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Neuromuscular and physiological characteristics of the early phase of running after cycling in elite triathletes

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Neuromuscular and physiological characteristics of the early phase of running after cycling in elite triathletes

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DECLARATION

I, Joel Walsh, hereby declare that this thesis, submitted in fulfilment of the requirements for the award of Masters of Research in the School of Science, Medicine & Health at the University of Wollongong, is wholly my own work unless otherwise referenced or acknowledged. This document has not been submitted for qualifications at any other academic institution.

Joel Walsh

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18 March 2015

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Sincerely,

Joel Walsh

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GLOSSARY

IR	Isolated run
C-R	Cycle-run
$\dot{V}O_2$	Oxygen uptake
HR	Heart rate
RPE	Rating(s) of perceived exertion
$t_{1/2}$	Halftime
MRT	Mean response time
bpm	Beats per minute
k	rate constant
r	Correlation coefficient
Δ	Change in/to
ΔO_2	Change in oxygen
ΔHR	Change in heart rate
EMG	Electromyography
rpm	revolutions per minute
ITU	International triathlon union

ABSTRACT

Background: In triathlon, the ability to run efficiently after cycling is considered paramount to an athlete's performance. However, it has been previously reported that when triathletes transition between cycling and running, termed the cycle-run transition, they are likely to experience noticeable levels of movement impairment and muscle activation disturbances. Previous research has specifically noted that prior cycling has a negative impact on neuromuscular control and increases the oxygen cost of subsequent running. Alternatively, research specifically observing the effects of prior exercise have reported a 'speeding' in oxygen uptake ($\dot{V}O_2$) during the early phase of subsequent exercise. Interestingly, there has been a limited analysis of the early phase response of muscle recruitment activity and metabolic variables during triathlon the cycle-run transition. However, research has suggested a potential link between cycling-influenced change to muscle recruitment patterns and an increase in the metabolic cost of subsequent running. There is also evidence to suggest that changes to muscle recruitment patterns do reflect changes to the metabolic cost during exercise, however when looking at triathlon, a potential link is not well established.

Objective: Therefore, the overall aim of this study was to investigate the muscular recruitment patterns and physiological response that occurs following prior exercise, in the form of cycling, on the subsequent early phase of running compared to isolated running among a cohort of trained triathletes.

Design: Fifteen (n=15) elite level triathletes (25.3±6.9 years) were successfully recruited for the study. Following a prior familiarisation session, all athletes were required to complete a highly repeatable, non-fatiguing exercise protocol comprised of a 10 min isolated run (IR), 30 min of seated rest, then a 20 min varied cadence cycle, followed by a 30 min transition run (C-R). All running exercises were completed on an indoor laboratory treadmill at an individually self-selected speed. Cycling was carried out using a stationary trainer with athletes mounting their personal bikes to the apparatus. During the IR and C-R breath-by-breath $\dot{V}O_2$ was recorded using a metabolic gas analysis and beat-by-beat heart rate was collected using a heart rate monitor strap. Muscle recruitment activity was measured at eight different muscle sites on the left leg using electromyography.

Results: Individual and group mean steady state $\dot{V}O_2$ were not different (p=0.44) between the IR (34.6±4.9 $ml.kg^{-1}.min^{-1}$) and C-R (35.2±5.1 $ml.kg^{-1}.min^{-1}$) conditions. Steady state HR values for the IR (139±12 bpm) and C-R (145±15 bpm) conditions were also not different. Respective halftime and mean response time values for $\dot{V}O_2$ were significantly different between the IR and C-R conditions (p<0.05). However, they were not for the early phase HR values. Rate constant (k) for $\dot{V}O_2$ were different between conditions (p<0.05), with a mean k of 0.90±0.1 for the C-R compared to the IR, 0.5±0.0. Mean muscle recruitment patterns for the IR and C-R demonstrated high levels of correlation (r=0.52-0.98; p<0.05). Similarly, no differences were recorded for peak muscle amplitude. However, coefficient of variation for muscle recruitment activity suggests relatively high variability recorded during the C-R (40.2-56.8%) condition compared the IR.

Conclusions: This study demonstrated that among elite level triathletes, prior cycling exercise does not adversely influence $\dot{V}O_2$ or heart rate at steady state during subsequent running, compared to isolated running. However, during the early phase response, prior cycling exercise does appear to influence the *k* and MRT, suggesting a potential 'speeding' effect on $\dot{V}O_2$ during the C-R condition compared to the IR. Our results also suggest that subsequent running muscle recruitment activity is not heavily affected by prior moderate intensity cycling, in spite of increased variability. Further, the absence of significant change to muscle recruitment activity, compared with $\dot{V}O_2$ and HR values during the early periods of running after cycling suggests that during moderate intensity exercise, there exists no meaningful link between neuromuscular and physiological variables until steady state is achieved. Additional research is however necessary to improve the understanding of the effects of prior cycling on subsequent running performance at higher exercise intensities, that are more likely reflective of the competitive demands of triathlon.

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1. BACKGROUND AND LITERATURE REVIEW

1.1 The Sport Of Triathlon

Triathlon is a multidiscipline endurance sport that involves swimming, cycling, and running in sequential order. The growth in popularity of triathlon has seen the sport diversify, particularly in terms of the distances raced by both age group and professional triathletes (Table 1.1). Professional categories exist in the majority of forms of triathlon; however the distance most commonly raced by professional triathletes is the Olympic distance. The current World Triathlon Series run by the International Triathlon Union (ITU) is raced over the Olympic distance that involves a 1.5 km swim, 40 km cycle and 10 km run. According to the ITU, the Olympic distance triathlon is the most commonly raced distance among amateur athletes on both a domestic and international scale.

The physiological demands experienced by single-sport swimmers, cyclists and runners are similar to those endured by Olympic distance triathletes. However, there are specific areas within triathlon, such as transitioning and running after cycling that may potentially alter the physiological stresses placed on athletes (Bentley, Cox, Green, & Laursen, 2008). Even within the field of triathlon, the physiological demands of competition and training will differ, so much so that preparation for an Olympic distance triathlon is vastly different from that required of an ironman triathlon. However, there are a few basic physiological factors that underlie the ability of an individual to participate in such a sport. The physiological ability to cope with the energy demands of competing in triathlon is considered to be a

principal factor in determining successful triathlon performance (Bentley et al., 2008; Suriano & Bishop, 2010).

Aerobic capacity has been documented as a determinant of successful endurance performance, not only in triathlon but also in single-sports such as cycling and running (Sleivert & Rowlands, 1996). Even for the fastest triathletes, the shortest triathlon will generally last for at least an hour, therefore the ability to utilise oxygen during energy transfer should be considered a significant performance indicator. Additionally, the economy of movement, which is the relationship between energy input and the resulting mechanical output, has been shown to account for large variations in endurance performance (O'Toole & Douglas, 1995; Sleivert & Rowlands, 1996).

For triathletes and endurance athletes in general, the ability to conserve energy through movement efficiency, whilst maintaining optimal mechanical work is critical (Bentley, Millet, Vleck, & McNaughton, 2002). Therefore, training to improve movement economy to reduce metabolic cost may directly translate into improved performance, especially during a triathlon.

Table 1.1: Triathlon event distances.

Event	**Distance**	**Swim***	**Bike***	**Run***
Long	Ironman	3.8	180	42.2
	Long/Half Ironman	1.90	90	21.1
Short	Olympic	1.5	40	10
	Sprint	0.75	20	5

*Denotes all distances in km. All distances are approximates and based upon those outlined by Triathlon Australia (triathlon.org.au).

1.2 The Cycle-To-Run Transition: Physiological, Neuromuscular And Biomechanical Factors Of Running After Cycling

The stage at which a triathlete moves from cycling to running is commonly termed the cycle-run transition. It is defined as the period from the last kilometre of the cycle leg to the end of the first kilometre of the run (Millet & Vleck, 2000). Running after cycling (C-R) is a specific demand of triathlon and for some triathletes, particularly those just starting triathlon, transitioning from cycling to running may lead to a feeling of awkwardness or distorted coordination. Past research has shown that the running performance of triathletes during the cycle-run transition is impacted upon by a prior bout of cycling, to varying extents (Hausswirth, Bigard, & Guezennec, 1997; Hue, Le Gallais, Chollet, Boussana, & Prefaut, 1998; Millet & Bentley, 2004). A number of studies have attempted to determine the underlying causes that affect running performance during triathlon, with the majority suggesting that changes tend to be either physiological, neuromuscular or biomechanically based (Bonacci, Green, et al., 2010; Bonacci, Saunders, Alexander, Blanch, & Vicenzino, 2011; Chapman, Vicenzino, Blanch, Dowlan, & Hodges, 2008; Hausswirth, Le Meur, Bieuzen, Brisswalter, & Bernard, 2010).

In terms of physiological impacts, research confirms an increase in the cost of running, reflected by an increase in oxygen uptake ($\dot{V}O_2$), during C-R compared to that of control running (Millet & Vleck, 2000). These authors suggested that an increase in $\dot{V}O_2$ relative to physical output was a potential limitation to running performance during triathlon. Several authors have suggested that the $\dot{V}O_2$ during running is affected by preceding exercise, in particular cycling (Guezennec, Vallier, Bigard, & Durey, 1996; Hausswirth et al., 1997; Hue et al., 1998). One such theory

suggests that the redistribution of blood flow around the trunk and upper body as a triathlete moves from a relatively static upper body position on a bike, to a more dynamic upper body posture during the run could potentially lead to higher levels of metabolic fatigue (Hausswirth et al., 2010). Further studies propose that any increase in $\dot{V}O_2$ when C-R is attributed to glycogen depletion, ventilator muscle fatigue, dehydration, and leg muscle fatigue (Guezennec et al., 1996; Hausswirth, Bigard, Berthelot, Thomaidis, & Guezennec, 1996; Hausswirth et al., 1997; Hue et al., 1998; Millet, Millet, Hofmann, & Candau, 2000; Millet & Vleck, 2000). Continuing research revealed that increases in $\dot{V}O_2$, HR and breathing frequency correlated with an increase cost when C-R as compared with an isolated run (IR) of the same speed and of similar distance (Bentley et al., 2002).

A review of past research highlights the negative impact that cycling, and in particular cycling cadence, has on the physiological, neuromuscular and biomechanical changes during subsequent running (Bentley et al., 2008; Bentley et al., 2002). Specific research outlined evidence to support the notion that participants (cyclists and triathletes) adopt cadences that minimise physiological variables such as oxygen demand, muscular activity, joint motion and force applied to the crank for a given power (Vercruyssen & Brisswalter, 2010). Moreover, it was suggested that higher cadences could minimise lower limb stress and reduce neuromuscular fatigue (Vercruyssen & Brisswalter, 2010). Neuromuscular changes including variation in muscle recruitment patterns resulting from C-R, have not garnered as much attention as the physiological change when C-R (Bonacci, Chapman, Blanch, & Vicenzino, 2009). However, the results of a study comparing the leg muscle recruitment patterns between elite cyclists and elite triathletes using electromyography suggested that leg

muscle recruitment during cycling in elite triathletes is less efficient when compared to elite cyclists (Chapman, Vicenzino, Blanch, & Hodges, 2007). The authors concluded that the lower levels of leg muscle recruitment in elite triathletes are potentially due to an alteration in the neuromuscular response resulting from multisport training. With respect to triathlete populations, elite athletes do not appear to demonstrate the same difficulties as non-elite athletes when reproducing pre-cycling running patterns (Millet et al., 2000). Subsequent studies looked at variations in muscle recruitment when C-R in moderately trained triathletes (Bonacci, Blanch, Chapman, & Vicenzino, 2010). The results showed that a third of participants demonstrated an inability to replicate pre-cycling running patterns, with one participant presenting significant changes in muscle recruitment patterns (Bonacci, Blanch, et al., 2010). Furthermore, lesser trained triathletes appear more susceptible to losses of coordination during running after cycling that are attributed to an interference effect between cycling and running muscle recruitment patterns (Bonacci, Blanch, et al., 2010). A subsequent investigation into the effects of how neuromuscular control through maintenance of muscle recruitment activity and running economy is preserved in elite triathletes following low-intensity and high-intensity cycling bouts, established that in elite triathletes there was no significant change in neuromuscular control, muscle recruitment patterns or running economy when C-R (Bonacci et al., 2011). The authors suggested that the preservation of neuromuscular control and running economy, as well as muscle recruitment was the result of training experience. In contrast, some elite international triathletes, leg muscle activity during running is directly affected by prior cycling despite their training history (Chapman, Vicenzino, Blanch, Dowlan, et al., 2008). These authors differentiated between the influences of fatigue and altered running speed on leg

kinematics and muscle activation. It was shown that a portion (5/14 or 30%) of elite triathletes exhibited muscle activity in the post cycling run leg that closely resembled the muscle activity of the prior cycling leg, rather than that of the control pre-bike run. The altered muscle activity during the first 40% of the gait cycle was immediately evident when C-R and persisted throughout the entire run (Figure 1.1). However, as only the tibialis anterior muscle was studied, alterations occurring in one muscle are likely not indicative of potential changes in other muscles, regardless of the changes being a direct result of cycling affecting the neuromuscular motor commands for running.

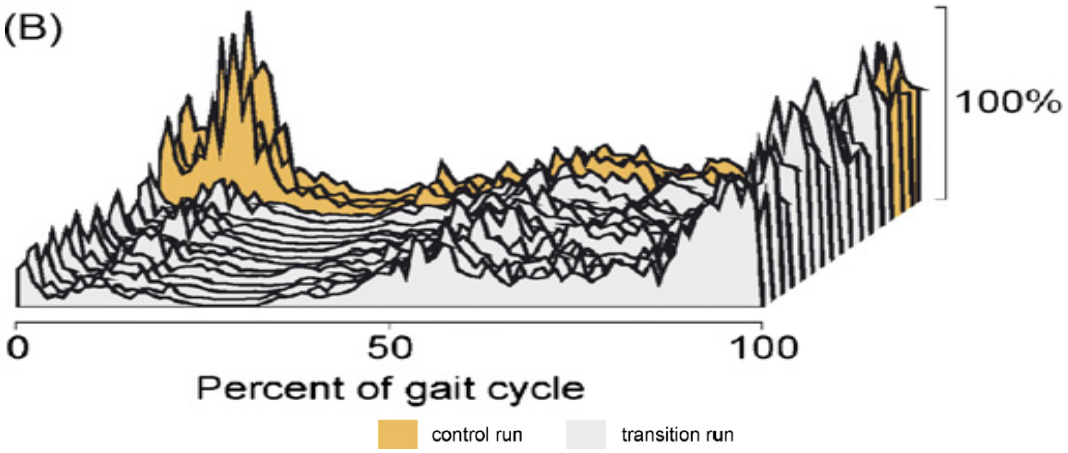

Figure 1.1: Is representative of the change in muscle fibre activation of the tibialis anterior muscle in an elite triathlete running after cycling. Notice at the start of the gait cycle the difference in toe-off muscle recruitment that is clearly absent in the transition run condition. The *y*-axis represents the peak amplitude of muscle recruitment activity. Image taken from Chapman et al. (2008).

Further research described that triathlon running is affected and potentially impaired by changes to the body's neuromuscular control patterns and leg muscle recruitment strategies (Bonacci, Blanch, et al., 2010). Moreover, alterations in ankle angle at foot contact during C-R explained a 67.1% variation in $\dot{V}O_2$ between a group of moderately trained triathletes (Bonacci, Green, et al., 2010).

Biomechanical research has also reported changes in kinematic variables when C-R, such as decrements to stride length (SL) and stride rate (SR), and also changes in trunk angle as a result of prior cycling (Hausswirth et al., 1996; Hausswirth et al., 1997). However, conflicting research has reported there to be no significant changes in running biomechanics, such as SL and SR when C-R (Hue et al., 1998; Millet, Millet, & Candau, 2001; Quigley, 1996). More recent research suggested that SL and SR are actually reduced during the C-R transition (Landers, 2011). These researchers cited that changes were likely the result of fatigue in the lower limbs and hip flexors, rather than simply due to the effects of prior cycling.

Indeed, research suggests that triathletes with more experience tend to display fewer mechanical and performance decrements than their less-trained counterparts (Millet et al., 2000; Millet & Vleck, 2000) and it is probable that previous experience through training contributes to an improved performance when C-R. However, researchers have pointed out that changes to the lower limb in triathletes during the C-R transition are inter-individual and each triathlete is likely to respond differently to the prescribed stimuli (Bonacci, Blanch, et al., 2010).

1.3 How Prior Exercise Affects Oxygen Uptake And Heart Rate Response At The Onset Of Subsequent Exercise

Oxygen uptake increases at a near-exponential rate in healthy young adults exercising at a moderate intensity (below the gas exchange threshold) until steady state is reached at approximately 2-3 min post exercise onset (Bailey, Vanhatalo, Wilkerson, Dimenna, & Jones, 2009; Jones, Berger, Wilkerson, & Roberts, 2006). However, previous research has noted that achieving steady state can be prolonged

due to the delayed-onset of the $\dot{V}O_2$ 'slow component', particular at moderate to high exercise intensities (Bailey et al., 2009; Ozyener, Rossiter, Ward, & Whipp, 2001; Whipp & Wasserman, 1972). Muscle oxygen supply has been cited as a primary influence on the slowing of $\dot{V}O_2$ at exercise onset (Krustrup, Gonzalez-Alonso, Quistorff, & Bangsbo, 2001; Sahlin, Sorensen, Gladden, Rossiter, & Pedersen, 2005). However, most prior exercise research has inferred that muscle oxygen supply does not limit $\dot{V}O_2$ kinetics during upright exercise among young, healthy individuals with high aerobic fitness (Jones et al., 2006; Jones & Poole, 2005): and effects on $\dot{V}O_2$ at exercise onset could occur at any point along the oxygen transport chain; from arterial oxygenation, haemoglobin concentration, cardiac output, muscle blood flow, muscle capillarity, mitochondrial density and muscle fibre type (Jones et al., 2006). Other variables that could impact $\dot{V}O_2$ limitations are the age and fitness of the participant studied, the intensity and body position during exercise and the muscle mass used during exercise (Jones & Poole, 2005). Furthermore, the withdrawal of parasympathetic activity has also been reported to influence the $\dot{V}O_2$ response after exercise onset (Hettinga, Monden, van Meeteren, & Daanen, 2014).

In an effort to combat the delayed onset of the $\dot{V}O_2$ 'slow component' and reach steady state earlier, 'warming-up' exercise has been used an as intervention method. The theory behind this concept was that prior exercise would 'warm-up' or 'prime' the cardiovascular system in preparation for exercise. Perhaps the most influential study showed that prior heavy exercise occasioned significantly faster $\dot{V}O_2$ kinetics during subsequent heavy exercise (Gerbino, Ward, & Whipp, 1996). The cause behind the faster kinetics was attributed to improved muscle oxygen delivery and greater muscle vasodilation (Gerbino et al., 1996). However, these authors also

reported that prior exercise of moderate or high intensity did not affect the $\dot{V}O_2$ response during subsequent bouts of moderate intensity exercise. Alternatively, prior heavy intensity exercise did influence the $\dot{V}O_2$ response during subsequent heavy intensity exercise.

Ensuing studies consistently reported an overall 'speeding' of $\dot{V}O_2$ response during heavy intensity exercise, following similar intensity prior exercise predominantly caused by a reduction in the amplitude of the $\dot{V}O_2$ 'slow component' (Bearden & Moffatt, 2001; Burnley, Doust, Ball, & Jones, 2002; Burnley, Doust, Carter, & Jones, 2001; Jones et al., 2006; Sahlin et al., 2005). 'Speeding' the $\dot{V}O_2$ response following prior exercise may well reduce the rate at which muscle fatigue develops via a reduction to the magnitude of the muscle oxygen deficit (Burnley & Jones, 2007; Jones, DiMenna, et al., 2008). Alternatively, studies that used moderate intensities for the prior and subsequent exercise bouts did not show any significant 'speeding' of the $\dot{V}O_2$ response (Gerbino et al., 1996; Macdonald, Pedersen, & Hughson, 1997). However, the $\dot{V}O_2$ response during a moderate bout of exercise subsequent to heavy intensity prior exercise has been related to improved microvascular oxygen delivery (Murias et al., 2011). Moreover, the 'speeding' of $\dot{V}O_2$ kinetics has been shown in older healthy participants completing moderate intensity exercise after prior heavy intensity exercise (Scheuermann, Bell, Paterson, Barstow, & Kowalchuk, 2002). However, these authors stated that among young healthy participants, no effect on $\dot{V}O_2$ kinetics was evident. Contrasting results have also been reported demonstrating that prior heavy intensity exercise, reduced the $\dot{V}O_2$ time constant of on-transient kinetics and improved local muscle oxygenation during subsequent moderate intensity exercise among young individuals (Gurd, Scheuermann, Paterson, & Kowalchuk, 2005).

The considerable amount of research into the effects of 'priming' or prior exercise has almost exclusively employed upright cycle ergometry exercise (Burnley et al., 2002; Burnley, Jones, Carter, & Doust, 2000; Gerbino et al., 1996; Gurd et al., 2005). However, $\dot{V}O_2$ kinetics during differing exercise, such as running, can have dissimilar effects on the $\dot{V}O_2$ response and the amplitude of the $\dot{V}O_2$ 'slow component' (Billat, Richard, Binsse, Koralsztein, & Haouzi, 1998; Carter et al., 2000b; Jones, DiMenna, et al., 2008). Furthermore, to our knowledge no study has specifically focused on the effects of prior or 'priming' exercise on the $\dot{V}O_2$ response during subsequent exercise using different exercise disciplines. As such, there is potential to suggest that the effects of prior exercise may vary if using differing exercise disciplines throughout experimental testing.

As is the case with $\dot{V}O_2$ response, the increase in HR after exercise onset stems from the withdrawal or diminishment of vagal activity of the parasympathetic nervous system, and concurrent activation of the sympathetic system (Hettinga et al., 2014; Jagoda, Myers, Kaminsky, & Whaley, 2014). The influence of sympathetic activation and parasympathetic withdrawal are influenced by increasing exercise intensity that drive the rise in heart rate (HR) however, a less rapid increase in cardiac frequency that prolongs the slow component response has been reported (Hettinga et al., 2014). Furthermore, a previous report suggests that the rate of cardiac frequency adjustment for a given exercise intensity is dependent upon an individual's fitness (Grucza, Nakazono, & Miyamoto, 1989). Moreover, endurance exercise reportedly improves the rate of HR increase when transitioning from rest to

exercise (Hagberg, Hickson, Ehsani, & Holloszy, 1980) yet, HR acceleration after exercise onset has presented wide individual variability (Jagoda et al., 2014).

The effects of prior or 'priming exercise on the increase in HR after exercise onset have often been reported as secondary measures when recording the $\dot{V}O_2$ response (Bailey et al., 2009; DiMenna, Wilkerson, Burnley, & Jones, 2008; Jones et al., 2006). These studies have reported that HR is significantly elevated at the onset of exercise following prior work with results suggesting that increased exercise intensity heightened the baseline HR differences. However, only one study demonstrated 'speeding' of HR kinetics, namely a faster mean response time, as a result of prior exercise (Bailey et al., 2009). In contrast, Scheuermann et al. (2002) reported that elevated baseline HR values result from an elevated cardiac output and muscle blood flow however, HR kinetics at moderate exercise intensities are not 'speeded' following prior exercise among young, healthy individuals unlike that of the $\dot{V}O_2$ response. Conversely, among older, healthy individuals HR kinetics after the onset of exercise following prior exercise are slowed. Furthermore, cardiac output and HR kinetics become dissociated during the 'speeding' of $\dot{V}O_2$ kinetics resulting from prior exercise (Bailey et al., 2009; Yoshida, Kamiya, & Hishimoto, 1995).

While the $\dot{V}O_2$ response is impacted, particularly among healthy, young individuals as a result of prior or 'priming' exercise, the evidence would suggest the HR response it not. Furthermore, prior exercise appears to impact the relationship between $\dot{V}O_2$ and HR, triggering dissociation as the increase of one variable, at the onset of subsequent exercise, becomes independent of the other. Interestingly,

evidence has proposed an association between the $\dot{V}O_2$ response and muscle fibre activation following prior 'priming' exercise (DiMenna et al., 2008; Endo et al., 2007; Krustrup, Soderlund, Mohr, & Bangsbo, 2004; Krustrup, Söderlund, Mohr, & Bangsbo, 2004).

1.4 The Effects Of Prior Exercise On Muscle Recruitment-Oxygen Uptake Relationship During Subsequent Exercise

The use of surface EMG is a popular technique used to assess muscle recruitment variation during human movement (Bonacci et al., 2009; Bonacci et al., 2011; Chapman et al., 2009). Previous use of surface EMG, to quantify 'muscle elasticity' demonstrated a potential relationship between EMG alterations and the changes in the metabolic cost of running (Bourdin, Belli, Arsac, Bosco, & Lacour, 1995). Further data purposed that increases in vastus lateralis (VL) EMG activity following four minutes of constant-load exercise correlates with the rise in $\dot{V}O_2$ during the same period (Shinohara & Moritani, 1992). Similar findings were presented when measuring muscle activation during low and high intensity cycling in a population of young, healthy individuals (Saunders et al., 2000). These authors concluded that gradual increases in the recruitment of fast-twitch muscle fibres were partly responsible for the concurrent slow rise in $\dot{V}O_2$ during constant-load exercise above the lactate threshold. However, no such alterations were detected during the lower intensity exercise protocols. Comparable results have shown that after heavy cycling exercise, the elevated primary $\dot{V}O_2$ amplitude was accompanied by a 19% increase in the average muscle activity for gluteus maximus, vastus lateralis and vastus medialis during the first two minutes of subsequent exercise (Burnley et al., 2002).

Similar findings have been reported on the relationship between metabolic cost and muscle coactivation during running (Abe, Muraki, Yanagawa, Fukuoka, & Niihata, 2007; Moore, Jones, & Dixon, 2014). Abe and colleagues showed an increase in vastus lateralis muscle contraction during prolonged running (90 min). An increase in the vastus lateralis eccentric/concentric ratio was associated with an increase in $\dot{V}O_2$ (mean increase of 15%) when recorded and compared at the 10^{th} min and 90^{th} minute of running. The authors cited previous studies, using highly trained athletes and similar exercise protocols, having only observed increases in $\dot{V}O_2$ of 5-10% during prolonged running. As a result it was acknowledged that using novice runners would likely present inconsistent results due to substantial difference in individual participants and could not rule out the effects of significant fatigue. Heise et al. (2008) reported lower $\dot{V}O_2$ was related to greater muscle coactivation between rectus femoris and gastrocnemius, among trained female runners, during the stance phase of running at self-selected speeds. In contrast, among recreational runners significant increases in coactivation of proximal and leg extensor muscles have reflected greater $\dot{V}O_2$ across running speeds between 9.1 and 12 km/h (Moore et al., 2014). Interestingly, these authors stated that as running speed increased muscle coactivation became shorter in duration. However, it was not reported whether the decrease in muscle activation reflected a similar reduction in $\dot{V}O_2$ at higher running speeds.

The overwhelming majority of changes to muscle recruitment and $\dot{V}O_2$ when exercising occur during high intensity exercise (above lactate threshold). However, previous studies have demonstrated that the scale and duration of muscle activity increases (Hortobágyi, Finch, Solnik, Rider, & DeVita, 2011) and the alteration to

muscle activation patterns are associated with the metabolic cost (Franz & Kram, 2012; Hortobágyi et al., 2011; Silder, Besier, & Delp, 2012) during low intensity exercise. These results suggest the possibility that changes to muscle recruitment activation under less intense exercise protocols may reflect simultaneous changes in $\dot{V}O_2$. However, the considered mechanisms behind the muscle activation/ $\dot{V}O_2$ relationship including increased ventilatory work, lactate production, epinephrine and increases in body temperature (Saunders et al., 2000) are only noticeable during high-intensity exercise. In contrast, research has also suggested that alterations in $\dot{V}O_2$, following prior exercise, do not correlate with changes to muscle activity during subsequent exercise (Barker, Jones, & Armstrong, 2010). Additionally, heavy intensity prior exercise has led to increased muscle recruitment during the early stages of subsequent exercise however; these changes were not reflected by increases in muscle energy cost (Layec et al., 2009).

Despite some differences in research findings, it would appear that altered muscle oxygen delivery, muscle fibre types and recruitment strategies do have an effect on $\dot{V}O_2$ during exercise (Barker, Trebilcock, Breese, Jones, & Armstrong, 2014; Barstow, Jones, Nguyen, & Casaburi, 1996; Jones et al., 2011), and have links to prior exercise and altered $\dot{V}O_2$ kinetics during subsequent exercise in adults (Burnley et al., 2002; Layec et al., 2009). Furthermore, alterations in muscle recruitment patterns have complimented the slowing in $\dot{V}O_2$ kinetics following varied pedalling rates during cycling (Breese, Armstrong, Barker, & Williams, 2011) and manipulated baseline metabolic rates (Breese, Barker, Armstrong, Jones, & Williams, 2012). Therefore, it is likely that muscle recruitment patterns of pedalling during varied-cadence cycle prior exercise will impact muscle recruitment patterns during

subsequent running. The anticipated alteration to muscle recruitment during the cycle-run transition may potentially provide an insight into the $\dot{V}O_2$ kinetics measured during this period.

1.5 Running After Cycling: An Indication Of Performance

Previous research has stated that success in triathlon is primarily dependent on a triathlete's ability to run after cycling (Chapman et al., 2009; Vleck, Burgi, & Bentley, 2006). Therefore, the ability to be able to 'run off the bike' is considered especially important for International Triathlon Union (ITU) Olympic distance triathletes, as race strategies for elite ITU triathletes often focus on the final run leg. Research has demonstrated that cycling prior to running does affect the physiological capabilities of triathletes and can increase $\dot{V}O_2$ during subsequent running (Guezennec et al., 1996; Hue et al., 1998; Landers, 2011; Millet & Bentley, 2004). In an attempt to reduce physiological effort and minimise accumulated fatigue and conserve energy ITU triathletes competing in draft-legal events will often ride as a group drafting off each other during the ride leg, in preparation for the upcoming running leg. Previous studies have compared the demands of the cycle leg on running performance in triathletes during both ITU draft-legal and non-draft legal races. For instance, run time to exhaustion was significantly greater following a variable intensity cycle bout, similar to that of ITU racing, compared with a constant intensity cycle bout (Suriano, Vercruyssen, Bishop, & Brisswalter, 2007). However, contrary evidence suggested that 5 km run performance was significantly better after riding for 20 km at a constant intensity than riding at varying intensities (Bernard, Vercruyssen, Mazur, Gorce, Hausswirth, & Brisswalter, 2007). Additionally, Lepers et al. (2008) commented that the degree of central (of the central nervous system)

and peripheral (of the musculoskeletal system) fatigue was alike after cycling at varied intensities and concluded that cycling for 30 min at either varying intensities or at a constant intensity did not induce any significant difference in neuromuscular fatigue. Furthermore, it was proposed that a significant decline in stride length when running after cycling was correlated with a decrease in running performance during a triathlon (Landers, 2011). The authors further stated that triathletes who maintained a similar pre-cycling run patterns or longer stride length during subsequent running after cycling ran faster and finished in a better position than those who failed to maintain stride length. Despite some conjecture, the literature supports the claim that the ability to efficiently link the cycle and run segments of a triathlon optimally results in improved running efficiency (Millet & Vleck, 2000).

1.6 Overview

The association between the effects of prior exercise on muscle recruitment and physiological variables appears to be strong. However, there appears minimal research observing the effects of exercise prior upon subsequent exercise compared with isolated exercise, and if or how muscle recruitment and physiological variables differ. In particular, and with respect to triathlon, there appears to be a lack of detailed research analysing the responsive effects of $\dot{V}O_2$ and HR between isolated running and cycling prior to running at a submaximal intensity. Furthermore, any link between the physiological response of the metabolic cost of exercise and muscle recruitment patterns following cycle-run and isolated running exercise appears to lack clarity. That previous authors have emphasised the importance of the cycle-run transition period due to large variabilities in running performances, in comparison to swimming (Bentley et al., 2008; Landers, 2011; Vleck et al., 2006) would advocate

the importance to optimally transition between cycling and running during competition (Bentley et al., 2002; Millet & Vleck, 2000). The importance of the cycle-run transition during triathlon should therefore warrant further analysis and in particular, correlating alterations in muscle recruitment activity with physiological variables during the initial stages of running following prior cycling exercise with isolated running. The results may assist in sufficiently detailing the physiological response that triathletes are likely to experience when transitioning from cycling to running. Furthermore, of the research that has sought to identify the physiological, neuromuscular and biomechanical responses during the cycle-run transition, timeframes for analysis appear to be based solely upon arbitrary time points, i.e. 1 min, 2 min and 3 min after exercise onset, rather than being based upon individually calculated variables.

Analysis of the relationship between $\dot{V}O_2$ and HR during various exercise intensities and under numerous prior and isolated exercising conditions is well documented. However, there appears to be minimal research that compares this relationship under isolated and running after cycling conditions, completed at submaximal intensities, which therefore justifies further investigation.

1.7 Aims And Hypotheses

The overall aim of this study was to investigate the muscular recruitment patterns and physiological response that occurs following prior exercise, in the form of cycling, on subsequent early phase of running compared with isolated running among a cohort of trained triathletes.

Chapter 3 describes a study that investigated potential alterations in muscle recruitment patterns and physiological variables during running after cycling in comparison to isolated running.

Based on the reviewed literature, it was hypothesised:

1. that in comparison to isolated running, muscle recruitment patterns during cycle-run exercise would demonstrate significant alterations as shown by increased variability and changes in mean EMG waveforms to muscle of the lower left limb.
2. that changes to muscle recruitment patterns would be greatest during the early phase of running after having cycled. Further, alterations to muscle recruitment patterns during subsequent running after cycling would be sustained throughout the entirety of the C-R condition.
3. that significant alterations to muscle recruitment patterns would reflect an increase in the metabolic cost of running as indicated by elevated levels of $\dot{V}O_2$ and HR at steady state during running following prior cycling exercise.
4. that during the early phase of running after cycling, muscle recruitment activity and $\dot{V}O_2$ and HR variables would demonstrate a greater level of variability compared to the same period during isolated running, and
5. that alterations to muscle recruitment patterns will be sustained throughout the cycle-run exercise and reflect similarly elevated changes in $\dot{V}O_2$ and HR variables measured during the cycling to running condition.

Chapter 4 describes an analysis of the data intended to demonstrate that $\dot{V}O_2$ and HR variables evolve differently during the early-phase of running after cycling as compared to isolated running.

Based on the reviewed literature it was hypothesised that $\dot{V}O_2$ and HR:

1. would be significantly elevated at the start of the cycle-run condition compared to the isolated run condition.
2. that during the early phase of running after cycling $\dot{V}O_2$ and HR values will be significantly higher to those recorded at the same time during the isolated run.
3. would be significantly elevated at steady state during running after prior cycling exercise, reflecting an overall increase in the cost of running.
4. rates of $\dot{V}O_2$ and HR response would be faster during running after cycling compared to isolated running, and
5. that under submaximal exercise intensity $\dot{V}O_2$ and HR would demonstrate a near linear relationship during both running conditions, regardless of prior cycling exercise.

2. PROCEDURES, PROTOCOL AND METHODS

2.1 Procedures

2.1.1 Experimental Overview

Elite level triathletes between the ages of 18 and 28 who were currently in a stable training phase were recruited for this study. Contact with participants was made through their respective coaches via face-to-face communication or email. Participants who showed interest in participating in the research were required to first attend an initial familiarisation session during which they were provided with a detailed explanation of the testing procedure, the level of intensity of the exercising protocols and the practical applications of the experiment. Participants were also provided with a 'participant information package' that provided further detail in regards to the experimental research. Participants that agreed to participating in the experimental research where asked to fill out a consent form, a PAR-Q and a physical activity questionnaire. Furthermore, a testing date for each participant was scheduled during the following 1-2 weeks post familiarisation and participants were asked to refrain from completing any training on the day of testing.

2.1.2 Participant Selection

A total of fifteen (n=15) participants (25.3 ± 6.9 years) were successfully recruited for the study. Using the information provided in the physical activity questionnaire participants were matched as closely as possible for triathlon training history using weekly training distance, training session duration, number of training sessions per week per discipline, weekly training intensity and years of previous triathlon experience as previously described (Chapman, Vicenzino, Blanch, & Hodges, 2008).

The study population were defined as highly trained in accordance with an accepted definition of 'highly trained triathlete' based upon training factors previously outlined (Chapman, Vicenzino, Blanch, & Hodges, 2008; Chapman et al., 2009). Participants who did not satisfy the definition of 'highly trained triathlete' were excluded from participation. An exclusion criterion was based upon that employed by Chapman et al. (2009) being that participants with a previous history of musculoskeletal or neurological disorders affecting the spine or lower limb were also excluded from this study. Further, participants with prior high-level competitive history in other sports within the preceding three months were also excluded. Furthermore, PAR-Q questionnaires were screened for each individual prior to testing to ensure each participant was in good health and free from any cardiovascular issues.

2.1.3 Participant Exclusions And Withdrawal

Of the fifteen participants recruited for this study, eleven were finally accepted following pre-screening and initial analysis of physiological data. One participant was excluded due to significantly altering their training patterns in the weeks prior to testing. A second participant withdrew from the study on the day of testing of their own volition. A third participant was excluded from testing due to a lack of physical activity/training volume in the three weeks prior to testing. Lastly, upon data collection, one participant was excluded due to significant irregularities that appeared during the recording of their $\dot{V}O_2$ data. This resulted in physiological data analysis being carried out on eleven (n=11) participants, eight males and three females. Of the eleven accepted participants five were excluded from EMG data analysis due to significant irregularities appearing throughout their individual EMG traces (Table

3.2). These irregularities to the EMG data were potentially due to noise interference from monitors, transformers or the EMG amplifier itself. Furthermore, motion artefacts were detected when reconstructing EMG waveforms. The artefacts were likely due to poor skin-to-electrode contact points. While a certain amount of noise of artefact can be tolerated and smoothed, the level of signal-to-noise ratio was considered insufficient and highly likely to degrade trace analysis and significantly impact the experimental results. Therefore, EMG data analysis was conducted for six (5 males and 1 females; *n*=6) participants.

Table 2.1: Participant screening exclusions from the study.

Exclusions (n)	***Reason***
5	EMG trace irregularities.
1	Participant was excluded on the basis of having significantly increased training volume in the two weeks preceding testing.
1	Participant withdrew self from the study on day of testing.
1	Participant was excluded due to lack of physical activity/training volume in the weeks prior to testing.
1	Participant data excluded due to severe irregularities recorded during the collection $\dot{V}O_2$ data.

2.1.4 Anthropometric Measurements

All individual anthropometric measurements are detailed in tables in their respective chapters. A total of 16 anthropometric measures were taken from eight different sites, including height and body mass, as described by the International Standards for Anthropometric Assessment procedures outlined by the *International Society for the Advancement of Kinanthropometry* (ISAK). Two measures were taken from each

site. A third measure was taken where the second measure was not within 5% of the first measure for length and breadth measurements or within 1% of height and body mass. A mean value of the two-recorded measures was calculated and reported in respective anthropometric tables. However, if three measures were required, the median value is reported in data tables. All measurements were taken by the same ISAK level 4 qualified researcher.

Height was measured using a wall-mounted Harpenden 602VR Stadiometer (Holtain Limited, UK.) with a range of 60 cm to 210 cm and an accuracy of 0.1 cm. The stretch stature method was applied when collecting height measures. This method requires the participant to stand with their heels together and having the heels, buttocks and upper back touching the stadiometer. The head was placed in the Frankfort plane by lining the lower edge of the eye socket (Orbitale®) with the notch superior to the tragus of the ear (Tragion®). Participants were instructed to take and hold a deep breath while keeping their head stable. Height measurements are then taken prior to the participant exhaling.

Body mass was measured using a set of electronic Charder MS5711 medical scales (Charder Electronic CO., Taiwan.) to 0.01 kg. Scales were zeroed prior to all mass measures with participants being weighted with the required exercising apparel worn. Left and right leg length measures were taken using a Lufkin W606PM steel tape measure (Apex Tool Group, LLC., USA.). Leg length was between the anterior superior iliac spine, crossing the tibiofemoral (knee) joint to medial malleolus of the tibia whilst the participant was supine. Leg length measurement is a combination of the Iliospinale and tibiale mediale-sphyrion tibiale measures outlined in the ISAK International Standards for Anthropometric Assessment procedures.

Breadth measurements were taken at the knee, ankle, elbow and wrist joints using Lafayette large bone callipers (Lafayette Instrument CO., USA.). Knee measures were taken using the lateral and medial epicondyles of the femur. The ankle joint breadth was measured as the distance between the medial malleolus of the tibia and the lateral malleolus of the fibula. Elbow breadth was measured as the distance between the lateral and medial epicondyles of the humerus. The measured distance between the styloid process of the radius and ulna were recorded for the wrist joint breadth. Hand thickness was measured using Lafayette large bone callipers (Lafayette Instrument CO., USA.), and considered the distance between the dorsal and palmer surfaces of the hand, at the thickest point.

2.2 Protocol

2.2.1 Experimental Conditions

All experimental testing was carried out in the School of Medical and Health Sciences, Exercise Physiology Laboratory between the hours of 9:00am and 5:00pm. Trials were conducted under thermo-neutral conditions; air temperature, 22°C and 35% relative humidity. Participants were instructed to refrain from completing any training or vigorous exercise prior to and on the day of testing. Testing equipment was calibrated prior to the arrival of each participant. Three researchers (two males and one female) were always present during testing. Upon arrival at the laboratory for testing, participants were again instructed on the experimental process and exercise protocols after which verbal consent was obtained.

2.2.2 Exercise Equipment

During experimental testing participants were required to wear training clothing for both the run and cycling portion of the protocol (see Appendix). Running bouts were conducted on a Landice L7 treadmill (Landice, Randolph, USA), with velocity increased at increments of 1 km/h to the required speed for the individual. Participants used racing footwear when running on the treadmill, to as closely replicate what they would be wearing during competition. Participants utilised their personal racing bikes that were fixed to a magnetic cycle ergometer (Tacx Satori Trainer, Tacx, Netherlands). Clipless pedals were used, as were each individual's cycling shoes. Cadence was controlled using Garmin® 500 ANT+ bike computers compatible with the speed/cadence monitors on the participant's bicycles. Prior to testing all participants were required to remove all jewellery including rings, any piercings, bracelets and/or necklaces, as these items may interfere with the collection of metabolic and neuromuscular data collection.

2.2.3 Running Protocol And Self-Selected Running Speed

Running tests were conducted on a treadmill at a 0% grade, in agreement with past research of a similar nature (Bonacci, Blanch, et al., 2010; Bonacci et al., 2011; Chapman, Vicenzino, Blanch, & Hodges, 2008). Treadmill velocity was constant to facilitate the control of running speed and continuous measurement of EMG and $\dot{V}O_2$. Running velocity was reached within the first 20 s of commencing running and remained constant for the entirety of IR and C-R conditions. Prior to experimental testing, a standardised 5-min warm-up run was completed. During this 5-min period participants were asked to self-select a running velocity that would be manageable and non-fatiguing for 30-min of running. This self-selected speed was used as the

treadmill speed for both the IR and C-R for that individual. Following the warm-up exercise, participants were required to rest in a seated position to allow their HR to recover to baseline values before beginning testing. The resting period between warm-up and testing exercise was 10 ± 0.6 min. The exercise format required each triathlete to complete a 10-min IR at the self-selected speed described during the familiarisation. After 30-min recovery period, 20-min of cycling was completed before a 30-min C-R. A standardised 60 seconds (s) period was used to transition between cycling and running.

2.2.4 Cycling Protocol

As previously mentioned participants used their personal road bikes mounted on a stationary magnetic cycle ergometer during the cycling trials. Cycling cadences were selected as representative for this population after collecting data of cadence from trained triathletes during training and during competition (Chapman, Hodges, Briggs, Stapley, & Vicenzino, 2010). Cadences ranges also replicate those used in an already established protocol that has been shown not to induce undue fatigue (Chapman et al., 2009). During the first 5-min and final 3-min participants cycled at an individually preferred cadence. Four cadence blocks of 3-min duration – (1) individually preferred cadence, 55-60 rpm, 75-80 rpm and 95-100 rpm – were randomly ordered between the 6^{th} to 17^{th} minutes. When a change in cadence was required, participants were instructed to adjust gear ratios that would sustain the level of intensity required (i.e. a consistent RPE of 14). Further, all participants were instructed to remain in an upright cycling position (i.e. holding either the brake hoods or handle bars) as changes in cycling position may influence muscle recruitment and metabolic cost.

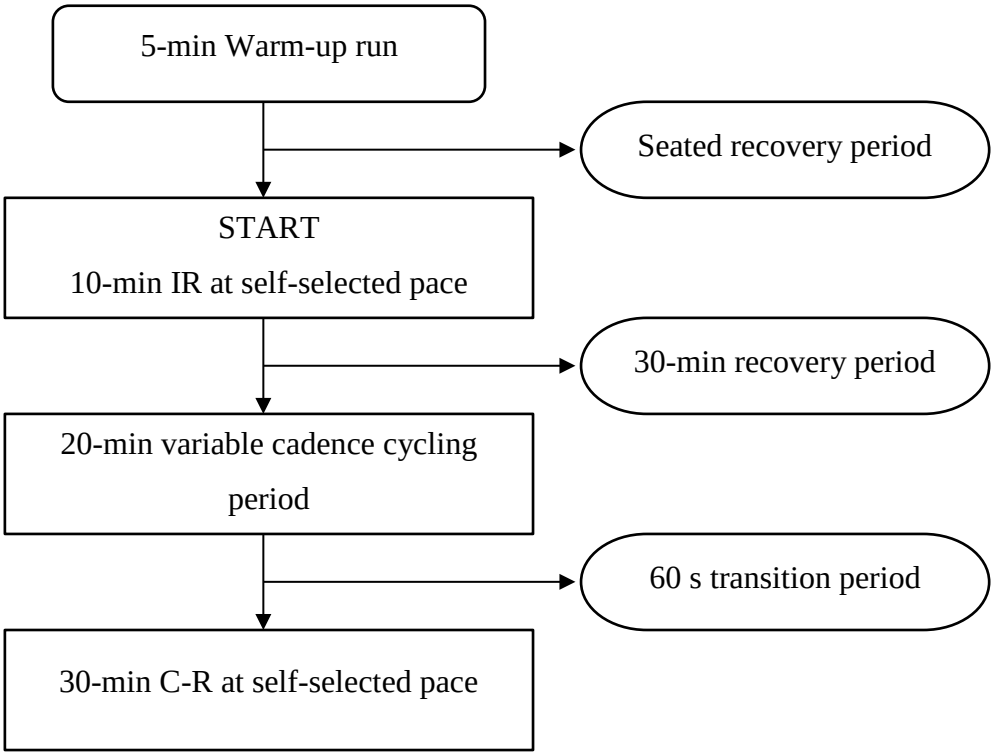

Figure 2.1: Flowchart of the experimental protocol.

2.2.5 A Submaximal Isolated Run Versus A Cycle-Run Protocol

A controlled single-group laboratory-based investigation was conducted to compare $\dot{V}O_2$ and HR values during an isolated and prior cycling bout of submaximal intensity running among a population of trained triathletes (Figure 2.1). This specific protocol has been previously developed to determine the direct effect of cycling on neuromuscular activity during proceeding running independent of undue fatigue (Chapman et al., 2009). Additionally, we were able to measure the metabolic ($\dot{V}O_2$ and HR) impact of cycling on running without it being significantly affected by preceding undue fatigue. Considering that fatigue has been documented to influence muscle activation, if undue fatigue was induced during the protocol any change in neuromuscular patterns during running would reflect fatigue rather than the impact of cycling on running. Therefore, this protocol was deemed appropriate as the basis of this study was to measure the effects of cycling-primed exercise on proceeding running, rather than the effects of a fatiguing bout of cycling on subsequent running.

2.2.6 Exercise Protocol: Repeatability And Stability

This protocol (Figure 2.1) was selected due to its (a) high level of repeatability when investigating the effects of cycle-primed exercise on muscle recruitment during running, (b) specific design when examining the influence of neuromuscular control during cycle-primed running independent of fatigue, and (c) isolated running bout that was short enough to prevent fatigue, therefore providing a control measure of neuromuscular activity during isolated running. Electromyography (EMG) measures reported when developing this protocol outlined the high level of repeatability for neuromuscular control during running after and before cycling. When developing the protocol, EMG measures at the 10^{th} and 30^{th} minute of the control run showed no

significant changes. This suggested that a 10-min control (isolated) run was sufficient to provide baseline measures with which to compare to 30-min of cycling-primed running. The absence of a rigorous warm-up procedure was the result of following the prescribed protocol. Regardless of the lack of warm-up the control run data has been reported as being highly reproducible and robust according to Chapman et al. (2009).

2.2.7 Regulation Of Exercise Intensity

Ratings of perceived exertion, collected at 1-min intervals, were the preferred method for regulating individual exercise intensity during the cycling protocol. Verbal feedback was employed to adjust exercise intensity until a rating of perceived exertion (RPE) of 14 was obtained. Further feedback was provided to ensure that participants held an RPE intensity of 14 throughout the cycling protocol.

The Borg 15 point (6-20) RPE scale (Borg, 1998) has been used previously as a valid and reliable way to self-regulate exercise intensity during cycle ergometry (Buckley, Eston, & Sim, 2000) and treadmill running (Eston, Lamb, Parfitt, & King, 2005). In terms of reproducibility, session RPE scores provided during aerobic exercise suggested a high correlation between average RPE values and session RPE values (Egan, Winchester, Foster, & McGuigan, 2006) Moreover, no significant variations in perceived exertion scores existed between male and female athletes, particularly when comparisons are made at relative levels of aerobic, metabolic or cardiorespiratory functioning (Faulkner & Eston, 2007). Research has also previously correlated the legitimacy of RPE scores with submaximal (50-70% $\dot{V}O_{2max}$) $\dot{V}O_2$ values (Dunbar et al., 1992) and HR (Kang et al., 1998). Cycling power

output also positively correlate with RPE scores (Dunbar et al., 1992) and provides a reliable measure of exercise intensity during cycling that is not significantly influenced by variations in power profiles between participants (Chapman et al., 2007). Furthermore, the simplicity of use and the absence of interrupting physiological monitoring and exercise performance were also considered an advantage of using a RPE scale to monitor exercise intensity.

2.2.8 Ethical Approval

The University of Wollongong Human Research Ethics Committee approved the application for the experiment research: HE 12/331. Under the provided ethics standards all participants completed an informed consent form as well as providing verbal consent prior to experimental testing.

2.3 General Methods

2.3.1 Electromyography Recording Of Muscle Recruitment

Neuromuscular activity was measured at eight different muscle sites on the left leg for all participants (Table 2.2; Figure 2.2). This research focused on muscles of the thigh and leg due to their functional importance to both cycling and running performance during triathlon.

Sodium/Sodium Cloride 1 mm parallel-bar surface EMG electrodes with an affixed inter-electrode contact area of 10 mm (Delsys®, USA) were used to record surface EMG. Electrodes were situated in a direction parallel to the muscle fibres of respective muscles and positioned anatomically according to procedures outlined by the European Surface Electromyography for the Non-Invasive Assessment of Muscles (SENIAM); Figure 2.2 presents a diagrammatic representation of electrode

placement sites. The skin surface of the leg was shaved and cleaned with alcohol around the belly of the muscle at the site of where the electrodes were to be fixed, in accordance with the procedures outlined in 'Standards for Reporting EMG Data (Electromyography and Kinesiology, 1997). Electrodes were attached to the skin sites using adhesive skin interfaces and along with the wires, were secured using Fixomull® (BSN Medical, Germany) stretch dressing to minimise movement during exercise. EMG electrodes were not moved or repositioned during the entire experimental procedure, including the recovery period, to maximise the EMG recordings reproducibility. However, outlines of each electrode were made on the skin of all participants to enable reproduction of placement if an electrode was to move or fall off. A ground reference Dermatrode® (American Imex, Irvine, USA) electrode was positioned on the left lateral malleolus.

Table 2.2: EMG electrode placement.

Muscle	Landmark 1	Landmark 2	Distance[a]	Angle[b]
Tibialis anterior (TA)	Lateral joint line of knee	Lateral malleolus of the tibia	30%	0
Gastrocnemius lateralis (GL)	Lateral joint line of knee	Lateral malleolus of the tibia	25%	15
Gastrocnemius medialis (GM)	Medial joint line of knee	Medial malleolus of the tibia	25%	15
Rectus femoris (RF)	Superior lateral patella	Anterior superior iliac spine	130 mm	0
Vastus lateralis (VL)	Superior lateral patella	Anterior superior iliac spine	120 mm	10
Vastus medialis (VM)	Superior medial patella	Anterior superior iliac spine	37 mm	55
Biceps femoris (BF)	Ischial tuberosity	Lateral popliteus	50%	15
Gluteus medius (GMed)	Line from crista ilica	Greater trochanter of the femur	50%	0

[a] Distance between landmark 1 and landmark 2, measured from landmark 1.
[b] Angle of inclination from the vertical to position the electrode parallel with the muscle fibre direction.

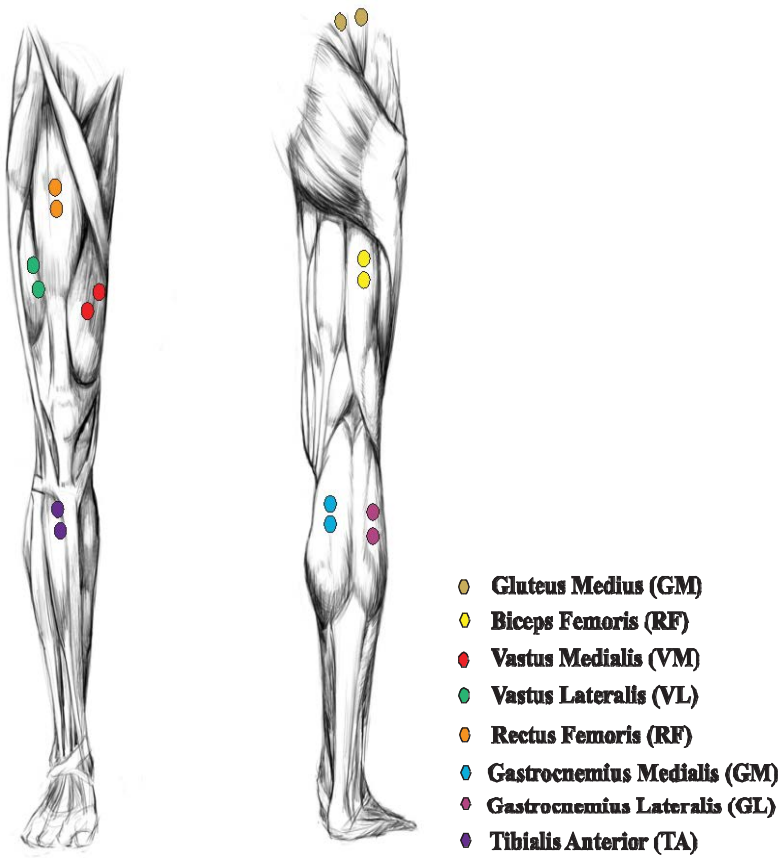

Figure 2.2: Diagrammatic representation of EMG electrode placement and muscle site placement on the left limb. See text for further details.

2.3.2 Measurement Of Stride Length And Stride Frequency

One stride was defined as being from foot contact to ipsilateral foot contact (Bonacci, Blanch, et al., 2010; Bonacci, Green, et al., 2010; Chapman et al., 2010). Strides were identified visually using the displacement and velocity of retroflective markers placed bi-laterally on the ankle, heel and second metatarsal head of each participant (Bonacci, Green, et al., 2010). The 3-dimensional positions of each marker were captured using an 8-camera VICON Bonita 2.2.1 3D motion capture analysis (Oxford Metrics Ltd, Oxford, UK) at a sampling frequency of 1000 Hz and filtered using a general cross-validation spline fit procedure to remove any low

frequency movement error. The cameras were positioned to ensure that each marker was captured by a minimum of three cameras at any one time. The system was calibrated prior to each testing session.

2.3.3 Measurement Of Oxygen Uptake

During all tests, pulmonary gas exchange and ventilation were measured continuously and recorded at breath-by-breath rate using a metabolic gas analysis cart (Parvo TrueMax 2400, Parvomedics, USA). The metabolic analyser was calibrated before each test with gases of known concentration (16% O_2, 4% CO_2 and Bal N_2) via a 3-liter syringe (Hans Rudolph, Kansas City, MO). Breath-by-breath inspiration and expiration were measured using a bi-directional, low dead-space (50mL) and low resistance volume turbine sensor. Prior to testing standing-resting $\dot{V}O_2$ was measured over a 5-min period to provide a comparative level of baseline $\dot{V}O_2$ values. Participants wore a nose clip and breathed through a low-dead space, minimal resistant mouthpiece that was secured via a capillary line attached to the mouthpiece, to the volume transducer. Starting $\dot{V}O_2$ was recorded as the first breath after the onset of exercise. Unlike previous oxygen kinetics studies, the first 15-20 s of data were not deleted as participants had had the oxygen mask fitted at least 60 s prior to exercise onset on both occasions.

2.3.4 Measurement Of Heart Rate

Beat-by-beat HR was measured using a short-distance telemetry Polar Interface module synchronised to a Polar heart rate monitor chest unit (Polar Electro, Port Washington, N.Y., USA) during both running conditions. HR was also collected at 15 s intervals during both running conditions. The Polar HR module was interfaced with the metabolic gas analysis cart.

2.4 Data Analysis

2.4.1 Electromyography Analysis Of Lower Limb Muscle Recruitment

Electromyography signal data was recorded using an eight channel Delsys® Bagnoli™ EMG System (Delsys®, USA). EMG measurements were recorded at a sampling frequency of 1,000 Hz and digitised by a 16-bit Analog-to-digital converter. The bipolar signal was amplified (input impedance > 1 MΩ) and band-pass filtered between 10 and 500 Hz with a mode rejection ratio of 110 dB, gain of 305 and maximum noise of 1.6 μV or a second order Butterworth filter was applied to the data to remove contamination from movement artefacts before being full-wave rectified, DC offset. Following, EMG data were integrated into specific time bins; control data recorded from the 9^{th}-10^{th} minute of the IR, variable data was collected at individually calculated times representative of $t_{1/2}$, MRT and 180 s (steady state) during the C-R condition. The rectified EMG data were exported in an embedded VICON c3d file. All data processing and analysis was performed off-line using a commercial software package (MATLAB 6.1, The MathWorks Inc., Natick, MA, 2000). Electromyography data were time standardised to 100 points for each stride during the IR and C-R exercise conditions. EMG amplitude was expressed as a percentage of the peak record EMG amplitude for the respective muscle, in accordance with previous procedures (Bonacci, Blanch, et al., 2010; Chapman et al., 2007). All EMG waveforms were visually screened and data of inadequate quality (i.e. traces containing high levels of artefact, that could not be adequately removed by signal filters) were excluded from analysis, as previously recommended (Bonacci, Blanch, et al., 2010). Analysed EMG data was reported using indices of: (i) the pattern of muscle recruitment and movement indicated by EMG waveforms; (ii)

mean EMG amplitude for the duration of each stride during respective time periods; (iii) peak EMG amplitude for each muscle; (iv) Root Mean Square Error of muscle activity; and (v) coefficient of variation.

2.4.2 Stride Length And Stride Frequency

Control data was taken as the average of 20 strides during the 9th-10^{th} minute mark of the IR condition. For the C-R exercise conditions, data was averaged for 20 strides and exported to time bins representative of individual $t_{1/2}$, mean response time (MRT) and 180 s time periods. Stride length (cm) was calculated as the anterior-posterior between ankle markers at the moment of heel strike (Hak, Houdijk, Beek, & van Dieën, 2013).

Stride frequency was calculated as strides per minute (stride/min) using a mean of 20 strides recorded from the 9^{th}-10^{th} minute during both IR and C-R conditions.

2.4.3 Oxygen Uptake

Breath-by-breath $\dot{V}O_2$ data from all tests were screened to exclude errant breaths caused by coughing, swallowing, sneezing, etc., any breaths that were four standard deviations from the mean were removed as outline previously by (Bailey et al., 2009). For each participant, breath-by-breath data was linearly interpolated from exercise onset to pulmonary steady state (~180 s). Starting or pre-exercise $\dot{V}O_2$ was recorded as the average $\dot{V}O_2$ during the 30 s prior to commencing exercise. Mean $\dot{V}O_2$ at the 10^{th} minute was calculated as the average between the 9^{th} and 10^{th} minute and steady state $\dot{V}O_2$ was accepted as the mean $\dot{V}O_2$ recorded during the final one minute (60 s) of running for both conditions, and were calculated for all participants during both IR and C-R conditions. The 10^{th} minute variable calculation was used to compare with the steady state values to ensure that $\dot{V}O_2$ did not drift under exercising

conditions. At submaximal levels of exercise, physiological steady state is generally achieved after 2.5 to 3 min in young health adults (Pringle et al., 2003; Xu & Rhodes, 1999). Therefore, this study considered the trained participants as having achieved steady state by the 3rd minute of constant velocity running, therefore, participant steady state was deemed to be reached by the 3rd minute (180 s) using a ±5% range prediction calculated from steady state $\dot{V}O_2$.

A single-phase, logarithm model was used to calculate halftimes ($t_{1/2}$), that represents the time-related change to stabilisation or equilibrium (Stupnicki et al., 2010). In this case, $t_{1/2}$ was considered the mid-point (in time – s) to theoretical $\dot{V}O_2$ steady state. Calculating $t_{1/2}$ for $\dot{V}O_2$ would assistance in portraying variations in the rate of change for $\dot{V}O_2$ from the onset of isolated and cycle primed running, to a level of physiological steady state. Net $\dot{V}O_2$ (x_i) was processed by subtracting baseline values from the screened $\dot{V}O_2$ values. Net values were then calculated at each 15 s interval for the full duration of the IR and C-R for each participant, before undergoing decimal logit transformation using the remodelled logarithm equation (equation 1) and correlated with log time. The logarithm was reconstructed and the maximal characteristic of the original equation (x_m) was substituted with a calculated submaximal value. Furthermore, log $t_{1/2}$ was defined as corresponding to logit = 0.

$$\text{Logit} = \log [(x_i) / (x_m - x_i)]$$

$$\text{Logit} = \log (x_i) - \log(x_m - x_i)$$

Equation 1

$\dot{V}O_2$ kinetics were assessed during the first three minutes of running exercise. The first three minutes of exercise were used to calculate oxygen kinetics as it has been

previously reported that steady state among healthy participants completing submaximal exercise is achieved within approximately three minutes of exercise onset (Barstow, Casaburi, & Wasserman, 1993; Whipp, 1987; Xu & Rhodes, 1999). Oxygen deficit was defined as [t * $\Delta\dot{V}O_2$ - $\sum\dot{V}O_2$] where t represents the time from exercise onset to steady state (i.e. 180 s), $\Delta\dot{V}O_2$ was calculated as the difference between starting $\dot{V}O_2$ and steady state $\dot{V}O_2$ (mean value of $\dot{V}O_2$ during the final one minute of running during respective conditions), and $\sum\dot{V}O_2$ was the cumulative oxygen consumption from starting $\dot{V}O_2$ to SS, considered the sum of measured exercise from onset to steady state (Figure 2.3) (Chatterjee et al., 2013). Mean response time (MRT; Figure 2.2) is considered the exponential time constant of $\dot{V}O_2$ on-kinetics (Whipp & Ward, 1990) and represents approximately the time required to achieve 63% of steady state $\dot{V}O_2$ (Chatterjee et al., 2013). MRT was calculated using the algebraic formula reported by Whipp, (1971) (equation 2). The equation was expanded and MRT defined as [oxygen deficit]/[$\Delta\dot{V}O_2$] (Lewalter et al., 1997).

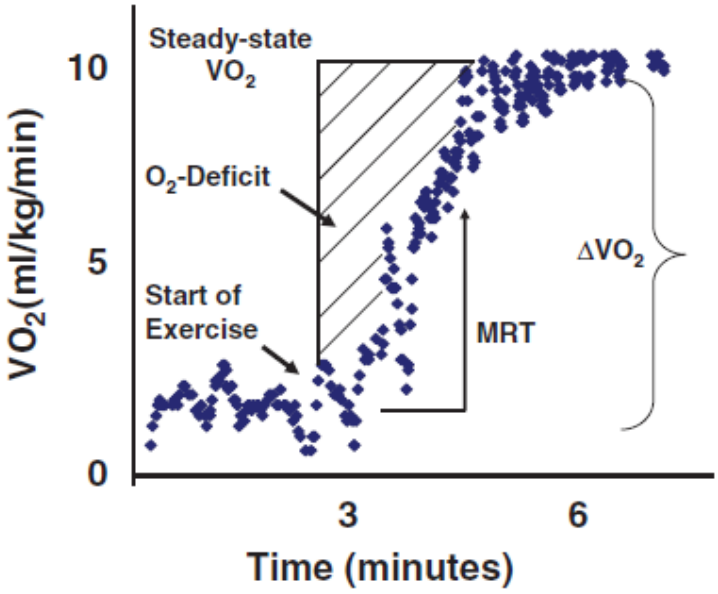

Figure 2.3: MRT is the time required to reach ~63% of steady state. MRT was calculated for all individual participants under both running conditions to provide a reference value for EMG analysis Figure adapted from Chatterjee et al. (2013).

$$\text{Oxygen deficit} = \Delta\dot{V}O_2/k$$

$$\text{Mean response time (MRT)} = \text{oxygen deficit}/\ \Delta\dot{V}O_2$$

$$\text{Expansion of Whipp formula} = [t * \Delta\dot{V}O_2 - \sum\dot{V}O_2] = \text{oxygen deficit}$$

$$\therefore \text{MRT} = [t * \Delta\dot{V}O_2 - \sum\dot{V}O_2]/\ \Delta\dot{V}O_2$$

Equation 2

Rate constant (*k*) values were also calculated for $\dot{V}O_2$ under both exercising conditions in accordance with the equation (equation 3) (Whipp, 1971). The rate constant, *k*, values were used to determine potential transient changes in $\dot{V}O_2$ and the relative effectiveness of the cardiopulmonary and metabolic systems response after the onset of the IR and C-R conditions.

$$k = \dot{V}O_{2\ (\text{steady state})}/\ \dot{V}O_2 \text{ deficit}$$

Equation 3

2.4.4 Heart Rate

Heart rate data were screened for any anomalies before the second-by-second data was linearly interpolated and, for each participant, time aligned at 15 s intervals from exercise onset to the end of exercise. Standing-resting HR was also measured over a 5-min period to provide a comparative level of baseline HR values. Starting or pre-exercise HR was recorded as the average HR during the 30 s prior to commencing exercise and steady state HR was accepted as the mean HR recorded during the final one minute (60 s) of running for both conditions (Nelson, Thomson, Rogers, Howe, & Buckley, 2013). Mean HR at the 10^{th} minute was calculated as the average between the 9^{th} and 10^{th} minute. The 10^{th} minute variable calculation was used to

compare with the steady state values to ensure that HR did not drift under exercising conditions.

Similar to $\dot{V}O_2$, recorded beat-by-beat HR data were used to calculate individual $t_{1/2}$ and MRT values. The same logarithm model proposed by Stupnicki et al., (2010) was used to calculate HR $t_{1/2}$ from exercise onset to steady state. Net HR (x_i) was calculated from screened HR data and considered the difference between starting HR values and all HR values until steady state. Net values were calculated for each beat until three minute steady state and continued until exercise end during the IR and C-R condition. Net HR values were subject to decimal logit transformation using the remodelled logarithm equation (equation 1) and allied with log time. The logarithm was reconstructed and the maximal characteristic of the original equation (x_m) was substituted with the peak HR value for each participant during both exercising conditions. Log $t_{1/2}$ was defined as corresponding to logit = 0.

MRT for individual HR values were also calculated from beat-by-beat data. HR kinetics was also assessed during the first three minutes of running exercise to steady state. Please see 'Pulmonary Oxygen Uptake' section for justification of the use of three minute steady state. O_2 values were substituted with respective HR values and HR deficit ([t * ΔHR - ∑HR]) and HR MRT ([t * ΔHR - ∑HR]/ ΔHR) values were calculated for each participant during the IR and C-R conditions as per the corresponding $\dot{V}O_2$ values (equation 2). Rate constant, k, values were also calculated for HR according to equation 3 (k = $HR_{(steady\ state)}$/ HR deficit), where O_2 values were substituted with HR values.

3. MUSCLE RECRUITMENT ACTIVITY AND METABOLIC RESPONSE DURING RUNNING AFTER CYCLING COMPARED TO ISOLATED RUNNING.

3.1 Introduction

Successful performance in triathlon is dependent on the ability of an athlete to overcome the complications of transitioning between disciplines, the most crucial of which is the cycle-run transition (Bonacci, Green, et al., 2010; Millet & Vleck, 2000). However, running after cycling is considered more stressful compared to IR and therefore, limiting cycling-induced changes to an athlete's biomechanical and physiological variables is of significant importance (Millet & Vleck, 2000).

Previous research has established the impaired effects of prior cycling on subsequent running economy performance among a variety of triathlete populations (Hausswirth et al., 1997; Hue et al., 1998; Millet & Bentley, 2004). Results showed that recreational athletes exhibit considerably higher levels of ventilatory frequency (Hue, Le Gallais, Boussana, Chollet, & Prefaut, 2000) (f_b), ventilatory efficiency ($\dot{V}_E$) and a higher oxygen demand of respiratory muscles (Millet et al., 2000) compared to their elite counterparts. Furthermore, C-R bouts have previously shown increases in mean $\dot{V}O_2$, $\dot{V}_E$ and HR (Guezennec et al., 1996) paired with increases to the overall cost of running (Hausswirth et al., 1996) ranging between 1.6-11.6% (Millet & Vleck, 2000) compared to IR. In particular, within the first minutes of running after cycling $\dot{V}O_2$, f_b, $\dot{V}_E$ and HR are all elevated compared to the same time during IR. Several possible reasons behind the observed increase in $\dot{V}O_2$ and running economy include a metabolic shift towards fat oxidation (Guezennec et al., 1996; Hue et al., 1998),

ventilatory muscle fatigue and dehydration (Bonacci et al., 2009). However, other studies have suggested that alterations to muscle recruitment patterns may influence running economy (Bonacci et al., 2009) and indirectly $\dot{V}O_2$ when running after cycling (Bonacci, Green, et al., 2010). This latter study looked at 15 moderately trained triathletes completed a control run and a transition run after 45 min of high intensity cycling. Muscle recruitment patterns were altered in seven of the 15 athletes and clinically meaningful alterations to $\dot{V}O_2$ were observed; with the authors concluding that cycling related muscle recruitment changes are closely linked with alterations to running economy during subsequent running. However, this same group of researchers later published contrary research (Bonacci et al., 2011) showing, no prior low or high intensity cycling effects influenced neuromuscular control or running economy in seven elite international triathletes. A relationship between muscle recruitment and $\dot{V}O_2$ when running after cycling is far from conclusive, and likely dependent upon an athlete's experience and training history (Hausswirth et al., 1997). However, past studies have established a reasonable link between metabolic cost (e.g. $\dot{V}O_2$) and muscle recruitment and activity during constant load exercise (Burnley et al., 2002; Moore et al., 2014; Paavolainen, Nummela, Rusko, & Häkkinen, 1999; Saunders et al., 2000).

The majority of the literature observing physiological alterations during running following cycling has stated the negative impact of the prior exercise, reflected by increases to mean $\dot{V}O_2$. Alternatively, research specifically focused on 'priming' exercise often demonstrate somewhat different changes, nominally 'speeding' of $\dot{V}O_2$ during the early phase of subsequent exercise (Burnley et al., 2001; DeLorey, Kowalchuk, Heenan, Dumanoir, & Paterson, 2007; Gerbino et al., 1996; Koppo &

Bouckaert, 2000; Murias et al., 2011; Scheuermann et al., 2002). The 'speeding' of $\dot{V}O_2$ likely reduces time to physiological steady state and the rate at which muscle fatigue develops by minimising muscle O_2 deficit (Burnley & Jones, 2007). The precise mechanisms behind altered $\dot{V}O_2$ following 'priming' exercise are not yet resolved however, alterations to motor unit recruitment profiles (Jones, DiMenna, et al., 2008) have been flagged as a potential contributor. Interestingly, similar 'speeding' effects may be likely when running after cycling, as cycling would represent a 'priming' exercise response. Paired with known changes to muscle recruitment patterns, there is cause to suggest that prior cycling or 'priming' exercise before subsequent running may not always be negative; it may actually serve to 'speed' $\dot{V}O_2$ during the C-R transition and have some positive effects. Understanding changes in muscle recruitment, resulting from prior cycling exercise, may potentially assist in identifying changes and physiological response of early phase changes in $\dot{V}O_2$ during the subsequent cycle-run transition phase within triathlete populations. The challenge is to recognise where within the transition period these $\dot{V}O_2$ and muscle recruitment pattern changes occur, whether they are related and if they are a result of prior cycling in the absence of fatigue.

The purpose of this chapter was therefore to investigate potential alterations in muscle recruitment patterns and physiological variables during running after cycling in comparison to isolated running. Our hypotheses are stated in section 1.8 under 'Aims and Hypotheses'.

3.2 Methods

3.2.1 Participants

All six (5 males and 1 females; *n*=6) elite triathletes had experienced Australian National level and/or International Triathlon Union (ITU) level competition (4.4±1.1 years) and had gained this experience in at least the year preceding testing. Table 3.1 outlines participant training history. All participants filled out a self-screening physical activity readiness questionnaire (PAR-Q) that was used to determine the level of safety and possible risk of exercise testing for the individual based upon their answers to specific health related and previous exercise history questions. Participants were also provided with an information package that described in detail all the procedures and requirements of the exercise testing. Prior to testing all participants provided written informed consent acknowledging their understanding of the procedures, requirements, associated risks, outcomes and any future publication of the research they were to participate in. On the day of testing verbal consent was also given by all participants prior to taking part in the experimental process. In order to preserve confidentiality and anonymity, participants were allocated a numerical code. All procedures employed with this study were approved by the University of Wollongong Human Research Ethics Committee in accordance with the *National Statement on Ethical Conduct in Human Research* (HE12/331).

Table 3.1: Participant training profiles (mean ± SD).

Age (years)		24.8 ± 3.6
Experience (years)[a]		4.4 ± 1.1
Training hours (h)[b]		24 ± 4
Training distance (km)[c]		
	cycling	328 ± 65
	running	60 ± 25
Sessions[d]		
	cycling	4 ± 1
	running	6 ± 2
	cycle-run	1.1 ± 0.4

[a] Years of triathlon experience at National or International level.
[b] Mean training hours recorded per week in the three months preceding testing.
[c] Mean training distance (km) recorded per week in the three months preceding testing.
[d] Mean number of training sessions recorded per week in the three months preceding testing.

3.2.2 Anthropometric Measurements

Anthropometric data was recorded for all six participants. Group mean values were calculated and are presented in table 3.2. Length, width and thickness measures were used to develop individual scale models to assist with reconstruction of EMG waveforms.

Table 3.2: Participant anthropometric measurements (mean ± SD).

Height (cm^{-1})		178.4 ± 7.2
Mass (kg^{-1})		69.1 ± 6.3
Leg length[a]	*R*	97.3 ± 3.3
	L	97.5 ± 3.4
Knee width[a]	*R*	9.8 ± 0.4
	L	9.9 ± 0.3
Ankle width[a]	*R*	7.6 ± 0.2
	L	7.3 ± 0.2
Elbow width[a]	*R*	7.3 ± 0.1
	L	7.4 ± 0.2
Wrist width[a]	*R*	5.5 ± 0.3
	L	5.5 ± 0.4
Hand thickness[a]	*R*	2.9 ± 0.2
	L	2.9 ± 0.1

[a] Mean value of two recorded measures from the same anthropometric site.
R Denotes the mean recorded value from the right side.
L Denotes the mean recorded value from the left side.

3.2.3 Measured Muscle Recruitment Activity

Muscle recruitment patterns were measured according to the procedures outlined under section 2.3.1 'Electromyography Recording of Muscular Recruitment'.

3.2.4 Measured Oxygen Uptake

Pulmonary oxygen uptake was measured according to the procedures outlined under section 2.3.3 'Measurement of Pulmonary Oxygen Uptake'.

3.2.5 Measured Heart Rate

Heart rate was measured according to the procedures outlined under section 2.3.4 'Measurement of Heart Rate'.

3.3 Statistical Analysis

3.3.1 Electromyography

Repeatability of EMG data for all muscle sites were analysed for all individual participants using a Pearson's coefficient of correlation (*r*) due to being sensitive to variations in waveform shape. Correlation coefficient (*r*) of between 0.80 and 1.00 were defined as representing 'good' reproducibility, scores between 0.60 and 0.79 were considered 'fair' and scores less than 0.60 as 'low' (Sleivert & Wenger, 1994). To determine if potential cycling influenced changes on muscle recruitment patterns between the IR and C-R conditions, individual mean EMG traces for $t_{1/2}$, MRT and 180 s (3-min steady state) during the C-R condition were reconstructed and compared with their respective IR (control) data collected from the final minute of running. Traces were deemed significantly different between the IR and C-R conditions if the mean EMG trace during C-R condition timeframes exceeded 10% of mean IR trace, in accordance with the analysis procedures outlined by Bonacci, Green, et al. (2010). Mean variation of EMG waveforms between IR and C-R EMG traces were assessed via calculation of the coefficient of variation for all four timeframes (IR, C-R $t_{1/2}$, MRT and 180 s). The coefficient of variation (%) was calculated as the root mean square of the standard deviation over the period of a stride divided by the mean collection waveform over the 20 stride-sampling period (Winter & Yack, 1987).

Group main effects were compared by differences retested using analysis of variance (ANOVA) with Bonferroni adjusted pairwise multiple comparisons of within-individual differences at each point. Comparisons of mean and peak EMG

amplitudes between time intervals were made using repeated measures ANOVA for all individual triathletes. Root mean square error of muscle activity recordings, from the 9^{th}-10^{th} minute of the IR were compared with data from $t_{1/2}$, MRT and 180 s, from the C-R using repeated-measures ANOVA.

3.3.2 Stride Length and Stride Frequency

Individual variance of stride length variation between the 9^{th}-10^{th} minute of the IR and $t_{1/2}$, MRT and 180 s for the C-R where calculated using Pearson's correlation of multiple coefficients (*r*). Similarly, stride rate variation between minutes nine and ten of both respective conditions were compared using Pearson's correlation of multiple coefficients (*r*). Percentages of variance were also recorded for individual stride frequency data to determine the level of variation.

3.3.3 Oxygen Uptake

Mean values for $\dot{V}O_2$ data were compared using paired *t*-tests. Similarly, individual $t_{1/2}$ and MRT $\dot{V}O_2$ values were compared and differences are reported as percentage variance. Similarities between group mean results for steady state, $t_{1/2}$ and MRT $\dot{V}O_2$ during the IR and C-R conditions were compared using Pearson's correlation of coefficients (*r*). Group mean $t_{1/2}$ and MRT $\dot{V}O_2$ values were correlated during the respective conditions to determine similarities between the two calculated variables. Analyses of the relationship between $\dot{V}O_2$ and HR values for $t_{1/2}$ and MRT during the IR and C-R conditions were compared using Pearson's correlation coefficients (*r*).

3.3.4 Heart Rate

Paired *t*-tests were used to identify significant differences between group mean HR for steady state, $t_{1/2}$ and MRT variables for the IR and C-R conditions. Pearson's

correlation coefficient (r) was used to determine the relationship between $t_{1/2}$ HR values during the IR and C-R conditions. Similarly, HR MRT was correlated between respective running conditions. Differences between individual HR values during the IR and C-R conditions were reported as percentage change.

Statistical analysis was carried out using IBM SPSS 21 (IBM Corporation, Armonk, NY), with statistical significance set at $p<0.05$, unless stated otherwise. Descriptive data are reported as means with standard deviation (± SD).

3.4 Results

3.4.1 Running Stride Control between Isolated and Cycle-Run Conditions

Mean participant running speeds for both the IR and C-R conditions was 12.1±0.8 km/h. Running speed was predetermined by each individual participant during the 5-min warm-up procedure and clamped at this speed during both running conditions (also addressed in Section 2.2.3). Stride length data for all participants during the IR and C-R bouts are displayed in table 3.3. Individual stride lengths demonstrated relatively small levels of variation between the IR and C-R conditions. Furthermore, C-R $t_{1/2}$, MRT and 180 s phases. Similarly, group mean results for stride length during the IR and C-R conditions showed no differences, instead demonstrating high levels of correlation (r=0.96-0.99) between each recorded timeframe. Mean stride frequency across both exercising conditions showed strong correlations demonstrating relatively high levels of homogeneity (r=0.97), while low levels of individual variation are reported between the IR and C-R conditions (Table 3.4).

Table 3.3: Individual and group mean stride length (cm).

Mean Stride Length (cm)								
Participant	1	2	3	4	5	6	Mean ± SD	*r*-value
IR	159.0	150.0	155.7	120.2	147.2	158.6	148.4 ± 14.6	
C-R 30 s	157.7	147.6	151.5	121.0	146.9	160.2	147.5 ± 14.1	0.96*
C-R t1/2	156.6	148.2	152.9	119.0	145.2	158.5	146.7 ± 14.5	0.98*
C-R 180 s	157.6	145.8	154.3	120.1	146.9	160.0	147.4 ± 14.5	0.99*

Note: * denotes significant similarities when compared with IR data, p<0.05.
Individual mean stride length was calculated as the average of 20 strides at the 9-10th minute mark of the IR condition. For the C-R exercise conditions, data was averaged for 20 strides most near to $t_{1/2}$, MRT and 180 s time periods.

Table 3.4: Group mean stride frequency (stride/min) presented with individual stride variance.

Mean Stride Frequency (stride/min)								
Participant	1	2	3	4	5	6	Mean ± SD	*r*-value
IR	135	143	136	137	137	134	137 ± 3.2	
C-R	137	144	138	138	139	135	139 ± 3.0	0.97*
% Variance	1.5	0.7	1.5	0.7	1.5	0.7	1.1 ± 0.4	

Note: * denotes significant similarities when compared with IR data, $p<0.05$.
Individual stride variation is presented as the percentage difference between the mean stride calculations for the IR and C-R. Twenty (20) strides collected at the 9-10th minute of each running condition was used to calculate mean stride frequency.

3.4.2 Oxygen Uptake during Isolated versus Cycle-Run Conditions

Table 3.5 presents the $\dot{V}O_2$ kinetic variables measured during the IR and C-R conditions for the six participants in which EMG were also recorded. Starting $\dot{V}O_2$ was higher for all participants in the C-R condition, compared to the IR condition (p=0.02). No difference in the rate of change in $\dot{V}O_2$, being the relative consumption of oxygen used per minute was identified when comparing running conditions. Group average 10th min and steady state $\dot{V}O_2$ during the IR and C-R conditions were not different, instead demonstrating relatively high levels of correlation (r = 0.77 and 0.76 respectively). Group average $\Delta\dot{V}O_2$ was different between conditions (p=0.01).

Table 3.5: Individual and group mean $\dot{V}O_2$ kinetic values presented for the IR and C-R conditions.

Participant		1	2	3	4	5	6	Mean ± SD
VO_2: *IR vs. C-R*								
Starting VO_2	IR	4.0	4.9	4.9	6.0	4.2	5.1	4.8 ± 0.7
	C-R	9.4	19.3	11.1	6.6	8.9	15.2	11.7* ± 4.7
10th min VO_2	IR	39.6	38.7	28.6	34.4	33.5	34.3	34.8 ± 4.0
	C-R	36.7	39.4	28.3	39.8	35.9	35.1	35.9 ± 4.2
SS	IR	39.6	38.7	28.6	34.4	33.5	34.3	34.8 ± 4.0
	C-R	37.1	40.1	29.5	40.5	34.7	33.6	35.9 ± 3.9
ΔO_2	IR	2541.9	2177.6	1318.1	2011.4	2077.8	2125.2	2042.0 ± 400.4
	C-R	1639.4	1578.8	945.5	1782.0	1456.5	1535.4	1489.6* ± 288.2

Note: * denotes significant difference between IR and C-R values, $p<0.05$. Group mean results are presented as mean ± SD. All variables units are represented as ml.kg^{-1}.min^{-1}, except ΔO_2. Steady state is abbreviated as SS.

Individual $t_{1/2}$ values for $\dot{V}O_2$ (Table 3.6), with the exception of participant four, showed considerable variance when comparing IR with C-R data (Figure 3.1). In particular participants two and six reached $\dot{V}O_2$ $t_{1/2}$ 19 and 29 s respectively sooner during the C-R bout than during the IR bout. The represented variance indicating faster $t_{1/2}$ values during the C-R condition and was further reflected in the difference between population mean $t_{1/2}$ for the IR and C-R conditions (p <0.01). $\dot{V}O_2$ MRT for individuals (Table 3.6) between conditions showed a relatively high level of variance, with the mean percentage difference being 55.2±10.1% (Figure 3.1). Group mean values for IR and C-R conditions demonstrate considerably faster MRT during the C-R bout (p<0.01). Pearson's correlations showed no significant similarity between MRT times during the IR and C-R conditions (r=0.15). Oxygen deficit was different between conditions (p<0.01), as was the mean rate constant (p<0.01). The rate constant difference (k diff) was reported for each participant. Figures 3.1 and 3.2 shows the percentage change in $t_{1/2}$ and MRT values for $\dot{V}O_2$ during the IR and C-R conditions, with both variables demonstrating a considerable reduction in $t_{1/2}$ and MRT values during the C-R condition.

Table 3.6: Individual and group mean $t_{1/2}$ and MRT values presented for the IR and C-R conditions.

Participant		1	2	3	4	5	6	Mean ± SD
VO_2 : IR vs. C-R								
$t_{1/2}$ (s)	IR	69.3	52.6	76.7	62.5	75.6	71.4	68.0 ± 9.1
	C-R	48.7	46.1	60.5	52.4	46.2	46.4	50.1* ± 5.7
MRT (s)	IR	135.4	133.2	113.9	127.5	126.5	126.3	127.1 ± 7.5
	C-R	69.7	45.8	44.5	75.3	44.4	62.6	57.1* ± 13.9
O_2 Deficit	IR	5737.2	4836.0	2501.2	4272.7	4379.8	4472.1	4366.5 ± 1058.2
	C-R	2197.0	1613.3	1184.6	2593.2	1364.7	1785.9	1789.8* ± 527.2
k	IR	0.4	0.5	0.5	0.5	0.5	0.5	0.5 ± 0.0
	C-R	0.7	1.0	0.8	0.7	1.1	0.9	0.9* ± 0.1
k diff		0.3	0.5	0.3	0.2	0.6	0.4	0.4 ± 0.1

Note: * denotes significant difference between IR and C-R values, $p<0.05$. Group mean results are presented as mean ± SD. O_2 deficit values are presented as $ml.kg^{-1}.min^{-1}$.

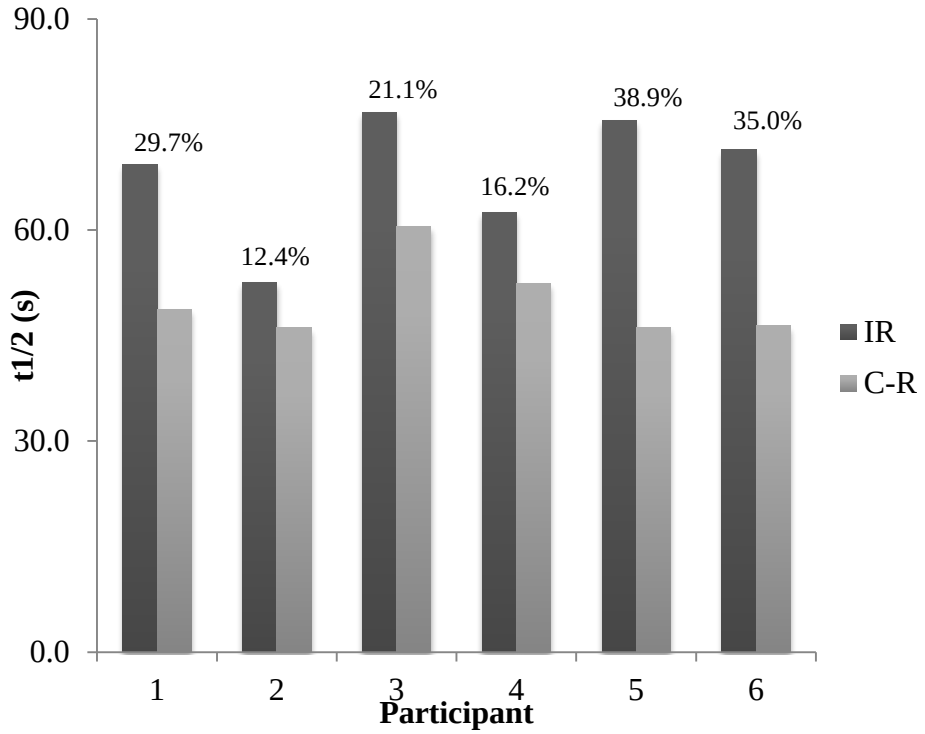

Figure 3.1: Individual $\dot{V}O_2$ $t_{1/2}$ values presented for IR and C-R conditions. Percentage differences between conditions are above columns. All participants recorded reductions in $t_{1/2}$ values during the C-R condition. Mean percentage variance between conditions was 25.5±10.6%.

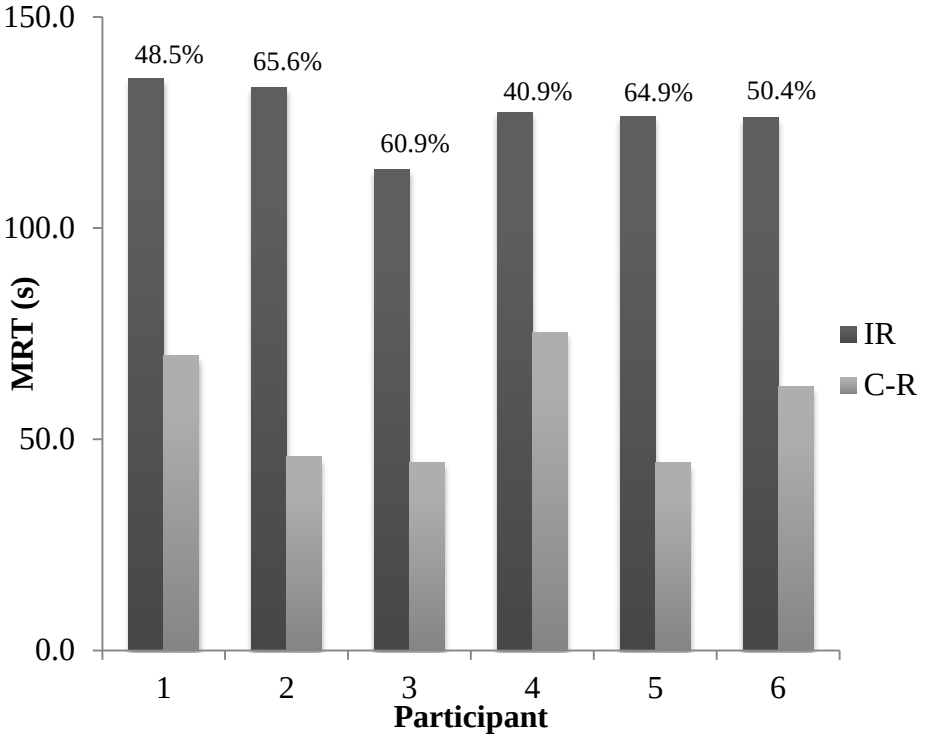

Figure 3.2: MRT for all individual participants are presented with percentage difference between the IR and C-R running conditions. All participants showed substantial decreases in MRT during the C-R condition with the mean percentage variance between conditions was 55.2±10.1%.

3.4.3 Heart Rate during Isolated versus Cycle-Run Conditions

Heart rate kinetics (Table 3.7) exhibited some similar trends to the $\dot{V}O_2$ results. Starting HR values for the C-R bout were significantly elevated ($p<0.01$) when compared with those for the IR bout. A mean difference of 28±16 bpm ($p<0.01$) was demonstrated when comparing the IR and C-R bouts. This was reflective of the variance in the rate ΔHR ($p<0.01$) between conditions, although these differences appeared to have no significant influence on the 10th min or steady state HR values that demonstrated a high degree of uniformity during both the IR and C-R conditions (r=0.99 and 0.99, respectively).

Table 3.7: Individual and group mean HR values present for the IR and C-R conditions.

Participant		1	2	3	4	5	6	Mean ± SD
HR: IR vs. C-R								
Starting HR	IR	53	68	80	65	65	77	68.0 ± 9.7
	C-R	114	98	110	112	79	121	105.7* ± 15.1
10th min HR	IR	151	136	156	135	129	159	144 ± 13
	C-R	158	143	162	143	133	168	151 ± 14
SS	IR	152	136	155	135	129	159	144.3 ± 12.5
	C-R	157	142	162	143	130	167	150.2 ± 14.1
Δ HR	IR	99	68	75	70	64	82	76.3 ± 12.7
	C-R	43	44	52	31	51	46	44.5* ± 6.9

Note: *Denotes significant difference between IR and C-R values, $p<0.05$. Group mean results are presented as mean ± SD. All variables units are represented as (bpm). Steady state is abbreviated as SS.

Changes in HR $t_{1/2}$ values (Table 3.8) were not different between conditions (p=0.20; r=0.75). Individual percentage variance (Figure 3.3) between IR and C-R conditions showed that participant's one, two, three and four had substantially different HR $t_{1/2}$ between conditions. Of those four participants, three demonstrated slower HR $t_{1/2}$ during the C-R bout, whereas the other participant demonstrated a 36.8% or 23.3 s

increase in HR $t_{1/2}$. Despite this, overall mean $t_{1/2}$ values for HR were not statistically different, exhibiting a negative correlation between running bouts (p=0.21; r= -0.75). Heart rate MRT values (Table 3.8) also showed relatively high levels of mean percentage variance. Three participants showed considerable increases in MRT, particularly for participants five who presented a difference of 103.6% between their IR and C-R MRT for HR. The remaining three participants all showed reductions in MRT for HR.

Despite the substantial individual difference in mean percentage variance, group mean MRTs were not different between conditions (p=0.45) and showed no correlation (r=0.06). Group mean HR deficit did exhibit a significant difference between the IR and C-R conditions (p=0.03). However the group mean k was unchanged between conditions.

Table 3.8: Individual and group mean values for calculated HR data.

Participant		1	2	3	4	5	6	Mean ± SD
HR: IR vs. C-R								
$t_{1/2}$ (s)	IR	35.8	35.5	63.3	40.1	40.4	47.8	43.8 ± 10.5
	C-R	54.6	58.2	40.0	51.9	41.1	44.5	48.4 ± 7.6
MRT (s)	IR	93.9	83.4	66.4	86.2	46.8	99.9	79.4 ± 19.6
	C-R	70.2	104.0	101.2	69.1	95.4	86.0	87.6 ± 15.2
HR Deficit	IR	155.0	94.5	83.0	100.5	50.0	136.5	103.2 ± 37.7
	C-R	50.3	76.3	87.7	35.7	81.1	65.9	66.3* ± 19.8
k	IR	0.6	0.7	0.9	0.7	1.3	0.6	0.8 ± 0.3
	C-R	0.9	0.6	0.6	0.9	0.6	0.7	0.7 ± 0.1
k diff		-0.2	0.1	0.3	-0.2	0.7	-0.1	0.1 ± 0.3

Note: *Denotes significant difference between IR and C-R values $p<0.05$. Group mean results are presented, as mean ± SD. HR deficit values are in bpm.

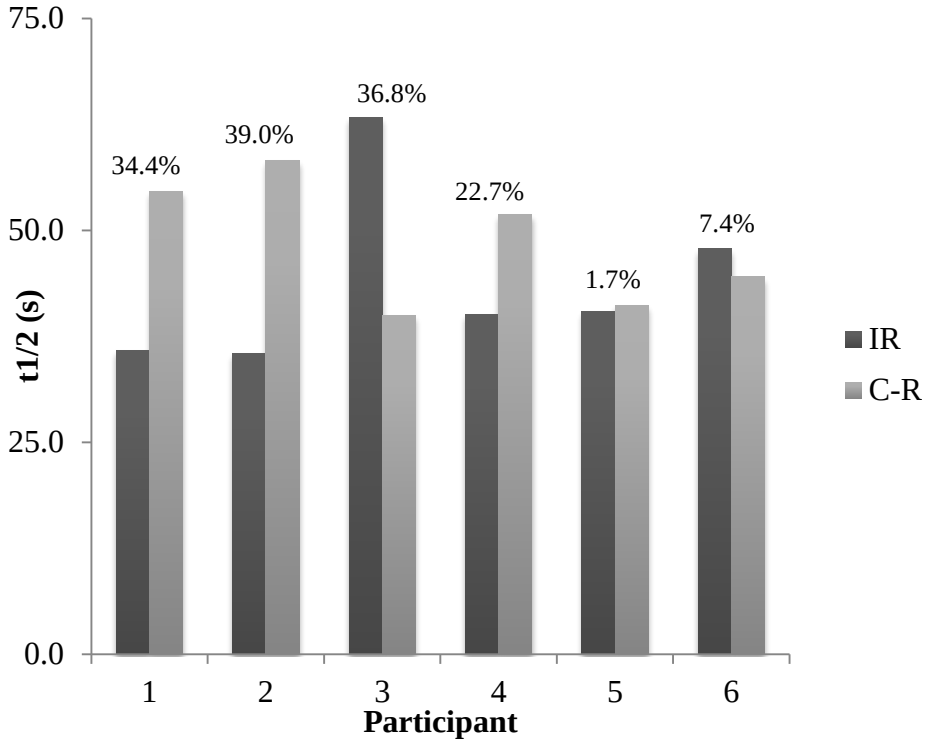

Figure 3.3: $t_{1/2}$ values for all individual participants are presented with the percentage difference between the IR and C-R running conditions.

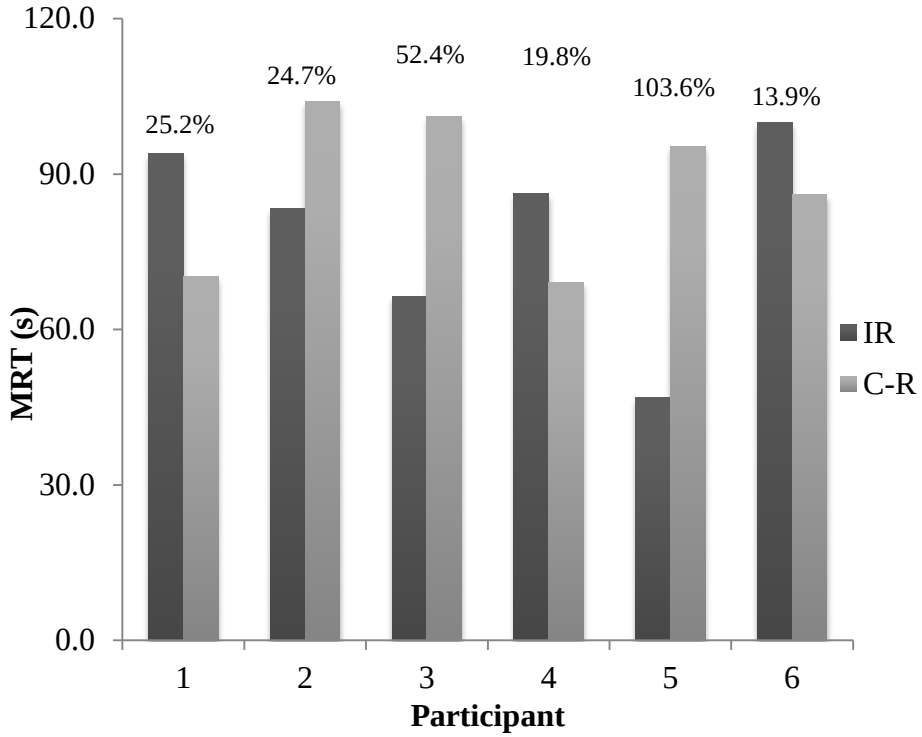

Figure 3.4: Individual MRTs for IR and C-R conditions are presented with the percentage difference between conditions.

3.4.4 The Relationship between Oxygen Uptake and Heart Rate during Isolated and Cycle-Run Exercise

An analysis of the relationship between $\dot{V}O_2$ and HR variables during respective IR and C-R conditions is presented in Figures 3.5 and 3.6. Neither of the two running bouts demonstrated any significant correlation between the $t_{1/2}$ values for $\dot{V}O_2$ and HR (IR: r=0.62; p=0.19 and C-R: r= -0.39; p=0.44). Comparison of individual results showed substantial variability for $\dot{V}O_2$ $t_{1/2}$ values during the IR compared with the C-R condition (Figure 3.5). Specifically, four of six participants saw marked decreases, although exceptions included participant two who showed little change in $\dot{V}O_2$ $t_{1/2}$. For HR $t_{1/2,}$ three participants showed considerable increases during the C-R condition. Of the other three participants, one showed a marked reduction in HR $t_{1/2}$ during the C-R compared with the IR, while the remaining participants only showed minor differences between conditions.

The relationship between $\dot{V}O_2$ and HR MRT's are plotted in Figure 3.6. MRTs for $\dot{V}O_2$ and HR during the C-R demonstrated a negative correlation (r= -0.96; p<0.01). No meaningful correlation appeared between $\dot{V}O_2$ and HR MRT during the IR condition. Similar to the $t_{1/2}$ values, group mean $\dot{V}O_2$ MRT saw a reduction during the C-R condition, while HR MRT saw a slight increase during the C-R compared with the IR condition.

The X and Y reference lines for the IR (full) and the C-R (dotted) represent the mean values for $\dot{V}O_2$ and HR respectively, outlining an overall drop in mean $\dot{V}O_2$ $t_{1/2}$, contrary to a small increase in HR $t_{1/2}$ when comparing the IR with the C-R results (Figure 3.6). Similar results are shown for $\dot{V}O_2$ and HR MRTs; however the

difference between $\dot{V}O_2$ MRT was much greater than the difference between the respective IR and C-R condition $t_{1/2}$.

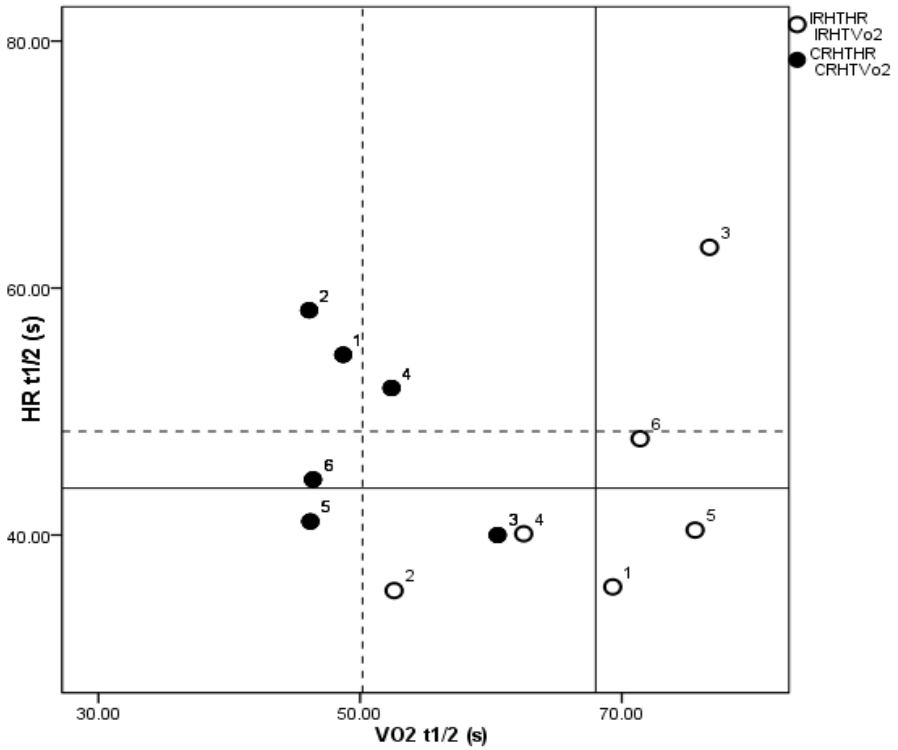

Figure 3.5: Presented are individual coordinates representative of respective $t_{1/2}$ calculated for the IR and C-R conditions. Axis lines represent the group mean values for the respective conditions. Mean lines show a significant (p<0.05) drop in $\dot{V}O_2$ $t_{1/2}$ for the C-R (broken line) compared to the IR (unbroken line) condition. However, mean lines representing HR from the C-R (broken line) and IR (unbroken line) conditions showed no significant variation.

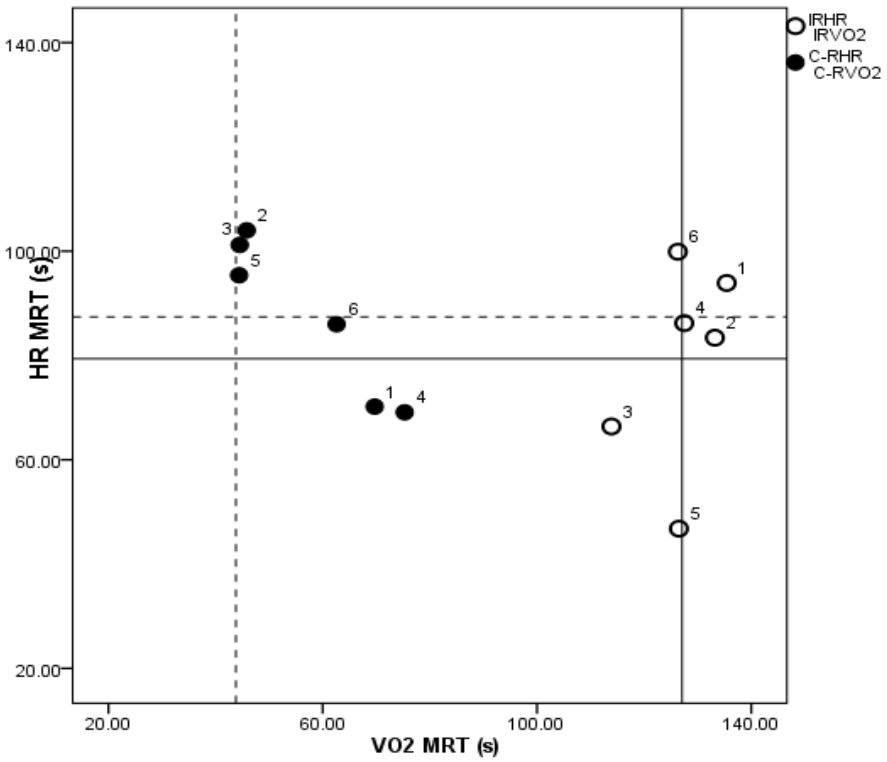

Figure 3.6: Presented are individual coordinates representative of respective MRT calculated for the IR and C-R conditions. The mean lines represent the group mean value for the respective conditions. Mean lines show a significant (p<0.05) drop in $\dot{V}O_2$ MRT for the C-R (broken line) compared to the IR (unbroken line) condition. However, mean lines representing HR from the C-R (broken line) and IR (unbroken line) conditions showed no significant variation.

3.4.5 Muscle Recruitment Activity during Isolated Run and Cycle-Run Conditions

Electromyography data for the IR and $t_{1/2}$, MRT and 180 s time periods of the C-R condition were reconstructed from the 20 stride averages for all six participants. Muscle activity in the IR and a four times during the C-R runs are plotted in Figure 3.7 as the average (±SD) waveform for all recorded muscles presented for one individual participant. The variability of the recorded EMG muscle activity as represented in Figure 3.7 does demonstrate a noticeable increase during the $t_{1/2}$, MRT and 180 s sampling periods when compared with the IR traces. The trend of increased variability in the C-R condition was consistent among all participants. Coefficients of variation values (group mean ±SD) representing the inter-subject

variability is presented in Table 3.9, along with mean difference (*diff*) values, and Figure 3.8 shows mean values for each muscle at each time period. These traces show that following cycling, running EMG muscle recruitment did demonstrate a reasonable level of variability that remained consistent across all measured muscles sites and further analysis of Table 3.9 indicates that the coefficient of variation measured during the C-R were substantially different. Across all sampling periods during the C-R condition, the coefficient of variation remained consistent however; variation appeared greater during sampling at 180 s (steady state), particularly for the GM, VL, RF, MG and LG muscles. Furthermore, difference in variation at $t_{1/2}$ (*diff* $t_{1/2}$) was slightly higher than those measured at individual MRT and at steady state. Although not substantially different, it is conceivable that the greater variation measured earlier on, at $t_{1/2}$, was more influenced by preceding cycling patterns.

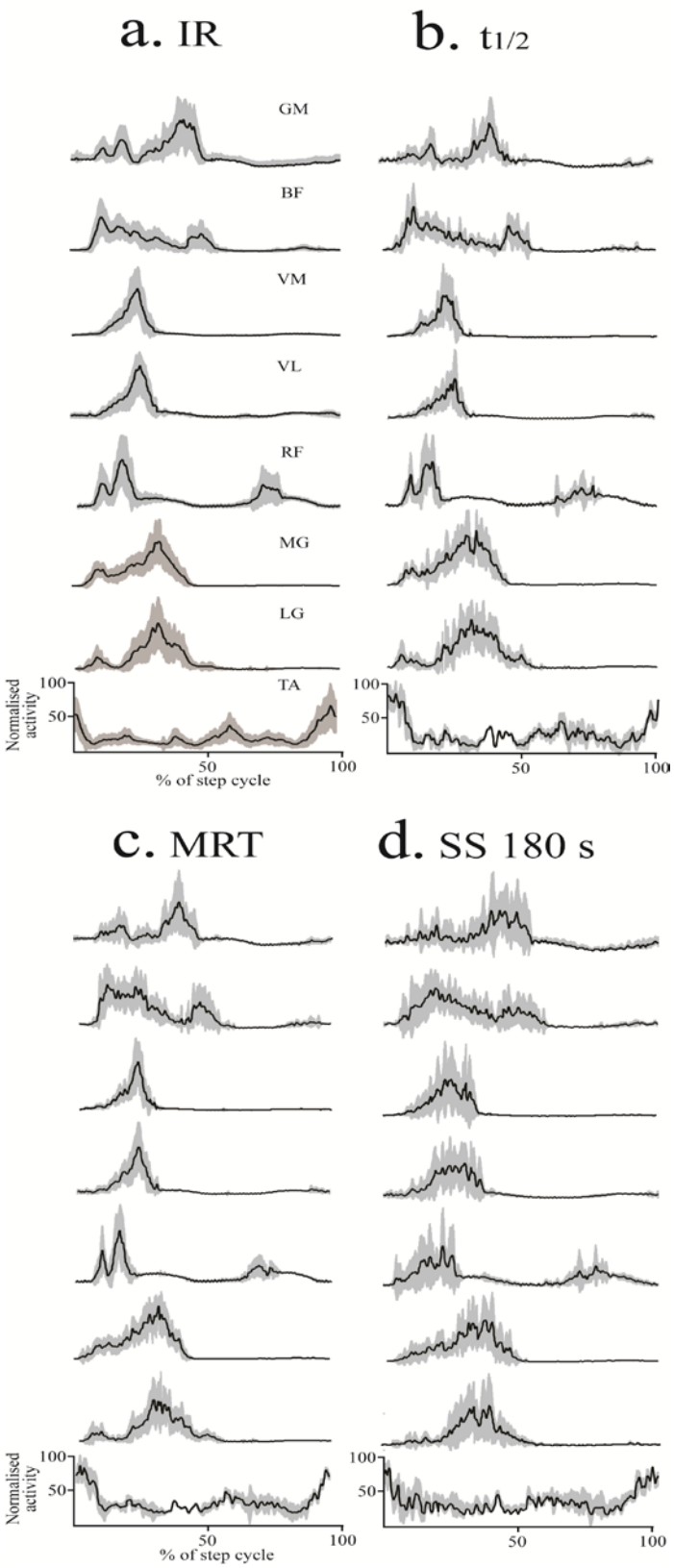

Figure 3.7: Plotted are the mean (± SD) EMG waveforms for all sampling periods for one representative participant. Normalised EMG activity was plotted as a percentage of the maximal recorded EMG in the y-axis and as a percentage of the step (gait) cycle on the x-axis. Individual mean waveforms (solid black line) were highly similar across each sampling period for all individuals. However, EMG variability as represented by the SD (grey shading) was reasonably high for all individuals during the C-R condition.

Note: For abbreviations of muscles, see section 2.3.1, Table 2.2 and Figure 2.1.

Table 3.9: Coefficient of variation and mean difference values pertaining to Figure 3.7 for one individual participant.

Inter-participant coefficients of variation (CV%)							
Muscle	**IR**	**$t_{1/2}$**	**MRT**	**180s (SS)**	***diff* $t_{1/2}$**	***diff* MRT**	***diff* 180s (SS)**
GM	21.3	44.3	45.3	46.2	24.8	20.6	23.0
BF	17.8	40.5	43.7	41.7	23.8	22.1	22.7
VM	20.3	41.3	44.5	42.3	22.0	20.7	21.0
VL	18.2	40.2	43.0	46.0	27.8	21.3	22.0
RF	23.2	41.5	42.7	44.2	21.0	16.7	18.3
MG	19.5	53.0	54.2	56.8	37.3	29.7	33.5
LG	22.7	50.0	51.0	52.3	29.7	24.3	27.3
TA	20.5	40.8	44.7	39.7	19.2	20.7	20.3

Note: For abbreviations of muscles, see section 2.3.1, Table 2.2 and Figure 2.1. Steady state is abbreviated as SS.

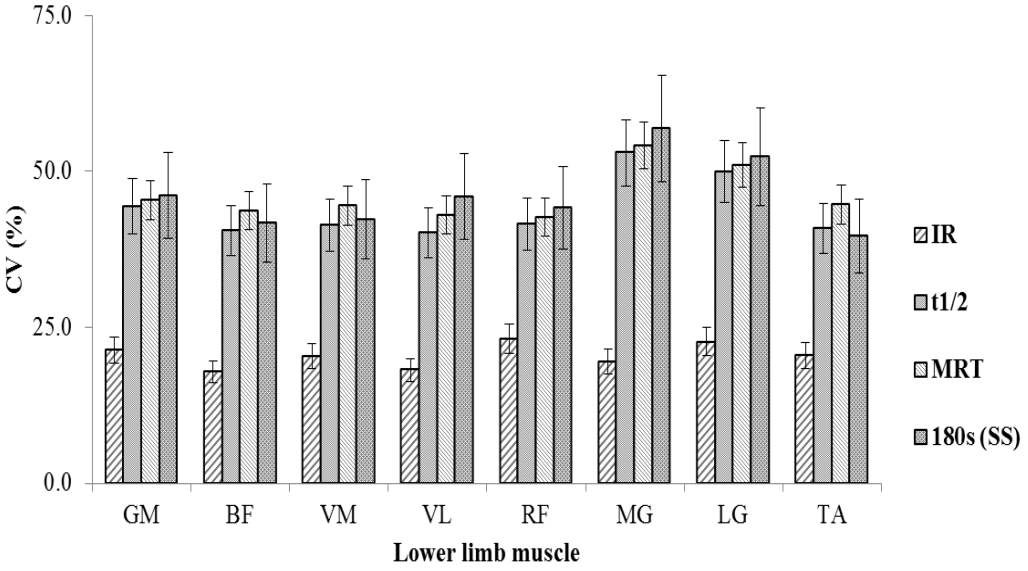

Figure 3.8: Plotted are the population mean (± SE) coefficient of variation (CV) for all recorded muscle sites over all sampling periods. Steady state is abbreviated as SS.
Note: For abbreviations of muscles, see section 2.3.1, Table 2.2 and Figure 2.1.

Table 3.10 presents the results for average EMG amplitude (as a percentage of peak amplitude) for all individual waveforms with no difference being shown between the IR and the C-R conditions. Additionally, correlation coefficients shown in Table 3.11

demonstrate that individual mean EMG waveforms showed consistently high levels of correlation and were statistically similar (p<0.05) between the IR and C-R conditions. Similarly, analysis of peak EMG data showed no difference in any peak muscular activity for any participant throughout the IR or C-R conditions. Furthermore, root mean square error for EMG traces for the IR condition averaged at 3.4±0.2% and 4.3±0.2 4.1±0.3 and 4.2±0.2% respectively for the C-R condition. Analysis of RMSE values showed no significant difference between the IR and C-R conditions or within the C-R condition.

Table 3.10: Mean EMG amplitudes for all muscle sites.

Average EMG Amplitidue (% of peak Amplitude)								
	GM	BF	VM	VL	RF	MG	LG	TA
IR	21.9 ± 12.8	21.5 ± 11.9	14.0 ± 7.7	11.7 ± 6.0	42.7 ± 18.5	21.8 ± 3.2	28.2 ± 11.4	27.4 ± 13.8
C-R 30 (s)	20.4 ± 12.3	19.5 ± 9.6	11.7 ± 5.4	12.3 ± 5.9	43.6 ± 13.5	20.1 ± 3.7	32.3 ± 10.9	33.0 ± 7.4
C-R t1/2	20.3 ± 11.8	21.5 ± 11.2	12.5 ± 6.7	16.4 ± 5.0	41.5 ± 13.7	25.0 ± 9.8	31.1 ± 13.8	33.9 ± 4.9
C-R 180 (s)	24.8 ± 14.4	20.1 ± 13.5	12.4 ± 7.2	13.8 ± 8.6	36.2 ± 20.3	26.1 ± 9.3	31.1 ± 12.6	38. 9 ± 10.6

Table 3.11: Individual correlation coefficient values for all recorded muscle sites taken from the respective time periods (EMG traces per muscle for each collection time was *n*=80).

Individual Correlation Coefficients (*r* -values)							
	Participant	1	2	3	4	5	6
BF	C-R $t_{1/2}$	0.79	0.91	0.90	0.86	0.93	0.94
	C-R MRT	0.90	0.92	0.97	0.86	0.95	0.85
	C-R 180 s	0.76	0.91	0.90	0.88	0.86	0.87
	Participant	1	2	3	4	5	6
RF	C-R $t_{1/2}$	0.70	0.66	0.74	0.88	0.90	0.84
	C-R MRT	0.59	0.70	0.79	0.90	0.95	0.73
	C-R 180 s	0.52	0.72	0.81	0.86	0.69	0.75
	Participant	1	2	3	4	5	6
VL	C-R $t_{1/2}$	0.66	0.80	0.95	0.93	0.90	0.93
	C-R MRT	0.76	0.86	0.95	0.91	0.95	0.94
	C-R 180 s	0.58	0.80	0.95	0.93	0.86	0.89
	Participant	1	2	3	4	5	6
VM	C-R $t_{1/2}$	0.78	0.85	0.97	0.89	0.90	0.79
	C-R MRT	0.85	0.88	0.97	0.96	0.96	0.80
	C-R 180 s	0.58	0.90	0.95	0.87	0.90	0.74
	Participant	1	2	3	4	5	6
LG	C-R $t_{1/2}$	0.93	0.73	0.94	0.92	0.95	0.88
	C-R MRT	0.93	0.81	0.96	0.93	0.97	0.83
	C-R 180 s	0.88	0.79	0.94	0.91	0.90	0.78
	Participant	1	2	3	4	5	6
MG	C-R $t_{1/2}$	0.94	0.90	0.98	0.89	0.96	0.95
	C-R MRT	0.94	0.94	0.97	0.89	0.98	0.91
	C-R 180 s	0.87	0.93	0.97	0.91	0.90	0.86
	Participant	1	2	3	4	5	6
TA	C-R $t_{1/2}$	0.80	0.52	0.92	0.88	0.83	0.96
	C-R MRT	0.84	0.66	0.90	0.93	0.83	0.82
	C-R 180 s	0.71	0.86	0.86	0.85	0.54	0.76
	Participant	1	2	3	4	5	6
GM	C-R $t_{1/2}$	0.81	0.59	0.92	0.94	0.89	0.91
	C-R MRT	0.89	0.77	0.94	0.95	0.94	0.90
	C-R 180 s	0.83	0.79	0.90	0.95	0.86	0.92

Note: For abbreviations of muscles, see section 2.3.1, Table 2.2 and Figure 2.1.

3.5 Discussion

3.5.1 *Outcomes*

Running efficiently after cycling requires a high level of neuromuscular control that is not adversely impacted by prior cycling (Bonacci et al., 2011; Millet & Vleck, 2000). The main results of this study were that among a cohort of trained triathletes, cycling did not significantly impact the metabolic cost of running or that prior moderate intensity cycling did not adversely affect each participants' average muscle recruitment activity during subsequent running. However, in agreement with our first hypothesis (p. 27), the variability of muscle recruitment activity of the lower limb does appear to be amplified during running following prior cycling when compared with isolated running. Although, in further contrast to our third and fifth hypotheses (pp. 27-28), cycling prior to running does not significantly alter $\dot{V}O_2$ and HR variables at steady state, despite elevated starting values for $\dot{V}O_2$ and HR at the onset of the C-R condition.

For all participants, muscle recruitment activity across all muscles and sampling timeframes within the C-R conditions demonstrated a relatively high correlation with the muscle recruitment activity sampled during the IR condition. Similarly, we observed no difference in mean EMG amplitude or root mean square error between the IR and C-R conditions. These results indicate that muscle recruitment activity is preserved during moderate intensity running after cycling at a relatively similar intensity, and is consistent with previous investigations concluding that cycling has no adverse effect on subsequent running mechanics (Quigley, 1996) or muscle recruitment patterns during running after cycling (Bonacci, Blanch, et al., 2010;

Bonacci et al., 2011). However, our findings are in contrast to a previous study reporting that muscle recruitment was altered after cycling in 36% of highly trained triathletes tested (Chapman, Vicenzino, Blanch, Dowlan, et al., 2008) and 53% of moderately trained triathletes tested (Bonacci, Green, et al., 2010). We did observe an increase in muscle recruitment variability during running after cycling in this current study that are consistent with previous results (Chapman, Vicenzino, Blanch, Dowlan, et al., 2008). These authors further reported that changes to muscle recruitment persisted for the duration of the 30 min transition run. Our results show a similar trend in that, across all sampling periods during the C-R conditions the variability of muscle recruitment remained consistent. However, our results suggest that individual muscle recruitment activity was conserved, regardless of waveform variability, indicating that cycling of a similar relative intensity does not adversely influence the muscle recruitment activity during subsequent running in trained triathletes.

The lack of individual effects of prior cycling on the muscle recruitment activity and metabolic cost of subsequent running were unexpected and are contrary to previous studies that demonstrated significant and occasionally large individual effects when running after cycling (Bonacci, Green, et al., 2010). The variation among individuals are generally masked when interpreting group results signifying that potential changes to running patterns or metabolic variables following prior cycling exercise are highly individually-specific. However, training status and performance levels of respective participants may explain physiological and neuromuscular difference when running after cycling. Past research has confirmed that experienced or highly trained triathletes tend to display less mechanical and performance decrements as

opposed to their lesser-experienced peers (Millet et al., 2001; Millet et al., 2000). For instance, compared to elite or highly trained triathletes, middle level or moderately trained triathletes have previously displayed greater vertical displacement, acceleration and deceleration of their centre of mass (Millet et al., 2001); greater change to muscle recruitment and sagittal plane kinematics (Bonacci, Green, et al., 2010; Chapman, Vicenzino, Blanch, Dowlan, et al., 2008). Alternatively, the absence of real change in muscle recruitment activity may have been a result of a training effect. Bonacci et al. (2011) discussed a potential training effect present among highly trained triathletes as an explanation for the lack of alteration in muscle recruitment during running after a prior high-intensity cycling bout. The notion of a training effect or response minimising or removing substantial alterations to muscle recruitment during running after cycling is plausible. A relatively common form of triathlon-specific training is short multicycle-run exercises (Boussana et al., 2002). This training strategy has revealed significantly improved the cycle-run transition response in competitive triathletes (Hue, Valluet, Blonc, & Hertogh, 2002), by stimulating the same responses as the cycle-run transition encountered during racing (Hue, Le Gallais, Boussana, Chollet, et al., 2000; Hue, Le Gallais, Boussana, Galy, et al., 2000). The participants in this current study do complete cycle-run transition training (Table 3.1). As a result, it could be considered that the absence of significant change in muscle recruitment activity among triathletes in this study was a result of a training effect stemming from adaptation elicited from ‘multiblock’ training.

Additionally, the relative moderate intensity and short duration of the prior cycling bout may also have influenced the lack of individual change in muscle recruitment activity during the current study. However, previous research has employed both low

and high intensity cycle protocols prior to running and have reported little or no significant change to muscle recruitment patterns (Bonacci et al., 2011). There is evidence however, to the contrary (Chapman, Vicenzino, Blanch, Dowlan, et al., 2008), as previous research has cited that prior submaximal or moderate intensity exercise was not sufficient enough to influence subsequent physiological variables, including the speeding of $\dot{V}O_2$ (Burnley et al., 2000; Gerbino et al., 1996; Jones, DiMenna, et al., 2008).

Akin to the lack of change to muscle recruitment activity during the C-R condition, no meaningful differences in overall $\dot{V}O_2$ and HR were observed during the respective condition compared with the IR condition in any individual. Despite this and in agreement with our hypothesis, individual $\dot{V}O_2$ and HR data did demonstrate the greatest variable differences during the first minute of the C-R condition compared to the IR condition. The difference between starting values of $\dot{V}O_2$ for the IR (4.8±0.7 $ml.kg^{-1}.min^{-1}$) and C-R (11.7±4.7 $ml.kg^{-1}.min^{-1}$) conditions were likely due to the prior cycling exercise. The elevation in starting $\dot{V}O_2$ was also likely responsible for the reduction in $t_{1/2}$ and MRT recorded during the C-R condition. Further, the increase in the rate constant (*k*) calculated for the C-R (0.9±0.1) compared to the IR (0.5±0.0) condition suggests that the prior cycling exercise likely accelerated $\dot{V}O_2$ during the initial stages of the C-R condition and may have resulted from enhanced muscle O_2 supply (DeLorey et al., 2007), an increase in blood flow (Krustrup et al., 2001) and/or improved oxidative enzyme activity (Sahlin et al., 2005). However, regardless of the differences, overall $\dot{V}O_2$ recorded at 180 s steady state (IR = 34.8±4.0 and C-R = 35.9±3.9 $ml.kg^{-1}.min^{-1}$) and at the 10^{th} minute (IR = 34.8±4.0 and C-R = 35.9±4.2 $ml.kg^{-1}.min^{-1}$) were not significantly different. These

results are in somewhat agreement with a previous study proposing that moderate intensity prior exercise has no meaningful influence on $\dot{V}O_2$ during subsequent exercise (Gerbino et al., 1996). These authors further reported that prior moderate intensity exercise had no effect on the $\dot{V}O_2$ kinetics during heavy exercise, although prior heavy exercise did result in significantly faster $\dot{V}O_2$ kinetics during ensuing heavy intensity exercise. Other studies have described similar results, confirming that to have a 'speeding' effect on $\dot{V}O_2$ prior exercise has to be of a high intensity (Burnley et al., 2001; Burnley et al., 2000; DiMenna et al., 2008; Jones et al., 2006). Our results are contrary to these past findings however, neither of these studies employed cycle-run protocols, instead using run-run or cycle-cycle exercise bouts. For example, Hue et al. (1998) tested nine competitive triathletes and observed an elevated $\dot{V}O_2$ following the cycle-run condition compared to a run-run (R-R) trial. Further, ventilatory responses during the first eight minutes of subsequent running after cycling was significantly greater compared to the R-R trial. However, following the first eight minutes of running, $\dot{V}O_2$ values were not different between the C-R and R-R (96.3±19.8 and 89.8±13.0 $L.min^{-1}$) conditions. The latter finding is in agreement with our results and suggests that steady state $\dot{V}O_2$ was relatively unaffected despite early-phase difference between C-R and R-R or IR exercise. Likewise, previous research has reported decreases in $\dot{V}O_2$ during C-R compared with IR at 2.3% among high and elite level triathletes compared with low level triathletes who noted an increase of 3.7% (Millet et al., 2000). However, more recently no meaningful change in $\dot{V}O_2$ during subsequent running has been reported in elite international triathletes following low and high intensity cycling exercise. These results appear conflicting and are likely dependent upon the experience of individual triathletes however; elite or more highly trained triathletes are generally

least affect by prior cycling. Unexpectedly, and despite a mean elevated starting value and deficits, HR variables do not appear to be influenced by prior cycling.

Our overall findings of a lack of change in muscle recruitment activity and overall $\dot{V}O_2$ and HR variables do not support our hypothesis that muscle recruitment activity would reflect $\dot{V}O_2$ and HR during subsequent running after cycling. This is in contrast to previous research that suggests a relationship between changes in muscle recruitment activity and $\dot{V}O_2$ during exercise following prior exercise (Barker et al., 2014; Layec et al., 2009), particularly an increase in muscle recruitment activity correlated with an increase in $\dot{V}O_2$ (Burnley et al., 2002; Saunders et al., 2000). These studies all employed prior high-intensity exercise protocols and this was likely one of the major reasons behind the differences between our results and those above.

Compared to IR, following C-R, we saw no individual or group mean change in respective running stride frequency (87.0±3.2 vs. 88.5±3.0 strides/min^{-1}, respectively) or stride length (74.2±7.3 vs. 73.7±7.3 cm, respectively) during the current study. These results are contrary to those reported (Bernard et al., 2003) where an increase in stride frequency during running after cycling, most noticeably after pedalling at cadence >100 rpm was observed. Furthermore, the relationship between stride frequency and cycling cadence has been described (Hausswirth et al., 2001), emphasising a higher stride frequency during the first 500 m of running following cycling at 102 rpm. Furthermore, previous research has reported that changes in stride patterns and running velocity are found to occur specifically during the first few minutes of running after cycling (Hausswirth et al., 1997; Hue et al., 1998; Millet & Vleck, 2000; Vercruyssen et al., 2002). The absence of any change in

stride pattern in the current study may be due to participants running on a treadmill at a fixed self-selected speed, whereas over-ground running protocols where used during the aforementioned research. Although, there is evidence to support the use of treadmill-based analysis of running patterns and mechanics that can be representative of over-ground running (Fellin, Manal, & Davis, 2010; Riley, Paolini, Della Croce, Paylo, & Kerrigan, 2007)

4. A COMPARATIVE ANALYSIS OF THE MEASUREMENT OF OXYGEN UPTAKE AND HEART RATE VALUES DURING ISOLATED RUNNING AND RUNNING AFTER CYCLING

4.1 Introduction

Performing successive bouts of exercise has been reported to substantially 'speed' overall $\dot{V}O_2$ kinetics in response to repetitive bouts of exercise (Burnley, Davison, & Baker, 2011; Gerbino et al., 1996). The physiological response of speeding of $\dot{V}O_2$ kinetics is documented as increasing the amplitude of the primary $\dot{V}O_2$ component, coupled with a reduction in the $\dot{V}O_2$ slow component (Burnley et al., 2001; Burnley, Doust, & Jones, 2005). The effects of prior exercise are understood to occur in the exercising muscle (Burnley et al., 2005). Past research has proposed several physiological changes that could contribute to the speeding of $\dot{V}O_2$ kinetics including, increased blood flow (Bailey et al., 2009), oxygenation (Jones, Fulford, & Wilkerson, 2008), oxygen extraction (Bailey et al., 2009) and oxidative enzyme activity (Barker et al., 2010), none of which are critical to speeding $\dot{V}O_2$ kinetics during subsequent exercise. Whilst there is a clear understanding of the 'speeding' of $\dot{V}O_2$ after subsequent prior exercise, this only holds for exercise performed at heavy to severe intensities (above lactate threshold or supra gas exchange threshold). For intensities below the lactate threshold, prior moderate intensity exercise has no effect on the $\dot{V}O_2$ kinetics of subsequent exercise performed at heavy or moderate intensities (Gerbino et al., 1996). A similar study has suggested that prior bouts of moderate-intensity cycling exercise have no influence on the 'speeding' of $\dot{V}O_2$ kinetics in successive moderate-intensity cycling exercise among populations of either young (26 ± 1 years) or older (65 ± 2 years) adults (Scheuermann et al.,

2002). Furthermore, no ‘speeding’ effects were reported after prior running exercise on $\dot{V}O_2$ kinetics during subsequent submaximal intensity treadmill running (Jones, DiMenna, et al., 2008).

The majority of research published in reference to ‘priming’ or prior exercise on $\dot{V}O_2$ kinetics has been done during upright cycle ergometer tests or treadmill running, however, it is known that different modes of exercise can affect the characteristics of $\dot{V}O_2$ kinetics (Jones, DiMenna, et al., 2008). Previous research has demonstrated that both the early phase $\dot{V}O_2$ response and amplitude of the $\dot{V}O_2$ slow component are reduced when comparing isolated running with isolated cycling exercise (Carter et al., 2000a). Therefore, the use of prior exercise, in the format of a cycle-run protocol, would likely reflect different physiological responses, as opposed to those currently presented. Previous research focused on triathlon does use a cycle-run format however; there is a limited degree of detailed analysis of the variable physiological response during the cycle-run transition.

In triathlon, varying levels of uneasiness during the cycle-run transition phase have been reported among triathletes of all levels, and this reported uneasiness appears to reflect an increased overall oxygen cost (Bentley et al., 2002; Millet & Vleck, 2000) and heart rate (HR) (Kreider, Boone, Thompson, Burkes, & Cortes, 1988) (Bonacci, Green, et al., 2010; Hausswirth et al., 1997) during the cycle-run transition period of triathlon. Oxygen kinetics following the onset of moderate intensity exercise, are considered to follow an exponential time course and reach steady state within 3-min in healthy subjects (Pringle et al., 2003). This is true when comparing run-run or cycle-cycle prior exercise, although, this may not be the case when looking at cycle-

run priming. Hue et al. (1998) has previously reported that steady state $\dot{V}O_2$ and oxygen cost varied between a control run and transition (cycle-run) run. These authors suggested that a difference in time to steady state $\dot{V}O_2$ and overall oxygen cost during the cycle-run phase, compared to the control run (3 min and 2 min, respectively) was indicative of an oxygen deficit during the cycle-run transition and that this prolongation was due to cycling prior to running, as opposed to control running. These results confirm that during subsequent running, prior cycling negatively impacts running economy through an increase in oxygen cost (Hausswirth et al., 1997). Furthermore, when comparing mean isolated running time and velocity (980 s and 18.2 km/h) with mean running time and velocity during triathlon running (1014 s and 17.4 km/h) the results confirm the negative affect prior cycling has on running performance (Bernard et al., 2003). Additionally, laboratory data similarly indicates increases in ventilation rate and HR during triathlon running (running after cycling) at submaximal intensities (Guezennec et al., 1996; Hausswirth et al., 1996; Millet & Vleck, 2000).

These results suggest that prior cycling does have a significant physiological impact on subsequent running. However, the previous research has not detailed the variable $\dot{V}O_2$ response during cycle-run transition in comparison to isolated running. The literature suggests that the ability to respond to the cycle-run transition is crucial to running performance during triathlon yet the lack of detailed analysis of physiological response during this period is surprising. As such, the rate at which $\dot{V}O_2$ rises after the onset of exercise in relation to varying intensity is likely to impact oxygen values and time to steady state when comparing isolated with cycle-run transition, and may provide a reasonable explanation of previously reported levels of uneasiness during this period. Similarly, HR kinetics during the same period has

been scarcely reported. Therefore, considering that rate constants for HR and $\dot{V}O_2$ have demonstrated a relatively linear correlation after exercise onset to steady state values during submaximal exercise (Kay, Ashar, Bubien, & Dailey, 1995), it may be expected that comparable $\dot{V}O_2$ and HR physiological response would likely occur in response to running after prior cycling exercise.

The purpose of this study was to investigate if $\dot{V}O_2$ and HR variables evolve differently during the early-phase of running after prior cycling exercise as compared to isolated running. Our hypotheses are stated in section 1.8 under 'Aims and Hypotheses'.

4.2 Methods

4.2.1 Participants

Eleven (8 males and 3 females; *n*=11) healthy competitive triathletes were recruited to participate in this study. All participants had experienced Australian National level and/or International Triathlon Union (ITU) level competition (4.7±1.4 years) and had gained this experience in at least the year preceding testing. Table 4.1 outlines participant training history. All participants filled out a self-screening physical activity readiness questionnaire (PAR-Q) that was used to determine the level of safety and possible risk of exercise testing for the individual based upon their answers to specific health related and previous exercise history questions. Participants were also provided with an information package that described in detail all the procedures and requirements of the exercise testing. Prior to testing all participants provided written informed consent acknowledging their understanding of the procedures, requirements, associated risks, outcomes and any future publication

of the research they were to participate in. On the day of testing verbal consent was also given by all participants prior to taking part in the experimental process. Anthropometric measurements were recorded on the day, prior to testing, in accordance with procedures outlined by the International Society for the Advancement of Kinanthropometry (ISAK) (Table 3.2). In order to preserve confidentiality and anonymity; participants were allocated a numerical code. All procedures employed in this study were approved by the University of Wollongong Human Research Ethics Committee in accordance with the *National Statement on Ethical Conduct in Human Research* (HE12/331).

Table 4.1: Participant weekly training profiles (mean ± SD).

Age (years)		25.3 ± 6.9
Experience (years)[a]		4.7 ± 1.4
Training hours (h)[b]		23 ± 6
Training distance (km)[c]		
	cycling	293 ± 102
	running	60 ± 25
Sessions[d]		
	cycling	4 ± 1
	running	6 ± 2
	cycle-run	1.1 ± 0.3

[a] Years of triathlon experience at National or International level.
[b] Mean training hours recorded per week in the three months preceding testing.
[c] Mean training distance (km) recorded per week in the three months preceding testing.
[d] Mean number of training sessions recorded per week in the three months preceding testing.

4.2.2 Anthropometric Measurement

Anthropometric data was recorded and group mean values calculated for a total of eleven (8 males and 3 females; n=11) and presented in table 4.2.

Table 4.2: Participant anthropometric profiles (*mean* ± *SD*).

Height (cm)		178.8 ± 7.3
Mass (kg^{-1})		69.5 ± 7.9
Leg length[a]	*R*	95.3 ± 4.6
	L	95.4 ± 4.9
Knee width[a]	*R*	10.0 ± 0.7
	L	10.0 ± 0.6
Ankle width[a]	*R*	7.5 ± 0.6
	L	7.4 ± 0.7
Elbow width[a]	*R*	7.6 ± 1.1
	L	7.5 ± 1.0
Wrist width[a]	*R*	5.7 ± 0.9
	L	5.7 ± 0.9
Hand thickness[a]	*R*	3.0 ± 1.0
	L	3.0 ± 0.9

[a] Mean value of two recorded measures from the same anthropometric site.
R Denotes the mean recorded value from the right side.
L Denotes the mean recorded value from the left side.

4.2.3 Measured Oxygen Uptake

Pulmonary oxygen uptake was measured according to the procedures outlined under section 2.3.3 ‘Measurement of Pulmonary Oxygen Uptake’.

4.2.4 Measured Heart Rate

Heart rate was measured according to the procedures outlined under section 2.3.4 ‘Measurement of Heart Rate’.

4.3 Statistical Analysis

4.3.1 Oxygen Uptake

Mean values for IR and C-R net $\dot{V}O_2$ data were compared using paired *t*-tests. Similarly, individual $t_{1/2}$ and MRT $\dot{V}O_2$ values were compared and differences reported as percentage variance (%*V*) where a difference >10% of the IR is considered significant. Differences between group mean results for steady state, $t_{1/2}$ and MRT $\dot{V}O_2$ during the IR and C-R conditions were compared using paired *t*-tests. Pearson's correlation of coefficients (*r*) was used to identify similarities between $t_{1/2}$ and MRT $\dot{V}O_2$ of respective conditions. Rate constant (*k*) values were compared using paired *t*-tests and individual difference between *k* of the exercising conditions was represented as percentage of variation.

4.3.2 Heart Rate

Paired *t*-tests were used to identify significant differences between group mean HR for steady state, $t_{1/2}$ and MRT variables for the IR and C-R conditions. Pearson's correlation coefficient (*r*) was used to determine the relationship between $t_{1/2}$ HR values during the IR and C-R conditions. Similarly, HR MRT was correlated between respective running conditions. Differences between individual HR values during the IR and C-R conditions were reported as percentage change.

4.3.3 Paired Oxygen uptake and Heart Rate Analysis

Pearson's correlation coefficient (r) was performed to determine the relationship between $\dot{V}O_2$ and HR $t_{1/2}$ and MRT values during the respective IR and C-R trials.

Descriptive data are reported as means with standard deviation (± SD). Group mean values are presented in the figures along with typical individual data where indicated, with error bars representing the standard error of the mean (SEM). Statistical analysis was carried out using IBM SPSS 21 (IBM Corporation, Armonk, NY), with statistical significance set at $p<0.05$, unless stated otherwise.

4.4 Results

4.4.1 Oxygen Uptake during Isolated vs. Primed Cycle-Running

Group mean (n =11) IR and C-R Borg 6-20 rating of perceived exertion scores were 13.8±0.4 and 12.9±1.0, respectively. Self-selected group mean running speed across the IR and C-R was 11.6±0.9 km/h. The group mean net $\dot{V}O_2$ response variables collected during the IR and C-R exercise bouts are presented in Tables 4.3. Starting $\dot{V}O_2$ for all individuals were higher at the onset of the C-R condition compared with the IR condition ($p<0.01$), with a group mean difference of 6.6 $ml.kg^{-1}min^{-1}$ between conditions. Individual and group mean values were not different between conditions for 10^{th} min $\dot{V}O_2$ (r=0.99; p=0.38) and steady state (r=0.91; p=0.40), despite the raised metabolic start point and prior exercise completed during the C-R. However, all individuals demonstrated greater ΔO_2 during the C-R condition, this reflects the difference ($p<0.01$) between the group mean values for the exercising conditions. The difference in ΔO_2 is likely due to the difference in metabolic starting points between conditions.

Compared with the IR bout, $\dot{V}O_2$ for the primed C-R exercise resulted in a significantly elevated starting ($p<0.01$) and 60 s ($p<0.01$) $\dot{V}O_2$ value. The elevated starting $\dot{V}O_2$ reflects a substantially reduced mean $t_{1/2}$ value for the C-R compared to

the IR (50.81 and 62.91 s, respectively; p=0.04). Similarly, the average $\Delta \dot{V}O_2$ to $t_{1/2}$ was also significantly reduced during the C-R compared with the IR (p<0.01). Furthermore, a 25% decrease in the rate $\Delta \dot{V}O_2$ was evident for the C-R, compared with the IR (0.18 and 0.24 $ml.kg^{-1}min^{-1}$, respectively; p=0.03). Interestingly, and regardless of the significant difference in total $\dot{V}O_2$ for the respective conditions (IR 29.5 and C-R 21.7 $ml.min.kg^{-1}$; p<0.01), the average $\dot{V}O_2$ steady state values did not differ between the IR and C-R (34.6 and 35.2 $ml.kg^{-1}min^{-1}$, respectively; p=0.44).

Table 4.3: Individual and mean net $\dot{V}O_2$ variable values collected during the IR and C-R conditions.

Participant		1	2	3	4	5	6	7	8	9	10	11	Mean ± SD
VO_2: IR vs. C-R													
Starting VO_2	IR	9.9	6.1	6.3	4.0	4.9	4.9	6.0	4.2	5.1	6.0	8.0	5.9 ± 1.7
	C-R	13.2	13.6	15.3	9.4	19.3	11.1	6.6	8.9	15.2	7.4	18.3	12.5* ± 4.3
10th min VO_2	IR	31.0	38.1	39.3	38.2	39.7	29.7	34.4	33.8	33.6	25.0	38.4	34.7 ± 4.7
	C-R	28.9	39.0	39.2	36.7	39.4	28.3	39.8	35.9	35.1	25.3	40.8	35.3 ± 5.4
SS	IR	30.3	37.7	39.5	39.6	38.7	28.6	34.4	33.5	34.3	25.2	39.2	34.6 ± 4.9
	C-R	29.2	38.3	39.3	37.1	40.1	29.5	40.5	34.7	33.6	25.5	39.4	35.2 ± 5.1
ΔO_2	IR	1344.3	2506.7	2213.1	2541.9	2177.6	1318.1	2011.4	2077.8	2125.2	1036.2	2311.0	1969.4 ± 505.9
	C-R	947.5	1790.5	1601.5	1639.4	1578.8	945.5	1782.0	1456.5	1535.4	1081.9	1428.2	1435.2* ± 308.1

Note: * denotes significant difference between IR and C-R values, $p<0.05$. All variables units are presented as $ml.kg^{-1}min^{-1}$.

Individual calculated variables for the IR and C-R conditions are presented in Table 4.4. Halftime values were considered significantly faster for the majority (n=9) of participants during the C-R, compared to the IR, for individuals between conditions and group mean results (p<0.01). Mean response time for individual participants showed a significantly quicker $\dot{V}O_2$ response following the C-R exercise (p<0.01), compared to the IR, with all participants demonstrating at least a 40.9% decrease in MRT during the C-R condition. Pearson's coefficient revealed no correlation between either $t_{1/2}$ or MRT for the IR and C-R bouts. Oxygen deficit values were also considered for each individual between the IR and C-R conditions (mean difference=58.4±9.6%). Difference was also reported for group mean O_2 deficit values (p<0.01). Group mean k values were faster for the C-R (p<0.01) compared to the IR condition. These differences in k values suggest a quicker increase in $\dot{V}O_2$ during the C-R with mean individual k difference between the IR and C-R conditions reported as 0.41±0.1.

Table 4.4: Shows individual calculated $\dot{V}O_2$ variables for the IR and C-R exercise conditions.

Participant		1	2	3	4	5	6	7	8	9	10	11	Mean ± SD
VO_2: IR vs. C-R													
$t_{1/2}$ (s)	IR	41.4	57.2	57.7	69.3	52.6	76.7	62.5	75.6	71.4	71.8	65.8	63.8 ± 10.8
	C-R	43.1	37.8	51.9	48.7	46.1	60.5	52.4	46.2	46.4	56.3	69.4	50.8* ± 8.8
MRT (s)	IR	115.5	107.2	135.0	135.4	133.2	113.9	127.5	126.5	126.3	102.5	132.2	123.2 ± 11.6
	C-R	68.3	58.4	44.9	69.7	45.8	44.5	75.3	44.4	62.6	47.0	59.1	56.4* ± 11.6
O_2 Deficit	IR	2587.9	4480.5	4979.8	5737.2	4836.0	2501.2	4272.7	4379.8	4472.1	1770.0	5091.9	4100.8 ± 1250.7
	C-R	1078.6	1813.4	1524.6	2197.0	1613.3	1184.6	2593.2	1364.7	1785.9	987.6	1933.1	1643.3* ± 489.3
k	IR	0.5	0.6	0.4	0.4	0.5	0.5	0.5	0.5	0.5	0.6	0.5	0.49 ± 0.1
	C-R	0.9	1.0	1.1	0.7	1.0	0.8	0.7	1.1	0.9	1.1	0.7	0.90* ± 0.1
k diff		0.4	0.4	0.6	0.3	0.5	0.3	0.2	0.6	0.4	0.5	0.3	0.41 ± 0.1

Note: * denotes significance between the IR and C-R conditions, $p<0.05$. O_2 deficit values are presented in $ml.kg^{-1}min^{-1}$.

4.4.2 Heart Rate during Isolated vs. Primed Cycle-Running

Standard HR variables for each individual collected during the IR and C-R conditions are displayed in Table 4.5, along with population mean (±SD). Starting HR values were vastly different between conditions ($P<0.01$), with all participants recording higher starting HR values at the onset of the C-R condition. Individual values for ΔHR showed a mean difference of 28±15 bpm, with the majority of participants demonstrating a clear reduction during the C-R condition, with population mean ΔHR from starting to steady state values exhibiting a significant level of difference ($p<0.01$). Individual steady state and 10^{th} min HR values showed only small margins of variation between conditions, with group mean results not showing any significant difference between the measure IR and C-R bouts. Instead correlation mean steady state and 10^{th} min HR demonstrated high levels of correlation ($r=0.97$ and 0.97, respectively).

Table 4.5: Individual and mean HR variables collected during IR and C-R conditions.

Participant		1	2	3	4	5	6	7	8	9	10	11	Mean ± SD
HR: IR vs. C-R													
Starting HR	IR	72	57	63	53	68	80	65	65	77	77	70	68 ± 8
	C-R	79	103	91	114	98	110	112	79	121	111	99	102* ± 13
10th min HR	IR	136	122	120	151	136	156	135	129	159	147	124	138 ± 14
	C-R	142	135	132	158	143	162	143	133	168	158	125	145 ± 14
SS	IR	136	127	129	152	136	155	135	129	159	147	123	139 ± 12
	C-R	144	131	131	157	142	162	143	130	167	163	125	145 ± 15
Δ HR	IR	64	70	66	99	68	75	70	64	82	70	53	71 ± 12
	C-R	65	28	40	43	44	52	31	51	46	52	26	44* ± 12

Note: Group mean (±SD) values are presented as bpm. * Denotes statistical significance set as $p<0.05$.

Individually calculated HR variables are presented in Table 4.6. Mean HR $t_{1/2}$ values were not different between conditions. However, individual $t_{1/2}$ did show varying levels of difference between conditions, particularly for participant two, who demonstrated a 37.1 s difference between the IR and C-R recordings. In contrast, participants one and eight displayed less than a second difference between individual $t_{1/2}$. Population MRT did demonstrate a difference when comparing average IR and C-R MRTs (p=0.04). The range of individual variance for MRT was considered high (7.3–48.8 s), with MRT's usually greater during the C-R condition. Variance within-individual HR deficit was also considered high, ranging between 5–105 bpm between conditions and represented a significant population mean difference during the C-R when compared with the IR (p<0.05). Rate constant for the group was lower during the C-R, compared with the IR, however not statistically different.

Table 4.6: Calculated HR variables for individual participants and population mean for IR and C-R conditions.

Participant		1	2	3	4	5	6	7	8	9	10	11	Mean ± SD
HR: IR vs. C-R													
$t_{1/2}$ (s)	IR	29.3	31.5	34.8	35.8	35.5	63.3	40.1	40.4	47.8	57.1	49.6	42.3 ± 10.9
	C-R	30.2	68.6	31.5	54.6	58.2	40.0	51.9	41.1	44.5	41.5	42.3	45.9 ± 11.5
MRT (s)	IR	77.4	58.3	80.5	93.9	83.4	66.4	86.2	46.8	99.9	58.3	56.1	73.4 ± 17.2
	C-R	110.7	82.7	87.8	70.2	104.0	101.2	69.1	95.4	86.0	107.0	47.3	87.4* ± 19.3
HR Deficit	IR	83	68	89	155	95	83	101	50	137	68	50	89 ± 33
	C-R	120	39	59	50	76	88	36	81	66	93	20	66* ± 29
k	IR	0.8	1.0	0.7	0.6	0.7	0.9	0.7	1.3	0.6	1.0	1.1	0.86 ± 0.2
	C-R	0.5	0.7	0.7	0.9	0.6	0.6	0.9	0.6	0.7	0.6	1.3	0.73 ± 0.2
k diff		0.2	0.3	0.1	-0.2	0.1	0.3	-0.2	0.7	-0.1	0.5	-0.2	0.14 ± 0.3

Note: Group mean (±SD) are also presented. * Denotes statistical significance set as $p<0.05$.

4.4.3 Oxygen Uptake and Heart Rate Relationship during Isolated vs. Primed Cycle-Running

Correlation between IR $t_{1/2}$ values for $\dot{V}O_2$ and HR kinetics (Fig 4.1), demonstrated a significant level of correlation (r=0.74; p<0.01) lending support the use of the single-phase model to reasonably predict $t_{1/2}$ for $\dot{V}O_2$ and HR during submaximal isolated running. However, there was no significant correlation between $t_{1/2}$ values for $\dot{V}O_2$ and HR kinetics during the C-R (r= -0.37), suggesting that primed exercise may influence the rate increase for $\dot{V}O_2$ and HR during subsequent exercise, particularly if that exercise is different to the preceding bout. Mean response time correlations for both IR and C-R $\dot{V}O_2$ and HR $t_{1/2}$ did not show any meaningful correlations. Mean response time correlations for the C-R condition (Fig 4.2) do clearly demonstrate the significant reduction in $\dot{V}O_2$ MRT, while the increase to HR MRT is also depicted.

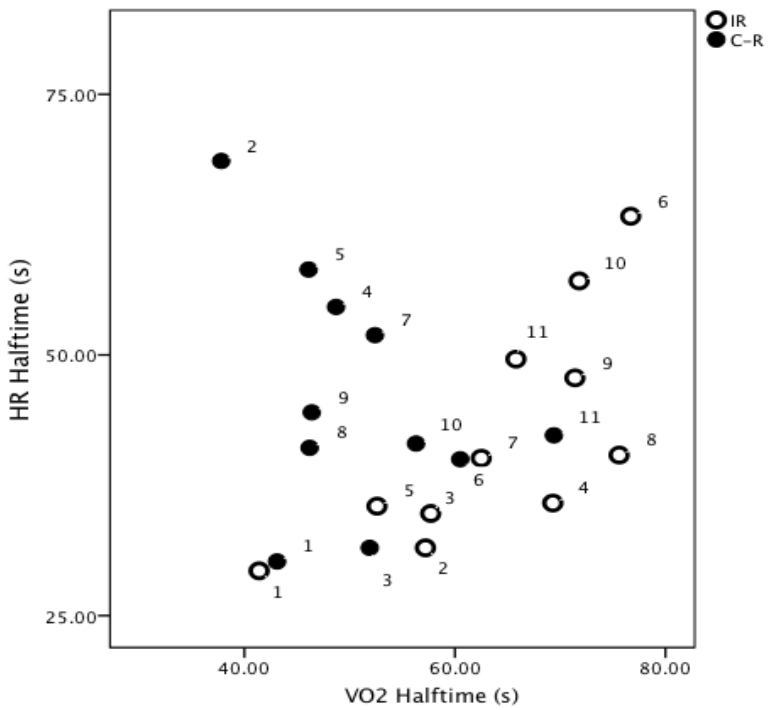

Figure 4.1: Individually correlated overlay of $\dot{V}O_2$ and HR $t_{1/2}$ values.

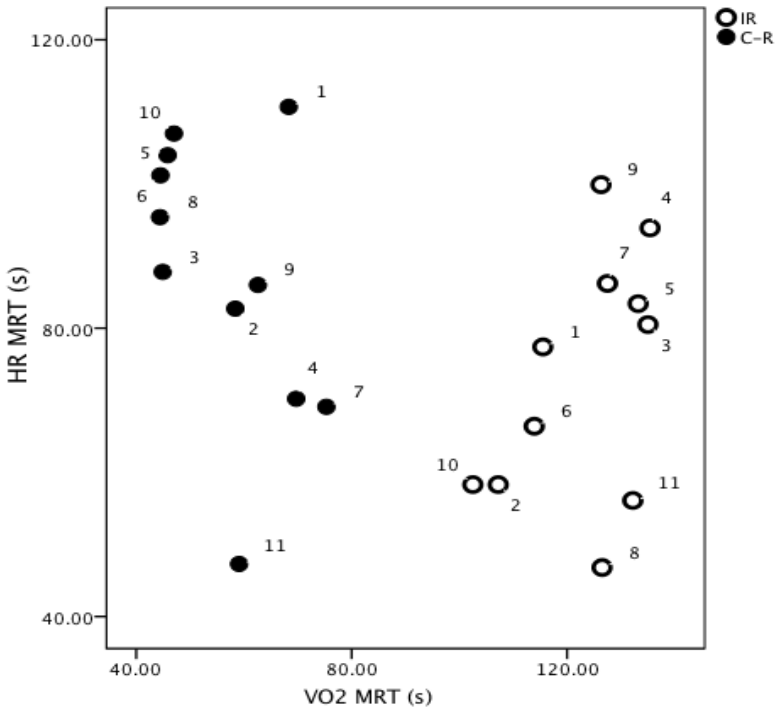

Figure 4.2: Individually correlated overlay of $\dot{V}O_2$ and HR MRT values.

4.5 Discussion

4.5.1 Outcomes

To our knowledge, this is the first study to quantify changes in $\dot{V}O_2$ and HR using a single-phase logit-log model to calculate $t_{1/2}$ and paired with MRT to analyse physiological responses at the onset of submaximal isolated and cycling-primed running in a population of trained athletes. We have shown that differences exist between $\dot{V}O_2$ and HR kinetics when comparing the early stages of isolated versus cycling-primed running at a moderate intensity. The major finding of this study that runs contrary to our hypothesis was that individual $t_{1/2}$ and MRT values appear to respond at a faster rate and independently during running following prior cycling exercise, compared with isolated running.

The differences in average $\dot{V}O_2$ $t_{1/2}$ across the two running conditions are suggestive of a differing speed of metabolic response, being faster during the C-R. Similarly, variations in MRT values across both running conditions were very evident. All individuals recording $\dot{V}O_2$ MRT values almost twice as fast during the C-R transition, as opposed to the IR condition and these differences were reflected in the group mean values. The fact that $\dot{V}O_2$ variables during the C-R condition responded at significantly faster rates are likely caused by the difference in starting $\dot{V}O_2$ values. All participants' recorded higher starting $\dot{V}O_2$ values at the beginning of running during the C-R condition, compared to the IR condition. This disparity between conditional starting $\dot{V}O_2$ values indicates that $\dot{V}O_2$ kinetics had not recovered to a comparative level following the end of the cycling bout and the beginning of the run during the C-R condition and are likely to affect the rate of $\dot{V}O_2$ during subsequent

running. These findings support previous research stating that prior exercise can 'speed' oxygen kinetics at the onset of subsequent exercise (Burnley et al., 2002; Burnley et al., 2005; Gerbino et al., 1996; Jones et al., 2006) where elevated starting $\dot{V}O_2$ values are likely. However, the 'speeding' of $\dot{V}O_2$ following prior exercise generally pertains to high intensity exercise. Previous research looking at similar affects using moderate or intensities below the lactate threshold suggest an absence of any $\dot{V}O_2$ 'speeding' (Hughson & Morrissey, 1982). However, with respect to triathlon, research has shown that cardiorespiratory variables do fluctuate when comparing isolated running with cycle-run exercise (Hue et al., 1998). These authors reported that $\dot{V}O_2$, minute volume and breathing frequency are all higher during the cycle-run transition when compared with isolated running under submaximal ($\%\dot{V}O_{2max}$ 78.1±4.9 $ml.kg^{-1}min^{-1}$) intensity. Additionally, these same authors looked at the differences in pulmonary ventilatory responses during cycle-run and run-run transitions. Overall, pulmonary values did not differ, however, during the first 10 min of subsequent running, the cycle-run condition demonstrated significantly higher values for $\dot{V}_E$ and breathing frequency. The authors therefore stated that higher levels of pulmonary stress were purely the result of prior cycling. Analogous results were further outlined by Bonacci et al. (2010), who showed that during control and cycle-run transition the effects of cycling either impaired or improved the overall $\dot{V}O_2$ values among a cohort of moderately trained individual athletes. Conversely, there is evidence to suggest that prior cycling at either low or high intensity has no subsequent impact on running economy or cost in elite international triathletes (Bonacci et al., 2011). The major difference between our results and those previously are that most studies report $\dot{V}O_2$ values as only group mean values or values recorded at arbitrary time points that generally fall outside the defined cycle-run transition

period or are selected based upon minimal rational. Our transition results were recorded during the three minutes of exercise, and based upon calculations using individual physiological values, therefore providing a greater level of detail of the underlying physiology of the cycle-run transition than that previously reported.

The faster individual and group mean $t_{1/2}$ and MRT values recorded during the C-R condition, compared with the IR condition are reflective of the individual difference in rate constant (*k*) values. For all individuals *k* was faster during the C-R condition, in comparison to the IR (0.90±0.1 min and 0.49±0.1 min, respectively), indicating the greater relative effectiveness of the cardiopulmonary and metabolic to respond (Whipp, 1971) following prior exercise as compared to isolated. Furthermore, the mean difference in *k* (0.41±0.1 min) highlights that the exponential metabolic rise was significantly faster during the C-R condition, indicating that the prior cycling exercise likely led to the 'speeding' or faster rise of $\dot{V}O_2$. The principal effect of prior exercise is to 'speed' $\dot{V}O_2$, illustrated by a reduced MRT, during subsequent exercise (Burnley et al., 2000; Gerbino et al., 1996; Jones, DiMenna, et al., 2008; Koppo & Bouckaert, 2000), that was evident in our results. However, the 'speeding' of $\dot{V}O_2$ is dependent upon preceding exercise being sufficiently intense to result in metabolic acidosis (Burnley et al., 2000; Fukuba et al., 2004; Gerbino et al., 1996; Koppo & Bouckaert, 2000) that enhances muscle blood flow and oxygenation of the working muscles (Burnley et al., 2000; Krustrup et al., 2001). Therefore, considering the faster response in all participants during the C-R condition, there is potential that individuals did achieve levels of acidosis and muscle blood flow and oxygenation were enhanced by the preceding cycling at what was considered moderate intensity exercise. In agreement, Di Prampero et al. (1970) reported faster pulmonary kinetics

during exercise following moderate-intensity prior exercise as opposed to exercise completed from rest.

Similarly reported is that steady state levels of $\dot{V}O_2$ are achieved sooner during transition running (from cycling to running), in comparison to isolated running (1 min and 2 min, respectively) among a group of trained male triathletes (Hue et al., 1998). These results further suggest a faster metabolic response during C-R exercising, despite these investigators using similar moderate levels of intensity for both C-R and IR conditions. In addition, these authors also showed that overall mean $\dot{V}O_2$ at steady state were considerably higher following the C-R condition as opposed to IR (51.7 ± 3.9 and 48.3 ± 1.4 $ml.kg^{-1}min^{-1}$, respectively), indicating an increased running cost during C-R exercise. This increase in the cost of running following cycling is well documented (Guezennec et al., 1996; Hausswirth et al., 1997; Hue, Le Gallais, Boussana, Chollet, & Prefaut, 1999; Hue et al., 1998; Millet et al., 2000), although speculation is still widespread as to the exact source of this increase. In contrast, our results show that despite prior cycling and the case of the 'speeding' in $\dot{V}O_2$, mean steady state levels of oxygen consumption were not different between the IR and C-R conditions at either three minutes (34.6±4.9 and 35.2±5.1 $ml.kg^{-1}min^{-1}$) or ten minutes (34.7±4.7 and 35.3±5.4 $ml.kg^{-1}min^{-1}$) recordings. Furthermore, the greatest individual difference in steady state oxygen consumption between conditions was only 6.1 and 5.4 $ml.kg^{-1}min^{-1}$ recorded at the 3^{rd} and 10^{th} minutes respectively. Moreover, results for the change in oxygen and the oxygen deficit show that consumption and demand of oxygen during the early stages of the IR were significantly greater than during the C-R and paired with a lower metabolic starting point suggests that achieving steady state during the IR condition would take a

considerably longer amount of time, when compared with the C-R condition. That said, by the 3rd minute of recording all participants for both conditions had reached a level of steady state.

Heart rate at the onset of exercise was significantly elevated above IR values in all participants during the C-R condition (68±8 and 102±13 bpm, respectively) and remained consistently higher until steady state, recorded at the 3rd minute after exercise onset, was achieved. The subsequently higher HR starting point recorded during the C-R bout likely reflects the difference in the change in HR to steady state between the IR and C-R conditions. Apart for one participant; all individuals presented reduced changes in HR during the C-R, compared to the IR bout. Similar starting HR values have been reported (Bailey et al., 2009). These authors showed that compared with control (isolated) exercise, starting HR was always significantly higher following prior exercise. Similar results were reported for HR values measured at 60 and 120 s. Calculated HR kinetic values for $t_{1/2}$, MRT and *k* demonstrated considerable levels of individual variability during the current study. Mean $t_{1/2}$ values were similar for both conditions however; mean MRT was significantly faster during the IR condition, suggestive of a more rapid exponential increase in HR during the control (IR) bout. This appears to be somewhat confirmed by the difference in *k*. Group mean *k* were not significantly different although, individual *k* were consistently higher during the IR in comparison to the C-R (0.86±0.2 vs. 0.73±0.2, respectively). These results advocate that the speed of HR kinetics at the onset of isolated exercise, as opposed to the C-R exercise were faster. Comparable results have indicated a slowing of HR kinetics when transitioning to moderate-intensity, opposed to isolated exercise from a significantly elevated

baseline HR (MacPhee, Shoemaker, Paterson, & Kowalchuk, 2005). Moreover, invariant HR time constants have been identified when transitioning to severe-intensity cycling from differing baseline HR's (DiMenna et al., 2008). The differing change in HR following exercise onset during IR and C-R in this present study could not be established. For trained individuals, the rate of increase in HR at exercise onset is faster than that of untrained individuals (Nelson et al., 2013), as the acceleration in HR during the early stages (fast component) of exercise is predominantly mediated by the withdrawal of parasympathetic/vagal activity (Fagraeus & Linnarsson, 1976; Hettinga et al., 2014; Maciel, Gallo, Marin Neto, Lima Filho, & Martins, 1986). Following this, HR acceleration is driven by an increase in sympathetic activity, with the magnitude of the HR increase being reflective of the sympathetic response (Hettinga et al., 2014). In conjunction, previous research has speculated that fitter or trained participants have greater parasympathetic tone at rest, resulting in lower resting HR, and/or stronger vagal withdrawal at exercise onset (Berne, 1977; Jagoda et al., 2014; Ricardo, Silva, Vianna, & Araújo, 2010). Therefore, the faster HR kinetics seen during the IR, compared with the C-R condition, may result from lacking the fast component that would negate the parasympathetic influence on HR acceleration due to the absence of any prior exercise. However, under higher exercise intensities, HR acceleration is less rapid due to a greater contribution of the slow component (Hettinga et al., 2014) consequently; prior exercise may influence the intensity of subsequent exercise resulting in a slower rise in HR during the C-R condition. This idea is in agreement with Bearden et al. (2001), whose results showed that repeated bouts of high-intensity exercise consistently slow HR kinetics via the elevated baseline work rate that in turn removes the rapid influence of the parasympathetic system. Likewise, the

variability in HR acceleration during the early stages of exercise in this study are in line with those reported for a cohort of fit and healthy individuals (Jagoda et al., 2014). Furthermore, group mean results showed no meaningful difference between steady state HR values recorded at the 3rd and 10th minute of running during the IR and C-R conditions. These results are similar to those previously reported (Bailey et al., 2009) where end exercise, being considered steady state, HR was no different between control values and prior exercise values. However, HR has previously been reported as significantly higher at assumed steady state following running after cycling as opposed to control (isolated) running (Guezennec et al., 1996; Kreider et al., 1988).

It has been well established that HR and $\dot{V}O_2$ are linearly related over a wide variety of exercise intensities and HR has been used to estimate $\dot{V}O_2$ (Achten & Jeukendrup, 2003). Correlations between $t_{1/2}$ and MRT values were used to determine potential differences in the linear relationship of HR and $\dot{V}O_2$ during the IR and C-R conditions. Group mean correlations of $t_{1/2}$ showed a meaningful linear relationship (r=0.74) between HR and $\dot{V}O_2$ during the IR, confirming past results. However, no such relationship was evident during the C-R condition. This result suggests that prior exercise can affect the relationship between HR and $\dot{V}O_2$ when performing cycle-run exercise at moderate intensity. Similar results have shown that transition between repeat heavy exercise bouts can dissociate the HR and $\dot{V}O_2$ kinetic relationship (Bearden & Moffatt, 2001). These authors stated that the separation was due to the elevated metabolic starting baseline (35% $\dot{V}O_{2max}$), resultant from prior moderate exercise.

5. RESEARCH SUMMARY

5.1 Conclusions

The results of this study show that compared to isolated running, prior moderate intensity cycling does not considerably influence muscle recruitment patterns or the overall metabolic cost of subsequent running in elite triathletes. The adherence of relative stable muscle recruitment patterns appears to stem from the ability of experienced, trained triathletes to limit the negative effects of prior cycling. As has previously been stated (Bonacci et al., 2011) this unique ability may be one of several factors that contribute to the success of such athletes and therefore identifying such an ability in athletes may help to distinguish those who are likely to be successful in the sport.

Additionally, $\dot{V}O_2$ and HR variables at steady state during subsequent running do not appear significantly affected by prior cycling. However, we were able to determine a substantial difference in the initial rises in $\dot{V}O_2$ and HR during isolated and cycling-primed running to submaximal steady state, as demonstrated by individual *k* values. The difference in *k* values observed during the cycle-run condition shows that prior cycling is likely to influence the physiological response during the early phase of subsequent running. Prior cycling appears to accelerate $\dot{V}O_2$ during subsequent running that may provide some benefit in limiting metabolic cost during the cycle-run transition. Furthermore, following prior exercise the often cited exponential linear relationship between $\dot{V}O_2$ and HR appears to disappear, with these variables achieving steady state independently of each other, yet still within three minutes of exercise onset. We interpret these data to indicate that prior moderate intensity

cycling, does not meaningfully limit performance of subsequent running, either be way of alterations to muscle recruitment patterns or increases to the metabolic cost.

5.2 Limitations

A number of limitations of this study should be considered, the first being the small sample size of athletes. This was necessary to ensure participant homogeneity, also only athletes considered highly trained were recruited for this study, of which there are only small numbers and the inclusion of additional participants could have potentially diluted the results of a highly trained population. Secondly, running trials were conducted at constant speeds on indoor treadmills. We acknowledge that this does not reflect competitive demands of the athletes, however it was necessary to control running speed as muscle recruitment and physiological variables can vary with running speeds, further, running at a greater speed would require a more cautious interpretation of results (Bonacci et al., 2011). Additionally, the running speeds of participants were slower than what would be expected during competition, however there is evidence to suggest that physiological costs measured at moderate running speeds can predict performance at faster speeds (Saunders, Pyne, Telford, & Hawley, 2004). Also, previous findings suggest that testing of submaximal exercise parameters measured at variable workloads (i.e. running speed) have been reported to provide a better indication of endurance performance among triathletes (Suriano & Bishop, 2010). Moreover, running at higher speeds, such as at competition pace, may have seen a delay in attainment of steady state or led to $\dot{V}O_2$ kinetic drift to peak $\dot{V}O_2$ (Xu & Rhodes, 1999). Achieving steady state for each participant was imperative for this study, therefore; individually self-selected moderate running speeds were preferred to prescribing set running speeds. Similarly, the interpretation of results

relies heavily on the relative exercise intensity of the testing protocol and considering all exercise bouts were completed at moderate intensities it is reasonable to suppose that any muscle recruitment or physiological alterations would be exaggerated under higher exercise intensities and would more closely reflective competition demands. In addition, exercise duration completed during this research is not a replication of that undertake by the majority of tested participants. However, it has been cited that the most confounding methodological issue in the literature looking at the impact of prior cycling on running is the variation in testing protocols (Bentley et al., 2002). It is acknowledged that differing exercise protocols may potentially influence the metabolic and/or neuromuscular responses of individual participants, therefore making conclusions from the data of differing performances when running after cycling, compared with isolated running, difficult to establish. However, the suitability of testing participants over the respective exercise durations likely to be completed during competition could be viewed as unfeasible. Particularly with previous research demonstrating that high level triathletes will either adapt to running after cycling within the early phase of transitioning or, alternatively cycling influenced changes will likely persist for the during of the run segment (Bonacci, Blanch, et al., 2010; Bonacci et al., 2009; Chapman et al., 2010). This suggests that replicating the full length exercise duration of cycling and running completed during a triathlon event (Olympic distance in this case) may not be necessary to illicit the conditions of the cycle-run transition period. Furthermore, the literature does contain myriad of results with similar findings despite the use of differing exercise protocols (Guezennec et al., 1996; Hausswirth et al., 1996; Kreider et al., 1988). In particular, previous research has allowed for self-selected running speeds during testing protocols, in an attempt to replicate ‘surging’ during race conditions (Hausswirth,

Lehnaff, Drano, & Savonen, 1999; Kreider et al., 1988). In similarity, the exercise protocol used for this research employed a self-selected running speed based upon the participant's perceived effort, which is prescribed by the authors of the current implemented peer-reviewed protocol (Chapman et al., 2009).

With respect to the EMG analysis, measuring muscle recruitment patterns of the left limb only may have resulted in potential cycling-induced alterations of the right limb being overlooked. Lastly, the absence of a run-run protocol limited our capacity to compare $\dot{V}O_2$ and HR response of a run-run protocol against a cycle-run protocol. This may have enabled us to more distinctively identify any differences between the physiological responses of single-discipline (run-run) exercise with that of double-discipline (cycle-run) exercise.

5.3 Practical Implications

These results provide coaches and scientists with literature that suggests that running patterns of trained triathletes, competitive at national or international level, are not significantly influenced by prior cycling at moderate intensity. However, there was evidence to suggest an increase in variability to muscle recruitment activity when running after cycling. Therefore, training strategies aimed at minimising variation in muscle recruitment activity when running after cycling may benefit some triathletes' performance. The faster mean *k* value observed during the C-R condition provides an alternative view to those of previous studies, that prior cycling may provide some benefit to the physiological response (i.e. accelerated $\dot{V}O_2$ leading to earlier achievement of steady state that potentially minimises metabolic cost) during the early phase of subsequent running.

Furthermore, our results provide an alternate means of analysing and interpreting the ability of triathletes to effectively transition from cycling to running, by way of using rate constants for $\dot{V}O_2$ and HR variables and tracking fluctuations in $t_{1/2}$ and MRT during the rise to steady state. Finally, the dissociation of $\dot{V}O_2$ and HR during the cycle-run transition phase shows that these two variables respond independently for when running after cycling at moderate intensities and either $\dot{V}O_2$ or HR values should not be used to estimate the other variable.

5.4 Recommendations for Future Research

Based upon the results of the current study, we recommend future research should consider:

- Employing higher intensity (above lactate threshold) exercise protocols to further replicate competitions demands particularly as higher intensity prior cycling may reflect similar results cited in previous 'priming'/prior exercise effects during subsequent exercise.
- Further testing using two testing cohorts; highly trained and moderately trained triathletes, and a greater number of participants in an attempt to confirm or prove contrary evidence to the results of past research.
- Utilising a protocol inclusive of an isolated run, run-run and cycle-run bout of exercise would provide a well-rounded comparison of results with which to confidently determine the impacts of prior cycling on subsequent running performance.
- Further analysis of rate constants and time constants for $\dot{V}O_2$ and HR, with respect to running speeds and cycling power profiles that closely replicate

competition demands, in order to determine how increased running speed after prior cycling impacts the achievement of steady state.

- Further analysis of the specific cardiopulmonary responses taking place during the cycle-run transition may better improve the understanding of the physiological dissociation of the $\dot{V}O_2$ and HR relationship during this period; potentially providing a reasonable conclusion as to why certain triathletes experience discomfort during the cycle-run transition phase of triathlon.

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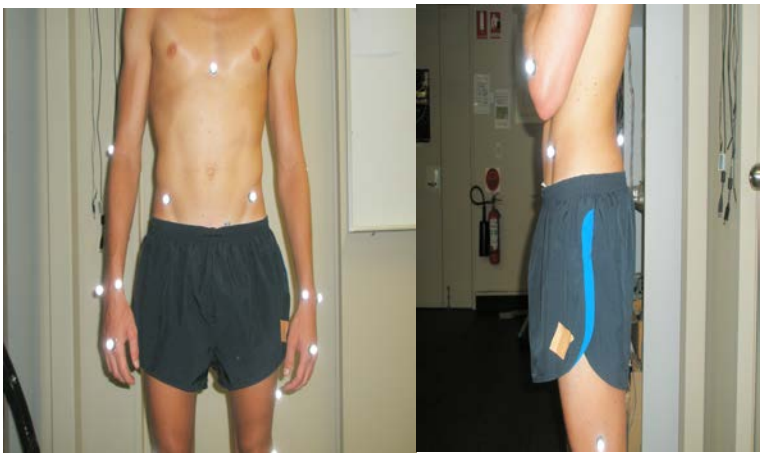
7. APPENDIX

Suggested participant testing apparel



Males: Running shorts, preferably marathon cut.

Females: 2 piece running outfit or 2-piece swimsuit