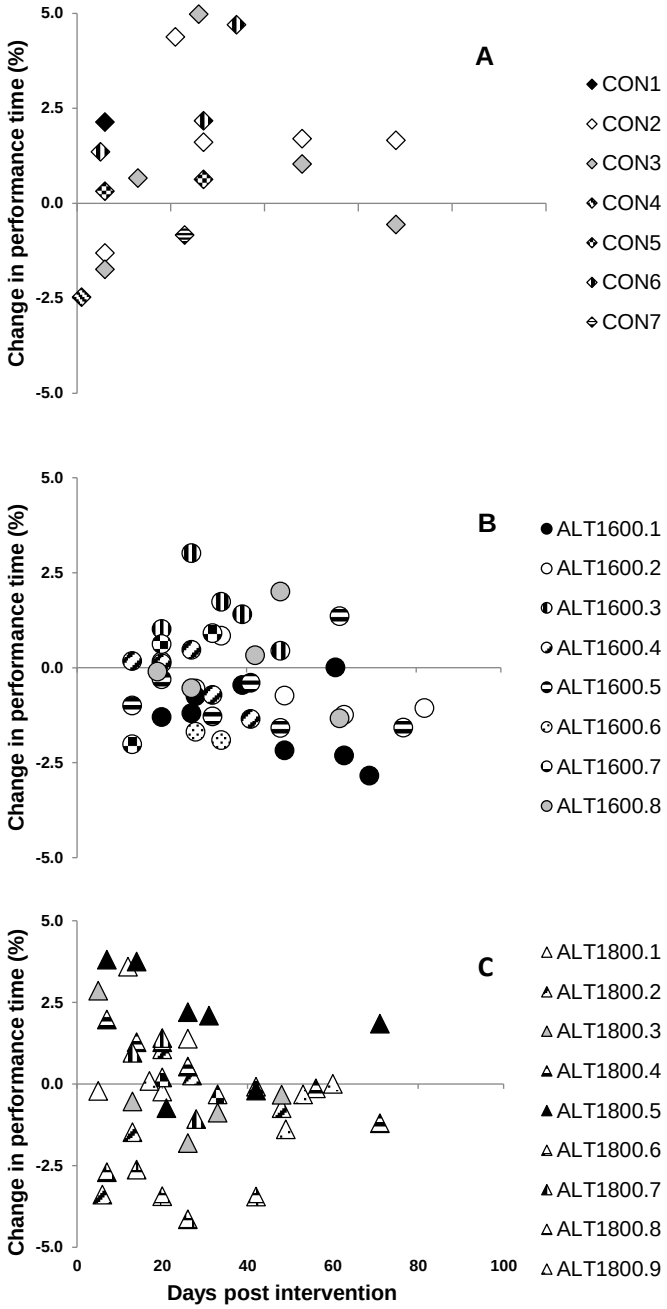

Figure 6.2. Individual time course of changes in performance time post intervention for CON (A), ALT1600 (B) and ALT1800 (C) respectively. Performance changes are relative to pre-intervention season's best times and negative values indicate faster times.

The individual time course of performance changes following the intervention period are shown in **Figure 6.2**. When individual post-intervention race performances from ALT1600 and ALT1800 were pooled (**Figure 6.3**) and grouped by time point into weeks post-altitude exposure, there were no differences between time points. The greatest variability in performances (i.e. highest SD) was observed for races completed within one week post-altitude (2.7%), with lower variability observed for major competitions occurring six to nine weeks post-altitude (1.0%).

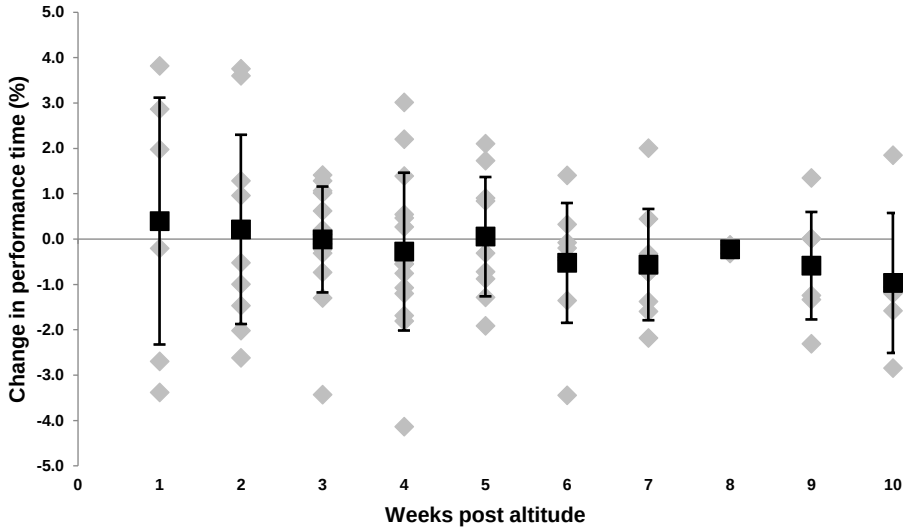

Figure 6.3. Time course of performance changes following altitude training. Individual post-intervention performances from ALT1600 (n = 41) and ALT1800 (n = 42) have been pooled and grouped into the week post altitude in which they occurred (e.g. week 1 races occurred between days 1 and 7 inclusive, week 2 days 8 to 14, and so on). Black squares represent the mean ± SD performance change for each week post altitude, and diamonds denote individual races. Negative values indicate faster times relative to baseline.

Within the first four weeks following the intervention, there were moderate improvements in race time within ALT1600 (-0.7 ± 0.9%) and ALT1800, (-1.0 ± 1.3%) when compared to CON (0.1 ± 1.6%); g = 0.59 (-0.28, 1.46) and 0.72 (-0.13, 1.58) respectively, with all participants competing within this period. Changes in race times from competitions four or more weeks following the intervention were -1.0 ± 1.4% (ALT1600), -1.0 ± 1.1% (ALT1800)

and 1.9 ± 2.2% (CON) with all 8, 8 of 9, and 3 of 7 participants respectively, competing during this period. Within ALT1600 and ALT1800, there were small (g = 0.25 [-0.58, 1.07]) and trivial (g = 0.00 [-0.83, 0.83]) differences in performance when comparing the two racing periods post-intervention.

Compared to baseline, changes in performance time after the intervention (negative changes are faster times relative to baseline) specific to event were as follows: 800 m specialists (800 m - ALT1600, n = 4, -0.9 ± 1.1%, 3 improved; CON, n = 2, 0.9 ± 0.6%, 0 improved. 1500 m – ALT1600, n = 1, 0.4 ± 0.0%, 0 improved); 1500 m specialists (1500 m - ALT1600, n = 3, -1.7 ± 0.9%, 3 improved; ALT1800, n = 6, -1.7 ± 1.2%, 6 improved; CON, n=1, 1.6 ± 0.0%, 0 improved. 800 m - ALT1600, n = 1, -2.3 ± 0.0%, 1 improved; ALT1800, n=3, -0.3 ± 2.7%, 1 improved. 3000/5000 m - ALT1600, n = 2, -0.4 ± 0.6%, 1 improved; ALT1800, n = 3, -1.4 ± 2.4%, 2 improved; CON, n = 1, -1.3 ± 0.0%, 1 improved); 3000/5000 m specialists (3000/5000 m - ALT1600, n = 1, -2.0 ± 0.0%, 1 improved; ALT1800, n = 3, -0.8 ± 0.5%, 3 improved; CON, n = 4, -0.7 ± 1.7%, 3 improved. 1500 m - ALT1600, n = 1, 0.6 ± 0.0%, 0 improved; ALT1800, n = 3, 1.0 ± 0.9%, 1 improved; CON, n = 1, -0.6 ± 0.0%, 1 improved).

Training

Periodisation of weekly TV and TL is shown in **Figure 6.4**. During the lead-in period athletes in ALT1600, ALT1800 and CON completed on average 78 ± 29, 74 ± 27 and 81 ± 26 km of training per week respectively (**Figure 6.4A**), with differences between groups being trivial to small (g = 0.11-0.26). During the intervention period, athletes in ALT1600, ALT1800 and CON completed on average 100 ± 26, 91 ± 26 and 92 ± 29 km of training per week respectively, with differences between groups being trivial to small (g = 0.04 - 0.35). Increase in TV from lead-in to intervention was on average 33 ± 16% in ALT1600, 29 ± 31% in ALT1800 and 14 ± 11% in CON (**Figure 6.4B**), with all participants increasing volume over this period (range ALT1600; 6-58%, ALT1800; 2-109%, CON; 6-35%).

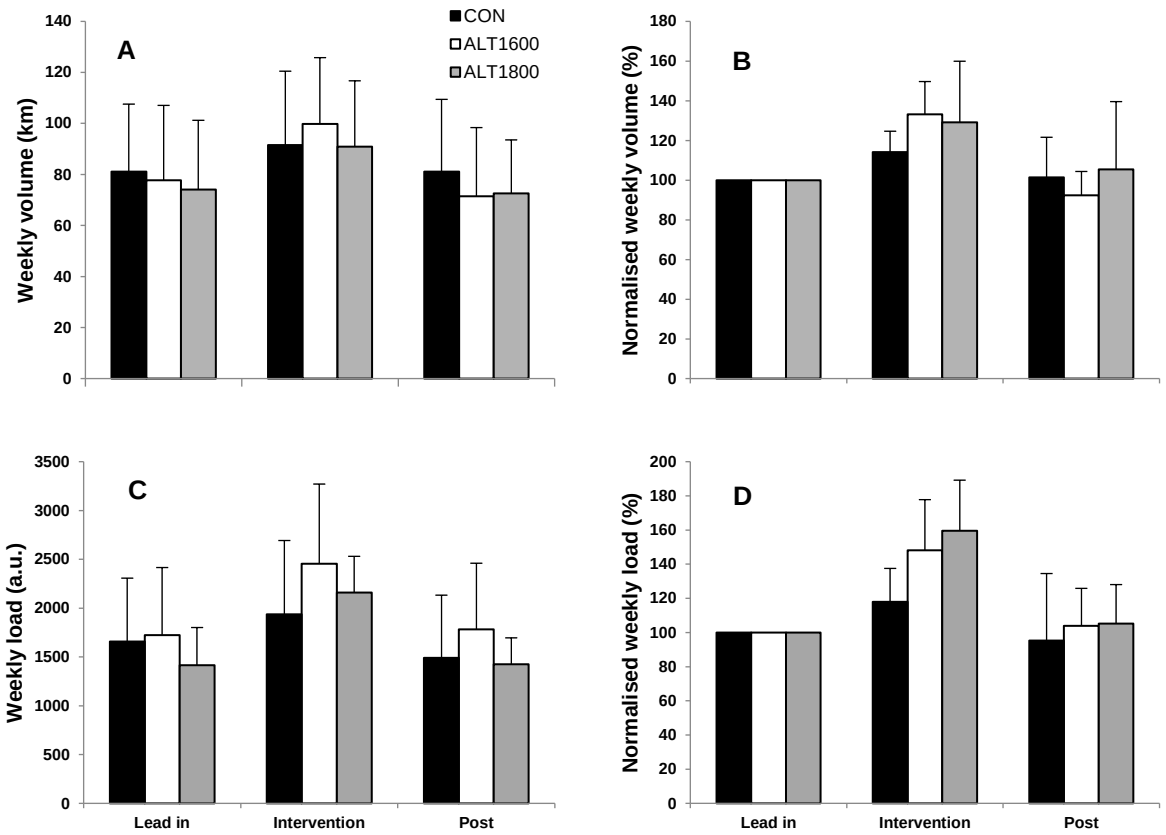

Figure 6.4. Periodisation of weekly training volume and load. A and B). Quantification of weekly training volume during the lead-in, intervention and post periods, in (A) total running kilometres and (B) normalised weekly volume (to weekly volume completed during the lead-in period). C and D). Quantification of weekly training load during the lead-in, intervention and post periods, in (C) total weekly load (collected via session rating of perceived exertion method following each session and summated to give a weekly value) and D) normalised weekly load (to weekly load completed during the lead-in period. Histograms are group mean ± SD. Lead-in is three weeks of sea-level training prior to the intervention, intervention is three weeks of living and training at either 580 m (CON), 1600 m (ALT1600) or 1800 m (ALT1800), and post is the four weeks of sea-level training following the intervention period.

There were large increases in weekly TL from lead-in to intervention within ALT1600 (1723 ± 693 to 2453 ± 818, *g* = 0.91 [0.05,1.77], % change = 48 ± 32) and ALT1800 (1414 ± 387 to 2161 ± 370, *g* = 1.88 [0.95,2.81], % change = 60 ± 31) compared to CON (1659 ± 649 to 1936 ± 756, *g* = 0.37 [-0.52,1.26], % change = 18 ± 20); *g* = 1.04 (0.13,1.95) and 1.56 (0.55,2.41) versus CON respectively (**Figure 6.4C and 6.4D**). All athletes in ALT1600 and ALT1800, and six of seven athletes in CON increased TL during the intervention relative to lead-in (range ALT1600; 2-92%, ALT1800; 24-101%, CON; -10-53%).

Changes in TSB are shown in **Figure 6.5**. Mean TSB following the first and second weeks of the intervention were higher than at the conclusion of the lead-in period in all groups (ALT1600, *g* = 1.9 and 1.2; ALT1800, *g* = 1.3 and 0.3; CON, *g* = 1.2 and 1.1). Mean TSB during the post-intervention period was 86 ± 6, 86 ± 7 and 88 ± 11 for ALT1600, ALT1800 and CON respectively, with trivial to small differences between groups (*g* = 0.00 to 0.22). Relative to weekly TL achieved during the intervention period, during the first four weeks of the competition phase immediately following, TL was reduced in all groups (ALT1600, -29 ± 12%; ALT1800, -34 ± 10%; CON, -21 ± 24%).

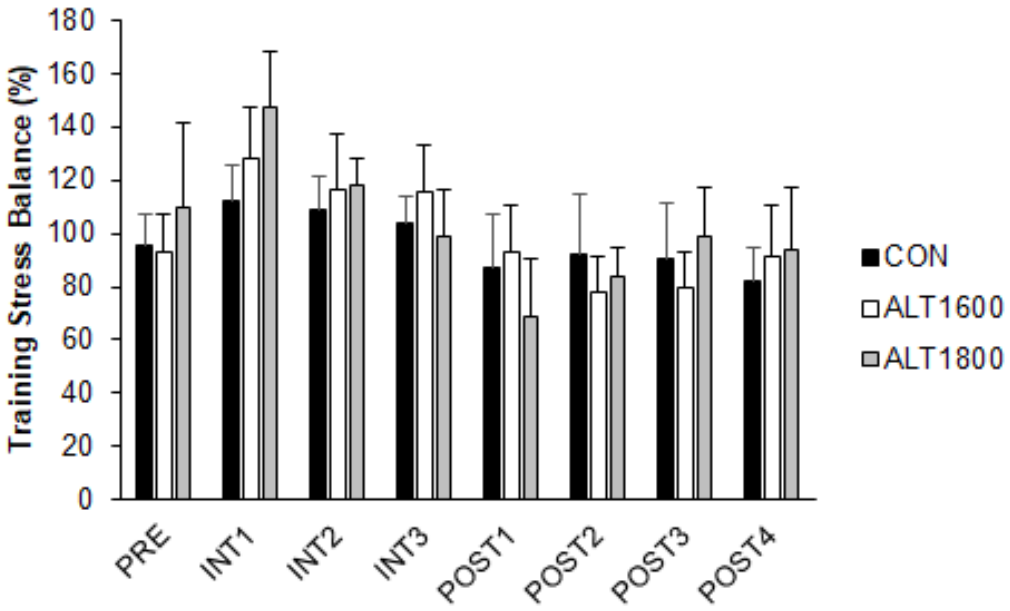

Figure 6.5. Changes in Training Stress Balance (TSB). TSB was calculated as the ratio of acute to chronic workloads (see methods) and is expressed as a percentage. Pre = TSB at the conclusion of the lead-in period, INT1-3 = TSB at the end of each week of the intervention period, and POST1-4 = TSB at the end of each of the first four weeks following the intervention period

During the lead-in phase of the study, participants in ALT1600, ALT1800 and CON completed 1.3 ± 0.4, 2.2 ± 0.3 and 1.7 ± 1.0 high-intensity interval-training sessions per week respectively, which increased to 2.8 ± 0.2, 2.9 ± 0.1 and 2.6 ± 0.4 per week during the intervention period. In weeks 1-4 following the intervention period, participants completed 1.8 ± 0.7, 1.9 ± 0.8, 1.0 ± 0.0 and 1.3 ± 0.4 (ALT1600); 1.9 ± 0.9, 2.0 ± 0.7, 2.0 ± 0.8 and 1.6 ± 0.5 (ALT1800); 1.5 ± 0.5, 2.2 ± 0.7, 2.5 ± 0.8 and 1.7 ± 0.7 (CON) of these sessions respectively. With respect to low intensity running, during the lead-in, intervention and post-intervention periods, participants completed 5.1 ± 1.3, 5.6 ± 1.3 and 4.7 ± 1.5 (ALT1600); 4.4 ± 2.0, 4.7 ± 2.4 and 4.5 ± 1.9 (ALT1800); 3.9 ± 1.3, 3.9 ± 0.7 and 4.0 ± 1.3 (CON) of these sessions each week respectively.

Haemoglobin mass

Individual changes in Hb_{mass} are shown in **Figure 6.6**. Increases in Hb_{mass} occurred within ALT1600 (790 ± 253 to 817 ± 256 grams, *g* = 0.10 [-0.72, 0.92], % change = 3.7 ± 3.8) and ALT1800 (921 ± 150 to 957 ± 153 grams, *g* = 0.23 [-0.55, 1.00], % change = 4.1 ± 4.4), but not in CON (935 ± 217 to 924 ± 216 grams, *g* = -0.05 [-0.93, 0.83], % change = -1.2 ± 3.3). Compared to CON, there were large increases in Hb_{mass} from baseline in ALT1600 (*g* = 1.29 [0.35, 2.22]) and ALT1800 (*g* = 1.26 [0.36, 2.17]). Seven of eight participants in ALT1600 and eight of nine in ALT1800 achieved increases in Hb_{mass} greater than the typical error of measurement for the CO rebreathing method (range ALT1600, 2.0-10.3%; ALT1800, 1.6-12.3%) compared to one of seven in CON.

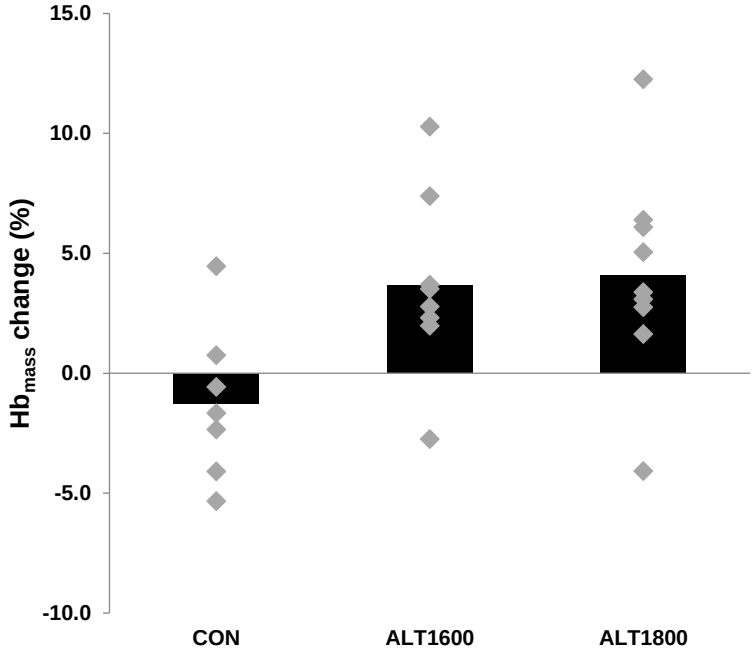

Figure 6.6. Individual and group changes in Hb_{mass}. Histograms indicate group means and diamonds denote individual responses.

6.5 DISCUSSION

We observed 1) greater improvements in race performance in the altitude training groups (~ 1.5%) compared to CON (0.4%), and 2) larger increases in Hb_{mass} in two altitude training groups (~ 4%) relative to CON (-1.3%). Despite similar volumes of training being completed by all three groups, greater TL relative to lead-in training was observed in the altitude training groups (48-60% increase in weekly load) versus control (18% increase), which suggests the relative importance of an altitude mediated increase in training load. Finally, season best performances were achieved over a wide time frame (5-71 days post intervention), suggesting the optimal window to race post-altitude training is very individual, and may not necessarily conform to prior recommendations regarding timing of competition post-altitude training (Baumann *et al*. 1994; Millet *et al*. 2010; Chapman *et al*. 2014b).

Performance

The combination of intensified training and three weeks of LHTH at 1600 m or 1800 m elicited a ~ 1.5% improvement in race performance, which was greater than performance changes achieved by athletes completing similar training at sea-level. Certain individuals responded particularly well, with performance improvements as great as 4% relative to their prior season's best, with only a single athlete out of 17 in the two altitude training groups not improving performance following LHTH. The degree of improvement observed here aligns with previous investigations examining the effects of LHTH sojourns at > 2000 m in elite athletes, with reported improvements in time-trial performance ranging from 1-4% (Gore *et al*. 1998; Bonne *et al*. 2014; Rodriguez *et al*. 2015). Data concerning changes in performance following training camps at < 2000 m is scarcer in the literature. Chapman and colleagues reported improvements of 0.8% during 3000 m time-trials (although not statistically significant) in runners residing at 1784 m and training between 1250-3000 m, with seven of ten athletes recording faster times following altitude exposure (Chapman *et al*. 2014a). Roels

and colleagues reported no change in 2000 m swimming time-trial performance after 13 days LHTH at 1850 m, with the camp occurring six weeks after a similar camp at 1200 m which induced a 2% performance improvement (Roels *et al*. 2006). Finally, Saunders and colleagues reported a 1.9% improvement in competitive race time in elite runners completing four, seven to ten-day blocks of natural LHTL (living at 1800 m, training between 1000-2200 m) interspersed with time at sea-level for competition; however, this was completed following 44 days of simulated LHTL sleeping at 2846 m and training at ~ 600 m (Saunders *et al*. 2009b). Compared to the previous literature, points of difference of our investigation include exclusively living and training at natural, low altitude, and utilising actual race performances to understand the effects of altitude on performance, with the repeat measures design mitigating the effects of tactics and variable motivation (Gore, 2014). The positive performance changes in line with previous research at higher altitudes provide encouraging evidence in support of athletes frequently utilising this low altitude training strategy to improve competitive performance.

Time course of performance changes

Our data suggest a wide window for optimal race performance following altitude training, with season's best performances being achieved 5 to 71 days after completing LHTH (**Figure 6.1B and 6.2**). Previous anecdotes from coaches and technical reports (Baumann *et al*. 1994; Millet *et al*. 2010; Chapman *et al*. 2014b) suggest competition either within three days of descent, or following three to four weeks at sea-level is optimal. In agreement with these recommendations, most peak performances observed in the current study were achieved following four or more weeks at sea-level, allowing for a lengthy re-acclimatisation process and potentially heightened training quality whilst physiological and haematological (including an elevated Hb_{mass}) adaptations conferred by altitude residence and training were at their peak. Additionally, major competitions in the season, towards which most athletes had been focusing their training occurred four or more weeks following altitude training. Performance was also

improved by ~ 1% in athletes' preferred events within the first four weeks following altitude training, with 12 out of 17 athletes (800 m – 3 of 4; 1500 m – 6 of 9; 3000/5000 m – 3 of 4) improving performance compared to baseline within this period, suggesting LHTH at low altitudes may be an effective strategy for improving performance over a range of middle-distance events. Three athletes achieved season best performances between days 3 and 14 post-altitude, and 60% of individuals competing within this time frame improved on baseline time, despite previous reports of sub-optimal performances in this period (Millet *et al*. 2010; Chapman *et al*. 2014b). We observed greater variability in performance immediately following altitude training, with performance changes ranging from 4% slower to 3.5% faster than baseline within the first two weeks of descent (**Figure 6.3**). These observations are reflective of the wide variation in response to altitude within and between individuals (Chapman *et al*. 1998), with the timing of a peak performance following altitude training likely to be a combination of altitude acclimatisation and de-acclimatisation responses, as well as periodisation of and responses to training conducted at altitude and immediately following (Chapman *et al*. 2014b). The rate of poor performances following altitude diminished with greater time at sea-level (**Figure 6.2B, 6.2C and 6.3**), suggesting there may be less risk in competing after a period of re-acclimatisation, however a peak response immediately following altitude exposure may be missed with this approach. Such considerations are likely influenced by an athlete's event distance and history with altitude training, amongst aforementioned factors.

Training

Strategic periods of intensified training may occur immediately prior to competition to promote adaptation and optimally prepare athletes (Aubry *et al*. 2014). In accordance with previous studies reporting performance improvements following altitude training (Levine and Stray-Gundersen, 1997; Bonne *et al*. 2014), there was a large increase in TL at altitude compared to sea-level reported in the current investigation (**Figure 6.4**). Contrary to previous

recommendations and practice (Baumann *et al*. 1994; Saunders *et al*. 2009a; Millet *et al*. 2010), the number of high-intensity interval sessions per week was increased at altitude. Most of the athletes in the investigation were experienced with altitude training, and thus were accustomed to completing intense training at altitude. Additionally, whilst the lower altitudes used in this study may not induce increases in Hb_{mass} as consistently in comparison to changes induced by moderate altitudes, a benefit of lower altitudes may be less of a need to make large modifications to training, compared to when training above 2000 m (Saunders *et al*. 2009a). Accordingly, the main alteration during the interval sessions in the current study were a slight lengthening of the recovery period to facilitate maintenance of training intensity, in line with recommendations (Saunders *et al*. 2009a; Millet *et al*. 2010). Whilst athletes in CON completed training of similar structure, intensity and volume to those in the altitude training groups, the addition of hypoxic stress allowed athletes training at altitude to achieve much higher training loads during the intervention period than the athletes in CON (despite these athletes also increasing load during this period). Increased training load and hypoxic exposure are inextricably linked, and with our observational design involving athletes completing individualised training, we recognise it is difficult to isolate which is the dominant of the two variables when explaining our performance results. Though we show that an intensified block of training completed at low altitude is more beneficial to performance than a similar block completed at sea-level, it is acknowledged that individual differences in training completed (e.g. number and timing of high intensity sessions) may have contributed to the differences observed between groups. Whilst having matched absolute TL between the three groups would help isolate the effects of training and altitude, in our estimation subjecting elite athletes to identical training programs would be sub-optimal for performance. The addition of hypoxic stress provides a mechanism to induce an increase in TL for a given running speed without increasing mechanical load, something that would generally be required to achieve an increase in TL at sea-level. Provided the necessary adjustments to training and recovery (e.g. modified training paces, appropriate selection of training altitude for different sessions, increased

recoveries) (Baumann *et al*. 1994; Saunders *et al*. 2009a) are made whilst at altitude to help facilitate a super-compensation response from a period of intensified training (Aubry *et al*. 2014), the added training stimulus may be a beneficial aspect of altitude training, in addition to a hypoxia induced increase in Hb_{mass}.

Studies modelling tapering strategies to optimise performance suggest greater training loads prior to a taper would allow higher performance gains (Thomas and Busso, 2005), a finding confirmed in subsequent experimental studies (Aubry *et al*. 2014; Hellard *et al*. 2017). Although all groups tapered training following the intervention phase (**Figure 6.4**) in accordance with guidelines (Bosquet *et al*. 2007), the greater TLs achieved prior to this in ALT1600 and ALT1800 compared to CON, as well as increased physiological capacities conferred by altitude residence (such as increased Hb_{mass}) and training may have put them at an advantage regarding performance enhancement during competition. Finally, training was individualised for each athlete based on preferred event, physiological characteristics, experiences from altitude training, and anecdotal results regarding the best training strategy for the current altitudes. In addition to reducing the volume of training completed each week during the post-intervention period, athletes in each group also completed fewer high-intensity interval sessions than during the intervention period, to prepare for competitions, with differences between groups being attributable to the varying racing schedules. Training individualisation was considered a critical aspect of training prescription which perhaps contributed towards the observed performance improvements, with previous investigations imposing uniform training structures upon athletes at altitude presenting negative performance outcomes (Adams *et al*. 1975; Bailey *et al*. 1998). Together, these results suggest the importance of individual training periodisation during and around altitude exposure with regards to maximising performance and reinforces the need for adequate monitoring of the training load and response of athletes.

Haemoglobin mass

Improvements in Hb_{mass} following altitude training reported in this investigation (~ 4%) are in line with previous findings in athletes completing low altitude training at 1800 m (Garvican-Lewis *et al*. 2015a). To our knowledge, we are the first to report an increase in Hb_{mass} in elite athletes completing three weeks of altitude training as low as 1600 m (**Figure 6.6**), equating to a hypoxic dose of ~ 800 kilometre hours, which is predicted to induce an improvement in Hb_{mass} similar to the magnitude observed here (Garvican-Lewis *et al*. 2016).

Limitations

Our investigation is not without certain limitations. Firstly, it must be noted that despite the similarities in overall training structure between the 3 groups, participants in the control group remained in their home environment throughout the investigation, thus a training-camp effect cannot be discounted when considering the improved performances after altitude training. Additionally, in contrast to participants in the two altitude groups, the low number of control group participants competing over an extended time frame (i.e. for more than three weeks post-intervention) makes comparison between groups difficult over this later period.

6.6 PRACTICAL RECOMMENDATIONS

- Competing immediately following an altitude camp can produce variable results, with very positive, but also negative performances being observed;
- Completing an intensified block of training prior to a competition phase is an effective strategy to improve race performance, with the greatest benefits occurring for those athletes completing this at altitude;
- A hypoxic dose of 800 to 900 kilometre hours is sufficient to increase haemoglobin mass by ~ 4% in healthy, iron supplemented, national level athletes undergoing an intensified training block at low altitudes.

6.7 CONCLUSIONS

Our results indicate that national and elite runners completing a pre-competition, three week block of living/training at 1600/1800 m had greater performance improvements in subsequent sea-level races than those athletes completing similar training at sea-level. These observations may be due to the greater overall load of training in hypoxia compared to normoxia, individualisation of training and competition schedules, as well as a hypoxia induced increase in red blood cells being relevant for longer duration events. The wide time frame for peak performances observed here suggests that this window is very individual, and factors other than altitude exposure *per se* may be important.

7.0 SUMMARY AND PRACTICAL RECOMMENDATIONS

7.1 SUMMARY OF KEY FINDINGS

The overall aim of this thesis was to optimise the training of elite runners during natural altitude training camps for performance improvement in subsequent sea-level competition. Studies One and Two sought to provide greater understanding regarding the physiological, performance and perceptual responses during interval training conducted at a variety of intensities relevant to middle-distance and distance running performance at low and moderate altitudes, to optimise the prescription (e.g. modifying work to rest ratios, altitude selection) of these sessions during altitude training camps. Studies Three and Four aimed to determine the effects of intensified training during LHTH completed at low and moderate altitudes on sea-level performance, by describing the training periodisation strategies (e.g. overload, taper, weekly training structure) employed by elite runners during these camps. A visual flow chart representing the thesis conception, progression and main findings is provided in **Figure 7.1**.

Given that elite runners, as well as athletes from other endurance sports frequently utilise LHTH as an ergogenic strategy, and inadequate management of training can result in a maladaptive or undesirable response, the optimisation of this practice is of great relevance to coaches and practitioners alike, with a well-executed altitude camp, or series of camps having the potential to provide athletes with a competitive advantage. Primarily, the data presented here suggest that during natural altitude camps involving elite runners with prior altitude experience, i) remaining at moderate altitude to complete some high-intensity training may be beneficial, as is ii) integrating established training practices such as overload (utilising hypoxic stress to facilitate the increase in load), taper and individualisation into a periodised and monitored training program.

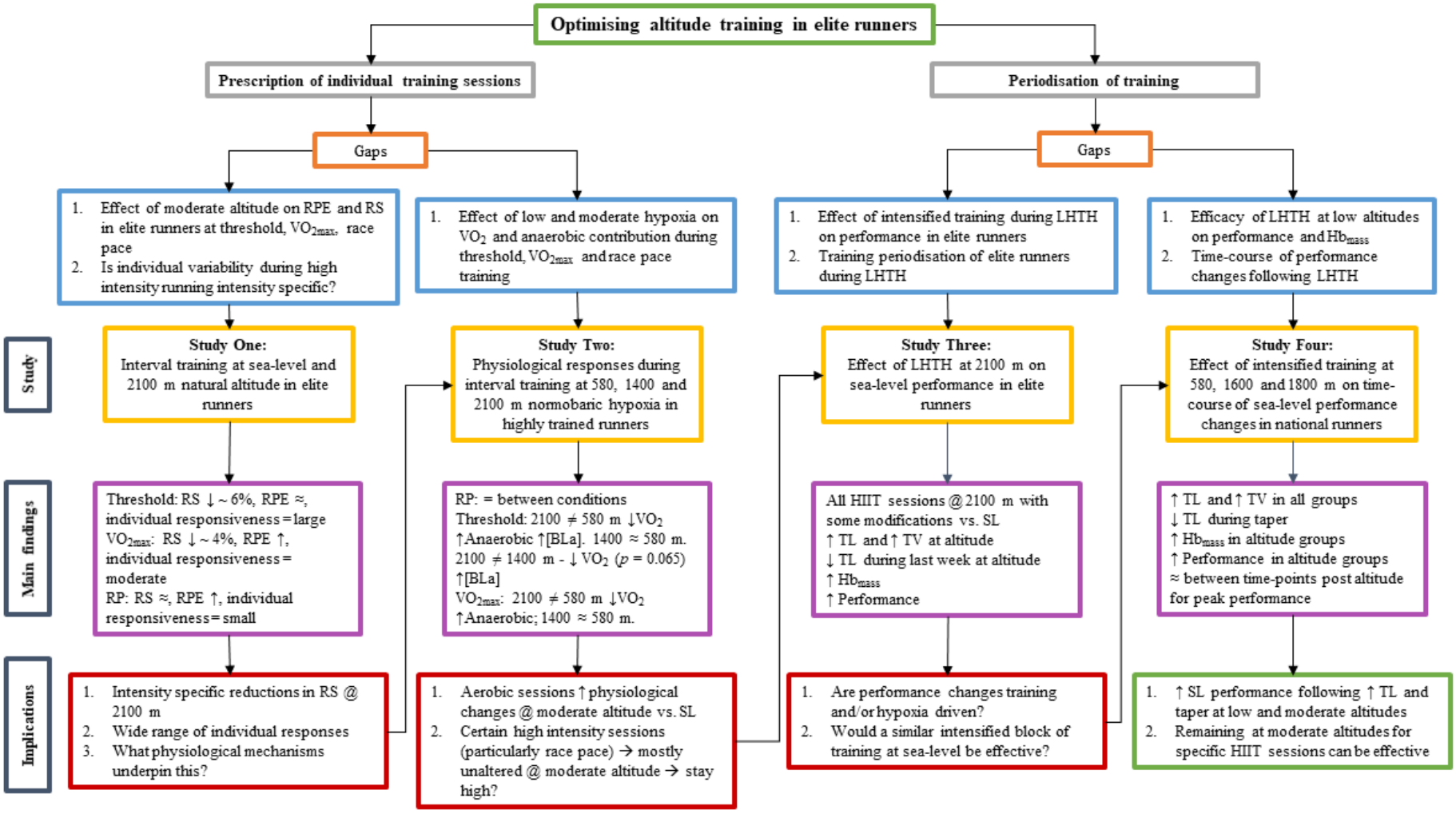

Figure 7.1. Flow chart denoting progression and key findings of thesis. RPE = rating of perceived exertion, RS = running speed, ↓ = decrease, ↑ increase, ≈ = unchanged, VO_2 = oxygen consumption, anaerobic = anaerobic contribution, [BLa] = blood lactate concentration, TL = training load, TV = training volume, HIIT = high intensity interval training, SL = sea-level, Hb_{mass} = haemoglobin mass, LHTH = live high train high

7.2 PRACTICAL RECOMMENDATIONS FOR ALTITUDE TRAINING IN ELITE RUNNERS

General recommendations for altitude training often pertain to issues including:

- Iron supplementation (Govus *et al*. 2015; Constantini *et al*. 2017; Garvican-Lewis *et al*. 2018);
- Optimising the dose of hypoxic exposure (Wilber *et al*. 2007);
- The number and duration of exposures per year (Saunders *et al*. 2009a; Millet *et al*. 2010; Solli *et al*. 2017);
- Completing high intensity sessions at a lower altitude (Levine and Stray-Gundersen, 1997);
- Timing of competition post-altitude (Chapman *et al*. 2014b).

These recommendations are geared towards facilitating the two main adaptive pathways of altitude training contributing to a performance response, namely the hypoxia induced acceleration of erythropoiesis, and the maintenance of oxygen flux and training intensity (Levine and Stray-Gundersen, 1997). Guidelines for optimising the prescription and periodisation of training during LHTH are less evident in the literature, despite its acknowledged importance in contributing to performance (Friedmann-Bette, 2008; Mujika, 2013; Chapman *et al*. 2014a; Brocherie *et al*. 2017). The investigations contained within this thesis aimed to help fill this void, and here we discuss a series of practical recommendations derived from the results presented herein, summarised as follows:

- Monitoring both the internal and external loads of training is crucial.
- Individualise training during acclimatisation to altitude based on training history/status, prior experience with altitude and duration of travel (overcoming jetlag).

- Experienced athletes may forego a lengthy period of low intensity training.
- Altitude training can be used as an overload stimulus to help induce performance super-compensation.
    - Athletes may prepare by reducing training load leading into LHTH, and shouldn't increase training volumes to previously foreign levels at altitude.
- Aim to maintain sea-level training intensity, particularly during race pace sessions, which may be achieved by:
    - Adequate periodisation of low effort and intensity training days within a weekly schedule.
    - Increasing work to rest ratios for high intensity training sessions.
    - Selecting training altitude appropriately to facilitate fast running and optimise the stimulus.
- Taper when concluding altitude training, regardless of whether competition is immediately upon descent from altitude, or after a period of re-acclimatisation to sea-level.

7.2.1 MONITORING LOAD AND TRAINING INTENSITY AT ALTITUDE

Concurrent use of simple internal and external monitoring tools can be effective in determining an individual response to an external stimulus such as hypoxia. While using an external measure of training such as running speed would be useful in determining which athletes were impaired to the greatest degree, and so allow for increased work/rest ratios to facilitate maintenance of training quality, the approach is limited in that it does not account for the level of effort/exertion (i.e. physiological stress) required to produce a given running speed. Accordingly, athletes and coaches should consider including a measure of internal load. In Study One, we expressed RPE as a function of running speed to produce an "exertion/velocity ratio". The ratio describes the relationship between exertion (in this case on a 10-point RPE scale) and running speed in $km \cdot h^{-1}$. An increase in this ratio is indicative of a greater level of exertion to maintain a certain running speed, while a decrease would infer the effort is easier. As expected, the ratio increased by up to 30% at altitude, suggesting that athletes perceive a much higher level of exertion for similar training sessions at altitude compared with sea-level. Implementing this relatively simple method of monitoring training could have application longitudinally; for instance, if there was an unexplained increase in the exertion/velocity ratio, training modifications may occur. Related synergistic methods of combining internal and external training quantification require further scientific validation.

7.2.2 INDIVIDUALISATION OF THE ACCLIMATISATION PHASE

Existing recommendations advocate for the reduction of training intensity during the initial 7-10 days of altitude exposure, generally characterised by a reduction in training volume relative to normal sea-level training, and an absence of anaerobic forms of training (Lange, 1986; Wilber, 2004; Millet *et al*. 2010). However, both anecdotal and scientific evidence suggests that athletes frequently undertaking altitude training may experience a faster acclimatisation response (Millet *et al*. 2010; Subudhi *et al*. 2014; D'Alessandro *et al*. 2016). As such, these athletes may be suited to foregoing a lengthy period of low intensity training upon arrival to altitude, a strategy which may be particularly relevant if the period at altitude is < two weeks, or LHTH occurs at low altitudes, as is frequently observed (Daniels and Oldridge, 1970; Saunders *et al*. 2009b; Solli *et al*. 2017; Schmitt *et al*. 2018). In Study Three, we observed high intensity training being completed as soon as Day 2 at altitude in athletes with extensive prior experience, with all athletes completing some threshold or $\dot{V}O_{2max}$ sessions on Day 4 (shorter intervals prescribed compared to sea-level) and race pace training on Day 6. A similar approach was observed in Study Four with athletes completing three weeks at 1600 or 1800 m. As such, several considerations related to the prescription of high intensity training upon arrival to altitude include:

- Close monitoring of athlete wellness (including sleep quality, hydration status, fatigue, and health status) particularly during the initial week at altitude, to ensure physical readiness to perform;
- A more typical acclimatisation of a few days low intensity training for athletes with no prior experience at altitude; and reduced training load and training volume during the weeks leading into live high train high to ensure athletes commence altitude training in a fresh, fatigue-free state.

Example acclimatisation weeks at altitude in novice and experienced athletes as observed in Study Three are shown in **Table 7.1**.

Table 7.1. Example acclimatisation weeks of training for novice and experienced athletes at moderate altitude. Novice athletes have a longer initial period of low intensity runs to acclimatise, as well as greater modifications and more conservative pace guidelines during high intensity sessions compared to those athletes with prior altitude experience.

	Day 1 (arrival)	**2**	**3**	**4**	**5**	**6**	**7**
New to altitude	30 min easy run	2 x 30-40 min easy runs, AM and PM	2 x 30 min easy runs or 1 x 60-70 min easy run	6 x 2 min with 2 min recovery @ threshold effort 30 min easy run PM	Gym	3 x (4 x 400 m on 2 min cycle), descend from 10 km to 1500 m pace	Long run
Previous altitude experience	30 min easy run	30-40 min easy run AM 10 km - build to threshold effort run PM	2 x 30 min easy runs or 1 x 60-70 min easy run	6 x 2 min with 2 min recovery @ $\dot{V}O_{2max}$ pace + 4 race pace strides 30 min easy run PM	Gym AM 30 min easy run PM	3 x (6 x 200 m @ 1500 m pace, 800 @ 10 km pace)	Long run

7.2.3 UTILISING ALTITUDE TRAINING AS AN OVERLOAD STIMULUS

In Studies Three and Four, substantial increases in training load (up to ~ 80%) were observed from the outset of altitude training, suggesting the degree of hypoxia at both low and moderate altitudes was sufficient to induce an additional stress on top of an increased volume of training. Moreover, in Study Four, it was observed that the frequency of high intensity sessions increased at altitude, relative to the preceding period of sea-level training. An important consideration characteristic of both studies is the lower training volumes observed during the lead-in period, helping to ensure athletes commenced LHTH in a condition conducive to tolerating increased volume and intensity of training, and facilitating adaptation. As identified previously (Solli *et al*. 2017; Turner *et al*. 2018), the additional time available for rest and recovery afforded on training camps away from the demands imposed by daily life (e.g. employment, education) is a crucial factor. Anecdotally, this allows for napping more frequently than usual, likely assisting in the tolerance and adaptation to increased training loads. Importantly, whilst training loads were increased at altitude, athletes were simply returning to volumes they would typically complete during regular training. Due to the hypoxic environment already placing limitations on training, we would not advocate for athletes to reach training volumes which they are unaccustomed to for the first time whilst at altitude, regardless of their level of experience. Furthermore, in lesser trained athletes, or those without prior experience with LHTH, it would perhaps be ill-advised to immediately increase training intensity at altitude to the magnitude seen in Studies Three and Four, irrespective of whether they were optimally prepared. An example describing the progression of training volume in elite middle-distance and distance runners leading into and during altitude exposure from Study Three is shown in **Figure 7.2**.

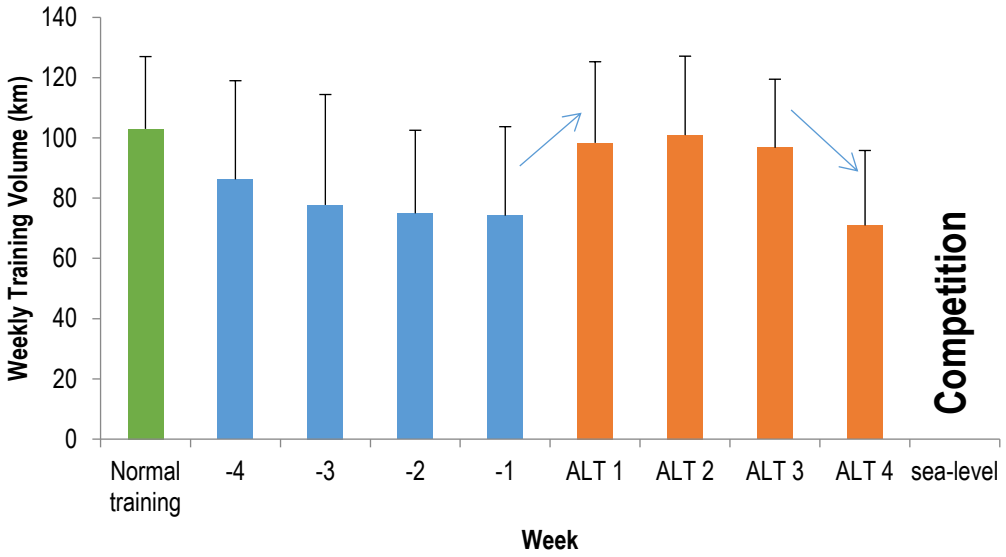

Figure 7.2. Return of training volumes to normal levels at altitude following a period of reduced volume at sea-level in elite runners. Blue histograms denote sea-level training weeks, and orange denotes weeks at altitude (ALT).

7.2.4 MAINTENANCE OF SEA-LEVEL TRAINING INTENSITY

Here, three strategies are provided which may be used to improve training quality at altitude; the weekly structure of training, modifications to work to rest ratios at altitude, and altitude selection for high intensity training sessions.

7.2.4.1 WITHIN WEEK PERIODISATION OF TRAINING

The distribution of effort (increased at all training intensities at altitude – Study One), as well as intensity across a training week is an important consideration for coaches when planning training. Training programs need to periodise intensity (measured externally and objectively) as well as exertion/effort (measured subjectively) in a way that facilitates an effective stress/recovery balance for the athlete. Specific high-intensity workouts can be separated by one or more long slow distance workouts, with the exercise intensity remaining below ventilatory threshold (Hydren and Cohen, 2015). These characteristics were exhibited within the weekly periodisation of training employed by athletes in Studies Three and Four (**Table 7.2**), whereby key sessions were prioritised with days containing low intensity and effort sessions preceding them.

Table 7.2. Weekly periodisation of training for middle-distance runners at altitude.

	MON	TUES	WED	THURS	FRI	SAT	SUN
AM	40 min - low effort	Threshold	50 – 70 min @ moderate effort	Race pace	Gym	Intervals or hills @ VO_2 max	80 – 120 min @ moderate to high effort
PM	Gym Optional 30 min - low effort	Optional 30 min - low effort	Rest	Optional 30 min - low effort	Rest (or short run)	Optional 30 min - low effort	Rest

7.2.4.2 MODIFICATION OF WORK TO REST RATIOS

The findings of this thesis reveal that perturbations to performance are greatest in training sessions where the duration and therefore aerobic contribution is highest (threshold and $\dot{V}O_{2max}$), likely due to the impairment of the aerobic system at altitude. Therefore, it would follow that these sessions may require the greatest modifications in their prescriptions to preserve sea-level training intensity.

With respect to threshold training sessions, in Study One, it was observed that athletes maintained their level of exertion at similar levels to sea-level, however running speed was reduced by ~ 6% as a result. Given that O_2 consumption was reduced at 2100 m even when running speed was maintained during similar threshold sessions (Study Two), such decreases in running speed would lead to even greater impairments in O_2 flux. Accounting for these considerations, to assist in preserving running speed, there is scope to modify from extended duration constant load or fartlek style (active recoveries) sessions often undertaken by elite runners, both at sea-level and altitude (Pugliese *et al*. 2014; Sharma *et al*. 2017), to interval-based threshold training with passive recoveries. For example, instead of a 20 min threshold run typically completed at sea-level, an athlete might instead complete 3 x 7 min with 2 min recovery at the same pace at altitude.

Our data suggest that the need to modify $\dot{V}O_{2max}$ training is greater than that of race pace sessions, given the changes in oxygen consumption and anaerobic contribution observed, as well as the larger impairment to performance at moderate altitude. Accordingly, recovery intervals should be lengthened for these sessions (e.g. whereas kilometre repeats are typically completed with 1 min recovery at sea-level, this is typically extended to 2 min at altitude). Anecdotally, during Studies Three and Four, it was observed the same number and length of intervals were completed at altitude compared to sea-level, with modifications occurring in the

form of increased recoveries, but not shorter intervals. There would appear little need to increase recovery intervals for short duration race pace efforts (e.g. 200 or 300 m intervals), however the ability to complete these at the required intensity at altitude will likely vary between individuals. It is suggested to increase recovery intervals for longer race pace intervals (i.e. speed endurance training sessions), where the aerobic contribution will be increased, and thus performance is subject to greater impairments.

Balancing the need to maintain training intensity at altitude to ensure optimal adaptation, whilst preventing athletes from overreaching is a key challenge when planning training during altitude sojourns. Therefore, the periodising into a program of certain training sessions whereby pace/intensity is regulated compared to sea-level may be an effective way of ensuring this equilibrium is achieved. In accordance with this, the eminent running coach Jack Daniels suggested that training paces for threshold and $\dot{V}O_{2max}$ be slowed during classic altitude training at 1800 to 2400 m, however in terms of maintaining normal sea-level intensity, shorter, faster repetitions at race pace should be prioritised (Baumann *et al*. 1994). A similar approach was observed in Studies Three and Four; race pace and some $\dot{V}O_{2max}$ intensity sessions were modified to include additional recoveries between intervals in order to maintain running speed, whereas threshold and low-intensity training were not modified (i.e. same interval/recovery length), but performed at a reduced speed and/or higher perceived effort compared to sea-level. The performance changes observed in both investigations indicates some merit in this approach, however further studies investigating manipulations to different intensities of training during LHTH are required to confirm these assertions. Additionally, the use of the exertion:velocity ratio to longitudinally monitor athletes at both sea-level and altitude may be useful to ensure the maintenance of training velocity isn't the result of over-exertion at altitude.

7.2.4.3 SELECTING TRAINING ALTITUDE BASED ON INTENSITY OF SESSION

We showed little difference between near-to sea-level and moderate altitude in performance, oxygen consumption, and anaerobic contribution for race pace sessions in particular, and observed athletes frequently undertaking threshold training sessions whilst remaining at moderate altitudes. Consequently, in contrast to traditional LHTL guidelines, we would propose completing race pace training, as well as certain other high intensity sessions at a moderate altitude, whilst descending to lower altitudes for particular sessions. For threshold and $\dot{V}O_{2max}$ sessions, descending to lower altitude would be beneficial in helping to defend O_2 flux. However, if the desired outcome was to increase the anaerobic contribution to exercise, which is relevant for middle-distance performance especially in the absence of an altitude induced increase in haemoglobin mass (Garvican *et al*. 2011), remaining at moderate altitude and increasing the length of recovery intervals to help maintain running speed would be suitable. Our recommendations, along with example training sessions and their modifications are summarised in **Figure 7.3.**

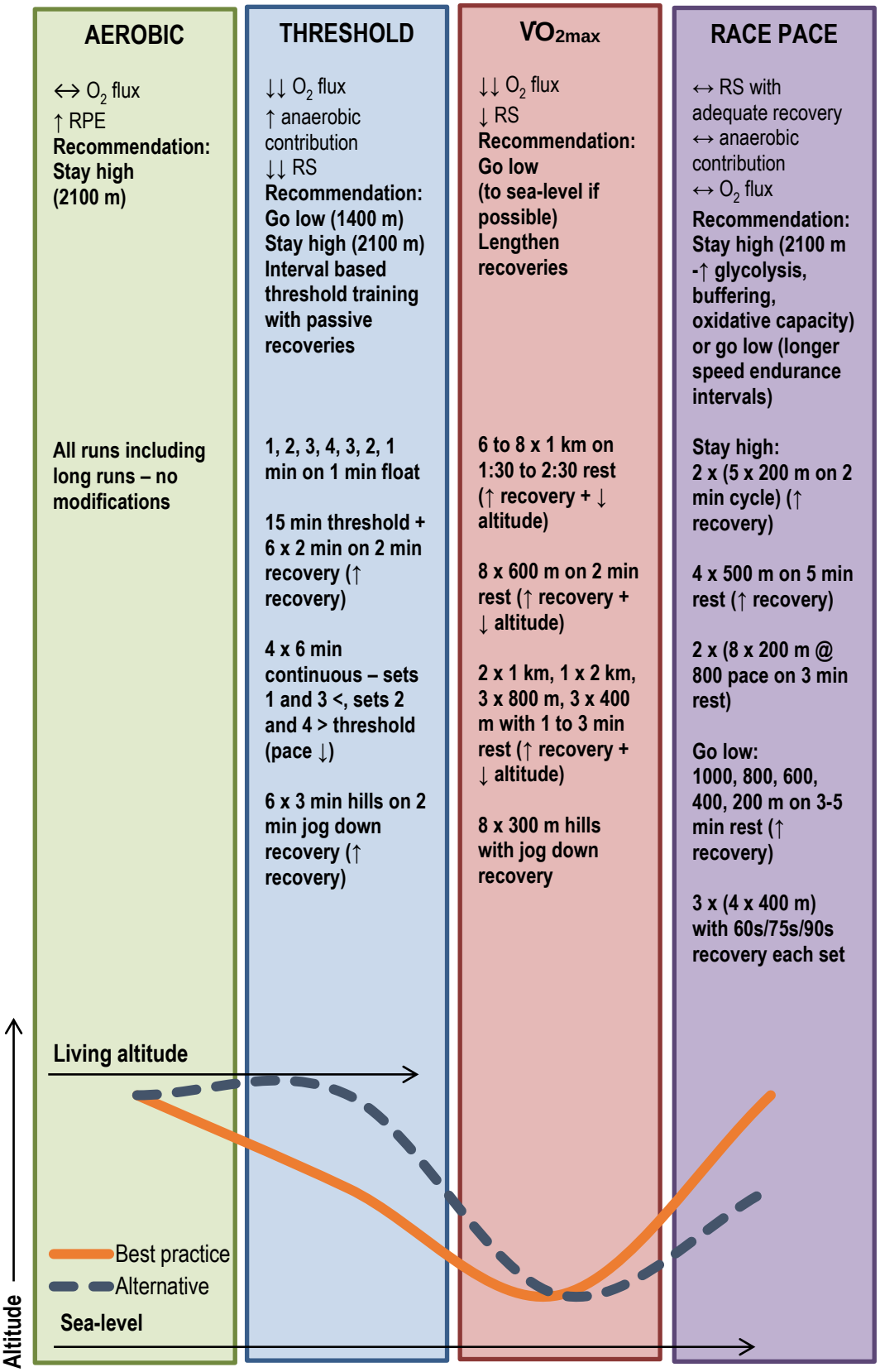

Figure 7.3. Altitude selection recommendations for different training intensities during residence at 2100 m. ↔ no change vs. sea-level, ↑ increase vs. sea-level, ↓ decrease vs. sea-level, RS = running speed

7.2.5 TAPERING

Tapering may have added relevance during altitude training, as the levels of accumulated fatigue resulting from residing and training under hypoxic stress are likely greater than equivalent periods at sea-level (Schmitt *et al*. 2018). In Studies Three and Four, reductions in training load of ~ 30 to 40% were observed in the final week of altitude training, with the largest decreases undertaken by athletes who completed the largest volumes of training. Given athletes would often compete within the first 3 days of return to sea-level, tapering commenced five to seven days prior to the altitude camp's conclusion, following a ~ two to three week period of intensified training. Additionally, the taper was predominantly achieved by a large reduction in the volume and frequency of low intensity training. Intensity and frequency of high intensity sessions was largely maintained, but with reduced volume in these sessions. The consistent performance improvements observed particularly in Study Three, as well as Study Four, suggest an aggressive reduction in training volume over a 7-10 day period similar to that observed here may be optimal. An extensive taper in training would likely be unnecessary for those athletes using LHTH differently (i.e. in early season general preparation, or choosing to race a few weeks after completion). However, it could be argued that a short sharp taper would freshen up the athlete for the subsequent period of hard training, effectively taking advantage of the beneficial adaptations conferred by altitude training. Adopting this strategy may help athletes avoid the anecdotally observed period of feeling "flat" after altitude training (Chapman *et al*. 2014a), and also help prepare athletes for onward (potentially long haul) travel to competition. Further investigations testing different manipulations of volume, frequency and intensity of training following altitude training are required to optimise the tapering process.

8.0 LIMITATIONS, CONCLUSION AND FUTURE DIRECTIONS

8.1 LIMITATIONS OF THE STUDY

Whilst consistent performance improvements of 1 to 2% were observed in Study Three, and replicated in Study Four at a lower altitude, the lack of control group (Study Three) and individualised training (Studies Three and Four) make it difficult to isolate the relative contributions of altitude acclimatisation and changes in training load to performance. However, it may be argued that by observing elite athletes undertaking altitude training under real world conditions (e.g. individual training and competition schedules, utilising competitions as performance measures), the investigations within this thesis maintain a much higher degree of ecological validity and thus practical application compared to studies undertaken under more controlled conditions. Indeed, the periodisation and individualisation of training has frequently been discussed as an important, yet often neglected aspect of altitude training studies perhaps affecting its implementation based on empirical evidence (Friedmann-Bette, 2008; Brocherie *et al*. 2017). Nevertheless, whether or not performance adaptations are driven primarily by training or altitude, acclimatisation is a crucial consideration and likely impacts on the prescription of training during altitude exposures. Though in Study Four we demonstrated that periods of intensified training are more efficacious when completed at altitude compared to sea-level, future investigations mirroring the cross-over design observed in the seminal investigation of Adams and colleagues (1975) (with athletes completing similar blocks of individualised and periodised training in line with current recommendations at the same absolute training intensity, at both altitude and sea-level) are required to answer this question with greater certainty.

When interpreting the practical recommendations arising from this thesis, in particular those pertaining to the modification of training sessions at altitude, we must acknowledge that these guidelines are derived from evidence collected under both hypobaric (natural altitude) and normobaric (simulated altitude) hypoxia. The differences between normobaric and hypobaric hypoxia has been the subject of discussion in the literature (Millet *et al*. 2012; Mounier *et al*. 2012; Beidleman *et al*. 2014; Coppel *et al*. 2015; Saugy *et al*. 2016), leaving it unclear as to whether different physiological responses are induced during exercise under these stimuli. Ideally, Study Two would have occurred at natural altitude to directly complement Studies One, Three and Four, however due to topographical and logistical limitations, the study could not be completed at the relevant altitude (2100 m) in Australia, and we could not obtain the necessary level of control on training sessions during Studies One and Three to measure interval training sessions under controlled laboratory conditions in Flagstaff, as it would create a great imposition on the training of elite athletes preparing for competition. As such, future studies investigating physiological responses during interval training at natural altitude are required to validate our recommendations for altitude training practice herein.

8.2 CONCLUSION

The primary theme of this thesis was a focus on training during altitude exposure, with the aim of optimising altitude training for performance improvement during subsequent sea-level competition. The achievement of the objectives in this thesis has provided further understanding of physiological and performance responses during training at altitude in elite runners, as well as training periodisation strategies employed by elite runners at altitude to improve performance in subsequent competition at sea-level. The key findings of this research were that:

i) Compared to sea-level, running speed in elite runners is adversely affected at 2100 m in an intensity-dependent manner (Study One);

ii) Completing high-intensity interval running at 2100 m simulated altitude, but not 1400 m, is likely to induce a lower $\dot{V}O_2$ and greater anaerobic contribution to exercise during threshold and maximal aerobic sessions when compared to training at 580 m; however race-pace training is largely unaffected (Study Two);

iii) Elite runners achieved personal best performances in sea-level competition immediately following LHTH at 2100 m (Study Three);

iv) A pre-competition, three week block of LHTH at 1600 or 1800 m yielded greater performance improvements in subsequent sea-level races than undertaking similar training at sea-level (Study Four).

Taken together, the greatest degree of individual variation in the decline in performance and $\dot{V}O_2$ is observed during high intensity sessions where the aerobic contribution is largest (threshold and $\dot{V}O_{2max}$), suggesting these sessions require individual adjustment in prescription at altitude, and the greatest scrutiny with regards to monitoring (Studies One and Two). The positive performance outcomes noted following altitude training may be due to the greater overall load of training in hypoxia compared to normoxia, effective tapering strategies,

individualisation of training and competition schedules, as well as a hypoxia induced increase in haemoglobin mass (Studies Three and Four). Moreover, the wide time frame for peak performances observed following LHTH suggests that the window for optimal performance is highly individual, and factors other than altitude exposure *per se* may be important (Study Four). During natural altitude camps, remaining at moderate altitude to complete some high-intensity training may be beneficial, as is integrating established training practices such as overload and taper into a periodised and monitored training program.

In summary, the findings of this thesis may be used to optimise the altitude training process at both low and moderate altitudes, with beneficial implications for elite athletes utilising this strategy during their competition preparation. The detailed characterisation of training load and implementation of ecologically valid practices in future studies is considered critical for future studies to further knowledge and optimise recommendations for altitude training.

8.3 RECOMMENDATIONS FOR FUTURE RESEARCH

A recent review providing a series of recommendations to improve the quality of exercise and sports science research suggested the lack of longitudinal and replication studies was an area of deficiency that required improvement (Halperin *et al*. 2018). Whilst the volume of research investigating the efficacy of various altitude training modalities from the last 50 years is extensive, uncertainty persists within the scientific community regarding the efficacy of altitude training, likely due to the lack of ecological validity with regards to training periodisation characteristic of many studies, and the failure to report training in others, making the interpretation of performance outcomes difficult. The findings of this thesis have increased our understanding of optimal training prescription and periodisation at altitude, used to provide a series of recommendations which may be implemented by coaches. However, further studies are required to confirm our findings, and as such the primary recommendation for future research would be further replication studies of altitude training in elite athletes from a variety of sports, under ecologically valid conditions, most importantly with theoretical sound training periodisation which is well characterised. Such studies would allow for valid interpretation of subsequent performance and physiological adaptation.

Several other opportunities for future research are evident. These include: studies systematically manipulating work-to-rest ratios during interval training at altitude in elite athletes, to determine the physiological and performance implications of this accepted practice; comparing the physiological responses induced by interval training during both externally paced and self-paced exercise in normobaric and hypobaric hypoxia; and investigating the effect of different tapering strategies at altitude on subsequent sea-level performance. Studies in these areas will help validate and refine the recommendations presented herein for implementation by practitioners, both in running as well as other endurance sports.

Taken together, the findings of the studies presented within this thesis suggest that in combination with prolonged (three to five weeks) residence at moderate altitudes, completing high intensity training sessions at both moderate and lower altitudes is optimal. Studies in team sports have established the superiority of this strategy above sea-level training, as well as LHTL for improving sea-level performance (Faiss *et al*. 2013a; Brocherie *et al*. 2015; van der Zwaard *et al*. 2018), however similar studies in elite endurance athletes are lacking.

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APPENDIX ONE: PUBLICATION ONE - QUANTIFICATION OF TRAINING AND COMPETITION LOADS IN ENDURANCE SPORTS

AUTHOR'S NOTE: parts of this book chapter are contained in the literature review of this thesis