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THE EFFECT OF CYCLING CADENCE ON THE CYCLE – RUN TRANSITION IN TRIATHLETES

A Masters Thesis presented to the Faculty of the Graduate Program in Exercise and Sport Sciences Ithaca College

In partial fulfillment of the requirements for the degree Master of Science

by

Sharon L. Fitzgerald

May 2007

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Committee Member:

Candidate:

Chair, Graduate Program In Exercise and Sport Sciences:

Dean of Graduate Studie

10,2001

Date:

ABSTRACT

Purpose: This study investigated the effect of deviating from triathletes' preferred cycling cadence (PC) on triathlon cycle-run transition and 10 km run performance. Methods: Trained triathletes (N = 12) underwent three Olympic-distance cycle-run trials at race pace, during which time data were collected. The first (baseline) trial established PC, average power output (PO) and 10 km run time. The second and third trials. performed in a counter-balanced order, increased (HC) or decreased (LC) cadence by 20% from PC during the last 13 km of the cycling while maintaining PO. Cycle time and run time over the three trials were analyzed using one-way ANOVA. Six physiological variables were measured at four time points: 27 - 28 km cycle (Time 1), 38 - 40 km cycle (Time 2), $1 - 8 \min \operatorname{run}$ (Time 3) and $8 - 10 \operatorname{km} \operatorname{run}$ (Time 4). Physiological variables were analyzed at Time 1 and Time 4 by one-way ANOVA, whereas variables at Time 2 and Time 3 (cycle-run transition) were analyzed by 3 x 2 (trial x time) ANOVA with repeated measures on both factors. Results: The cycle-run transition was influenced by cadence manipulation. When comparing HC to LC, HC resulted in a smaller change in energy requirements when transitioning from cycling to running. The LC strategy was more energetically efficient than HC during cycling, but resulted in a greater change in energy requirements during the cycle-run transition. Overall, the HC strategy was more physiologically demanding than the LC trial. The cadence interventions did not influence run time. Conclusions: Triathletes may minimize energy usage during cycling by utilizing a lower than preferred cadence prior to running, however, the cycle-run transition may be minimized by utilizing a higher than preferred cadence strategy.

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I have to admit that writing acknowledgements is a little scary, as I am almost certain to leave a vitally important person out. I'll start with the obvious people. I'll always need to thank my family for any of my life's achievements, as I was lucky enough to have the supportive, stable and loving upbringing that would leave a Cosby kid jealous. I also wish to thank the people directly involved in my thesis: my wonderful subjects, who always made the hours of data collection informative and entertaining; Jon Hudak, for helping with initial set-up and data collection; and Dr. Gary Sforzo and Dr. Tom Swensen, my thesis advisors, for the unlimited patience, direction, advice, time and skill they devoted to this study. They helped make this undertaking a rich and rewarding experience. Lastly, I thank my Ithaca family – my family away from home – my amazing IC peers who made me fall in love with this place. You know who you are.

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Chapter 1

INTRODUCTION

The nature of multi-sport disciplines, such as triathlon, calls for the athlete to efficiently transition from one event to the next. Depending on the sport, transitioning may require the athlete to utilize muscle groups in differing ways to perform each leg of the competition. In triathlon, the fatigue and awkwardness that can accompany the cycle-run transition is often referred to as the 'transition phase', and is considered by many to be a particularly difficult aspect of an already grueling event. The points within the cycle and run legs that define the beginning and end of the transition phase are not clear. For the purpose of their study, Millet and Vleck (2000) defined the transition phase as being from the last km of the cycle leg to the first km of the run. Hue, Valluet, Blonc, and Hertogh (2002) defined it as incorporating the cycle-run change and the first lap run around a 333 meter track, whereas Millet, Millet, and Candau (2001) simply state that it may last for up to 20% of the run in an Olympic distance race. Regardless, the transition from cycle to run results in a suboptimal run bout and a subsequent reduction in overall triathlon performance (Hue, Le Gallais, Boussana, Chollet, and Prefaut, 1999).

The reduced performance associated with the transition phase is attributed to various physiological events, such as glycogen depletion, dehydration and a metabolic shift toward fat oxidation (Millet and Vleck, 2000). The change in mechanical function of muscle, from primarily concentric to eccentric contractions of the quadriceps (due to cycling and running, respectively), may also negatively influence the transition phase (Bijker, de Groot, and Hollander, 2002; Heiden and Burnett, 2003). Other factors, including training technique and volume (Hue et al., 2002; Millet et al., 2001),

competition experience (Millet and Bentley, 2004), bicycle configuration (Garside and Doran, 2000; Gonzalez and Hull, 1989; Olds, Norton, Lowe, Olive, Reay, and Ly, 1995), race strategy and tactics such as drafting and cadence manipulation (Billat, Mille-Hamard, Petit, and Koralsztein, 1999; Gottschall and Palmer, 2002; Vercruyssen, Brisswalter, Hausswirth, Bernard, Bernard, and Vallier, 2002; Vercruyssen, Suriano, Bishop, Hausswirth, and Brisswalter, 2005) may also influence the duration and intensity of the triathlon transition phase.

Investigations into the transition phase phenomenon have identified possible causes and training methods that may overcome it via physiological adaptation, but not many attempt to identify a strategy to reduce its occurrence or duration. Since the transition between cycling and running significantly affects subsequent running performance, it would be useful to identify a technique that attenuates the negative impact of the transition phase. Altering cycling cadence during the final stages of the cycle leg may improve transition and ultimately enhance the subsequent run leg, and therefore, race time.

The optimal method of cycling cadence manipulation is a point of contention in recent research. Some athletes, including Tour de France champion Lance Armstrong, use lower gears to decrease pedal crank resistance, thereby reducing torque required to turn the crank (Coyle, 2005). Power output is maintained (power being a function of torque (τ) and angular velocity (ω), such that $P = \tau \cdot \omega$) by increasing cycling cadence. Armstrong's average time trial cadence is between 95 and 100 rpm (The Official Source for All Things Lance Armstrong, n.d.), which is in line with the cadence seen in other elite endurance cyclists competing in major cycling tours (Lucia, Hoyos, and Chicharro,

2001). Gottschall and Palmer (2002) also endorsed a high cadence strategy when they found that post-cycle run times improved by 4% when subjects cycled at a cadence 20% faster than their preferred cadence. In contrast, lower cadences improved run time to fatigue in a different study (Vercruyssen et al., 2005). The Vercruyssen et al. (2005) study differed from Gottschall and Palmer (2002) in that Vercruyssen et al. (2005) kept cycling power consistent across the baseline and the two trial tests, possibly allowing tighter control over the effect of cadence on ensuing running performance. However, the Vercruyssen et al. (2005) protocol of measuring run time to fatigue is not a realistic reflection of triathlon competition.

This study mimicked the cycling portion of the Vercruyssen et al. (2005) study protocol by altering cycling cadence by \pm 20% from an established preferred cadence (PC) during the final third of two experimental cycle-run bouts. However, it differed in that subjects performed a 40 km cycle followed by a 10 km run, as is required of triathletes in an Olympic distance race. The effect of each cycling condition on the performance time of a subsequent, race-simulating 10 km treadmill run was measured.

Statement of Purpose

This study had two primary purposes. The first was whether a particular cadence strategy had an altering effect on six physiological variables (which for the purposes of this study will operationally define physiological effort) during the transition phase. The second was to determine whether altering cycling cadence during the last 13 km of the cycling leg of an Olympic distance triathlon resulted in an improved run time. Additionally, we also investigated the influence of these cycling strategies on the physiological effort experienced during the final 2 km of the running leg.

Hypothesis

The null hypothesis for this study is:

Instructing a trained triathlete to increase or decrease cycling cadence by 20% from preferred cadence during the final 13 km of the cycle bout will not cause a change in running time or physiological effort when compared to baseline performance.

Assumptions of the Study

For the purpose of this study, the following assumptions were made:

- 1. The subjects are representative of typical trained triathlon competitors.
- 2. The adoption of a $\pm 20\%$ change in cycling cadence during the last 13 km of the cycle bout was not affected by a neuromuscular learning adaptation.
- 3. Subjects completed all trials as though they were competing under race conditions.
- Subjects did not alter their training regimen during the study period;
 further, the completion of these trials did not result in a training effect.

Definition of Terms

The following terms are operationally defined for the purpose of this investigation:

1. Anaerobic Threshold (VO_{2AT}): The highest sustained intensity of exercise for which measurement of oxygen uptake can account for the entire energy requirement (Svedahl and MacIntosh, 2003). Higher intensities produce a surge in lactate production as working musculature shifts towards anaerobic ATP production via glycolysis. The intensity at which VO_{2AT} occurs is represented as a percentage of VO_{2max} . For the purposes of this paper, anaerobic threshold is analogous to lactate threshold (LT), however, this paper will utilize the term 'VO_{2AT}' only.

- 2. Angular Velocity: The speed at which the pedal crank is turned by the triathlete.
- 3. Cadence: The angular velocity of the bicycle pedal crank, measured in revolutions min⁻¹ (rpm).
- 4. Cardiac Output: The volume of blood ejected by the heart in one minute, measured in ml·min⁻¹.
- 5. Drafting: The positioning of an athlete's bicycle in the proximity of
 another moving vehicle so as to benefit from reduced air resistance (USA
 Triathlon, 2006).
- Drafting Zone: A rectangular area 7 m long and 2 m wide surrounding each bicycle (USA Triathlon, 2006).
- 7. External potential work (W_{pot}): Calculated using the formula mass x gravity x height, and measured in Joules. Mass is the body mass in kg; gravity is constant at 9.81m·s⁻²; height is the change in vertical height of the body's center of gravity during the stride, such that height = height_{max} height_{min}, and is measured in meters.
- 8. External kinetic work (W_{kin}): Calculated using the formula $\frac{1}{2}$ mass x velocity², and is measured in Joules. Mass is the body mass measured in kg; velocity² is the change in horizontal velocity of a body's center of gravity during the stride, squared, such that velocity² = velocity_{max}² velocity_{min}². Units are m.s⁻¹.

- 9. Net Energy Cost (EC): Calculated using the formula (VO₂ 0.083) / V.
 VO₂ is measured in ml·kg⁻¹·min⁻¹, and 0.083 ml·kg⁻¹·min⁻¹ is the average resting metabolic rate in young adults (Millet and Bentley, 2004). V is the mean velocity of the treadmill, measured in m·s⁻¹.
- 10. Physiological Effort: Defined in the present study as the response of six physiological variables (heart rate, oxygen consumption, minute ventilation, respiratory exchange ratio, rating of perceived exertion, and blood lactate concentration) measured at four time points during a 40 km cycle / 10 km run trial.
- 11. Seat Tube Angle: The angle between a horizontal line (drawn towards the rear wheel through the axis of the pedal crank) and the seat tube.
- 12. Stride: The time period during running between the grounding of one foot, and the next time that same foot is grounded. A stride may be referenced in terms of stride length, measured in meters; stride duration, measured in seconds; or stride frequency, strides per second or strides s^{-1} (Hz).
- Torque: Angular force, and for the purpose of this study is the amount of force that must be exerted on the bicycle pedal to turn the pedal crank.
- 14. Transition: The time period during triathlon between when the athlete ceases one activity (e.g., cycling) and begins the next activity (e.g., running).
- 15. VO₂: The rate of oxygen consumption, measured in $ml \cdot kg^{-1} \cdot min^{-1}$.
- VO_{2max}: The maximal rate of oxygen consumption the body is capable of performing, usually determined during a maximal VO₂ test.

17. Venous return: The speed of blood return to the heart from the systemic circulation.

Delimitations

- 1. A stationary cycle, not influenced by drafting, was utilized in this study.
- 2. Only trained triathletes participated in this study.

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- Only male subjects between the ages of 18-48 years and female subjects between the ages of 18-55 years were recruited for this study.
- 4. A study protocol of a 40 km cycle followed by a 10 km run to mimic that seen in an Olympic distance triathlon was used in this study.
- A study protocol of varying cadence by ±20% during the last 13 km of the cycle bout was used in this study.
- Some subjects were unfamiliar with laboratory equipment and/or conditions prior to study participation.

Limitations

- 1. The results of this study may be limited in application to non-drafting races.
- 2. The results of this study may be limited in application to trained triathletes.
- 3. The results of this study may be limited in application to male triathletes aged between 18 and 48 years, and female triathletes aged between 18 and 54 years, who compete in Olympic distance competition.

- 4. The results of this study may be limited in application to varying cadence by $\pm 20\%$ during the last 13 km of the cycle leg of an Olympic distance triathlon.
- 5. Some subjects may have improved trial performance due to increased equipment familiarity, rather than due to the experimental conditions.

Chapter 2

REVIEW OF LITERATURE

The phenomenon of the triathlon transition phase is well studied, with most papers finding that it contributes to a decrease in competitive performance. The mechanisms involved in transition phase appearance have been previously studied, but research into minimizing the transition phase is a more recent trend. To better understand the transition phase, several factors considered essential to successful triathlon performance will be reviewed. These include the aerobic foundation required to perform any endurance event, and the impact of an athlete's ability to perform aerobically at increasing exercise intensities. The effect of physical efficiency and economy on triathlon performance will also be reviewed, as will the influence of training volume, training technique and quantity of competitive experience. Race strategy, including drafting, bicycle configuration and cadence manipulation will also be discussed, with particular emphasis on the effect of several cadence strategies on triathlon run performance.

Aerobic Capacity and Endurance Performance

An athlete's ability to both deliver and, to a lesser extent, utilize oxygen in working musculature significantly affects endurance performance (Bassett and Howley, 2000). There are many variables that determine ability to consume oxygen at a given intensity (Coyle, 1995). These variables included muscle capillary density, stroke volume, aerobic enzyme activity, and muscle fiber composition, which affect muscle

economy. Collectively, these variables affect performance velocity or race pace (Coyle, 1995).

The focus of much recent study has not solely been on performance velocity, but on the energy cost of performance and the influence of this cost on the athlete during competition. Coyle (1995) maintained that race pace is more determined by VO_{2AT} than VO_2 , which introduces the relationship between VO_{2max} and VO_{2AT} . It has long been accepted that VO_{2max} is a primary measure when analyzing endurance capacity and training adaptation, as well as being a major consideration when establishing an exercise prescription (Bassett and Howley, 2000). However, the validity of using VO_{2max} exclusively to predict endurance performance has recently been questioned. VO_{2max} is an important predictor, but to well-trained athletes, the percentage of VO_{2max} where lactate threshold occurs (i.e., VO_{2AT}) may be just as or more important than VO_{2max} (Hue, Le Gallais, and Prefaut, 2000). Coyle (1995) found that although two to three years of intensive training saw an increase in VO_{2AT}, changes in VO_{2max} were minimal after that point. This may be especially relevant to triathlon, a point supported by Roalstad (1989). She suggested that because competitive ultraendurance triathlon athletes demonstrated a wide variance in their VO_{2max} capabilities, the correlation of VO_{2max} to triathlon performance was even weaker than single sport events. A study by Coyle (1988) found VO_{2AT} to be a more relevant measure of elite endurance performance than VO_{2max} . Coyle (1988) demonstrated a strong relationship (r = 0.90) between VO_{2AT} and time to fatigue. Another study by Millet and Bentley (2004) also found that VO_{2AT} was significantly correlated to both cycling and running performance in triathlon.

Therefore, because performance velocity for most athletes is at or slightly above their VO_{2AT} (Basset and Howley, 2000; Coyle, 1995), a discussion regarding an athlete's performance capacity that references VO_{2max} should be done in conjunction with that athlete's VO_{2AT}. The combination of both values allows the translation of a relative term (VO_{2AT}) into an absolute value, which enables a fair comparison between athletes. For example, blood lactate will rise at a VO₂ of 49 ml·kg⁻¹·min⁻¹ in an athlete with a VO_{2max} of 70 ml·kg⁻¹·min⁻¹ and a VO_{2AT} of 70% VO_{2max}. Contrast this with another athlete, who has an identical VO_{2max}, but experiences VO_{2AT} at 60% VO_{2max}. A pace that requires this athlete to consume 42 ml·kg⁻¹·min⁻¹ of oxygen will elevate lactate concentrations considerably. Given that both athletes consume the same amount of oxygen at maximal exertion, a higher VO_{2AT} enables the first athlete to work harder while still primarily generating ATP aerobically. Thus, it stands to reason that the first athlete will maintain a faster race pace and post a superior time in competition.

Metabolic Cost of Triathlon Performance

Triathlon provides the athlete with a hurdle not experienced in single discipline events, i.e., performing multiple rhythmic movements (cycling then running) during the course of the event. The fact that cycling is a non-weight bearing exercise and running is a weight bearing one also provides an additional biomechanical challenge, as the athlete needs to maintain muscle coordination while shifting from the primarily concentric movement of cycling to the primarily eccentric movement of running (Heiden and Burnett, 2003). This shift affects muscle activation and therefore contributes to the difficulty of the transition phase.

The effect of the triathlon transition has been investigated by several studies. Hue et al. (1999) compared ventilatory response during the first 10 min of running after endurance cycling to those taken during running after endurance running. The initial run and cycle bouts were performed at equal intensities. Based on ventilatory data collected, the authors found that the ventilatory response after cycling was significantly higher than that seen after running at the same intensity. It was concluded that the pulmonary function changes seen during the first 10 min of running after a cycle bout may be associated with respiratory fatigue, and possibly with exercise induced hypoxemia. Since VO₂ during running after a cycle or run bout was not significantly different, the authors suggested there was no difference between the energy cost of running after cycling or running. As such, Hue et al. (1999) proposed that the awkwardness of the transition phase was due to a disturbance in the respiratory system, possibly due to respiratory muscle fatigue coupled with extravascular water accumulation. Data from Millet and Vleck (2000) confirmed and extended this proposition that respiratory fatigue may be due to hyperventilation, which was said to occur following a chain of events. Prolonged endurance exercise causes a depletion in glycogen stores, which increases the demand on fat oxidation to generate ATP. Accelerated fat oxidation will increase oxygen demand. which is satisfied by increased breathing frequency and tidal volume, thus leading to * hyperventilation. Dehydration was also said to negatively affect the transition phase, as it causes haematoconcentration, which decreases stroke volume, thus increasing heart rate. However, neither Hue et al. (1999) nor Millet and Vleck (2000) indicated whether respiratory fatigue is associated only with the transition phase: assuming that glycogen stores are not suddenly replenished, it seems the aforementioned chain of events would

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continue until the race is completed. As such, whether respiratory fatigue is associated only with the transition phase is unclear.

A study by Bijker et al. (2002) highlighted another possible basis for the transition phase. They compared EMG activity in the vastus lateralis (VL), biceps femoris (BF) and gastrochemius (GS) muscles during cycling and running as power output increased. It was found that the mean EMG activity in the VL and BF did not increase during running as power output increased, although GS EMG activity did. The stability of VL and BF activation during a concomitant power increase was unexpected, considering that muscle EMG activity and power output are positively correlated. The authors attributed the VL and BF response to the effect of the stretch-shorten cycle. This is seen during eccentric muscle contraction, which occurs due to the braking and stabilizing functions of the VL (and other quadriceps group muscles) after heel-strike and BF prior to heel strike. These actions store potential energy in the stretched tendons, which is re-used in the subsequent concentric knee extension movement. This increase in stored energy consequently increased the gross efficiency of these muscles, indicating they are more efficient during running than when contracted in isolation. In contrast to running, all three muscles demonstrated an increased EMG pattern during cycling as power output increased, suggesting cycling is almost wholly a concentric activity. This contrast in mechanical function (i.e., changing from a concentric to eccentric movement pattern), may temporarily result in decreased muscle efficiency, thus contributing to the difficulty of the transition phase.

Heiden and Burnett (2003) performed a similar study with a triathlon-oriented approach. They compared the level and duration of EMG activity in six lower limb

muscles [rectus femoris (RF), VM, BF, VL, GS, and gluteus maximus (GM)] during running following a previous cycling or running bout. During the flight stage of the running stride, all muscles investigated showed a slightly higher level of activation after cycling, although only the VL data were significant. These data suggested that knee extension demands greater muscle activation after a concentric activity such as cycling, as the pedaling motion does not require full knee extension. This elevated muscle activity may negatively influence performance during the transition phase. During the running stance phase, all quadriceps muscles studied (RF, VL and VM) had a higher activation level after cycling, although again only the VL data were significant. The authors accredited this to the possibly increased level of stability required in the knee joint when moving from a non-weight bearing to a weight bearing exercise. Thus, in addition to decreasing muscular efficiency, as highlighted by Bijker et al. (2002), the change in quadriceps function from concentric to eccentric contraction may result in a temporary increase in the level and duration of EMG activity of this musculature, seemingly until a more rhythmic movement is regained.

A study by Wells, Stern, Kohrt, and Campbell (1987) implied yet another reason for the difficulty associated with the triathlon transition when they investigated the effects of a sequential cycle-run bout on vascular and cellular fluid volumes. They found that the overall effect of this activity, regardless of ordering (i.e., whether cycling or running was performed first), resulted in significant weight loss regardless of water intake. However, performing a cycle-run bout, as occurs in triathlon, resulted in a significantly lower red cell volume compared to the run-cycle bout. While the authors did not speculate on either the cause or effects of this occurrence, Hue et al. (2002) hypothesized

that it may be caused by the abrupt change in posture required to transition from cycling to running.

Determinants of Triathlon Performance

Some elements involved in successful triathlon performance are well identified, while other aspects have been the basis for conflicting study results. The elements that will be discussed in this section are training technique, volume of competitive experience, equipment mechanics and race strategy.

Training Technique

Although triathlon is considered a multi-sport event, training cannot simply be composed of a series of swimming, cycling and running sessions. While it is accepted that each discipline is trained for independently, the athlete must also train for the transition phase; namely, transitioning from cycling to running. It is acknowledged that a swim-cycle transition also exists, but research shows that this transition affects overall performance less than the cycle-run transition (Laursen, Rhodes, and Langill, 2000; Millet and Bentley, 2004).

A common way for triathletes to train for the transition phase is by performing multicycle-run blocks, with the athlete repeatedly completing cycle bouts that are immediately followed by running bouts to simulate the transition phase. Hue et al. (2002) hypothesized that because European and Australian triathlon teams have successfully used this technique for several years, it seemed likely to improve cycle-run performance. The study randomly divided competitive triathletes into either an experimental or control group. The experimental group incorporated a multicycle-run protocol into their regular training for six weeks, while the control group continued their normal training regimen. For the purposes of the experiment, the transition phase was determined to incorporate both the cycle-run change and the first lap run around a 333 meter track. The study found that while the experimental group did improve their performance through the transition phase, the overall performance time of the experimental group was not significantly better than the control.

Sheer training volume may also play a part in transition performance. Millet et al. (2001) compared the transition phase response of middle-level and elite athletes. The study required all subjects to perform a seven minute run both before and after a maximal cycling bout. Run speed was recorded during the first and last minute of both run bouts. One of the variables measured was 'mechanical cost' of work performed, which was calculated as the sum of external potential and kinetic work, divided by stride length. The first minute of the run leg, in both the pre and post run conditions, was significantly more costly for middle-level athletes than elite athletes. The difference in the mechanical cost (mean \pm SD) of exercise between the pre and post run conditions was $0.4\% \pm 6.9\%$ for elite athletes, whereas the difference was $7.1\% \pm 6.0\%$ for middle-level athletes. Based on these results, the authors suggested that the middle-level athletes were more sensitive to cycling fatigue. The substantial standard deviation for both the middle and elite level triathletes, however, indicates a diverse range of individual responses to the test. By the sixth minute of running, the mechanical cost was similar for both groups, which was said to mark the end of the transition phase. Given these results, the authors concluded that the techniques utilized by elite athletes, such as increased cycling and

running mileage, should be adopted by middle-level athletes wishing to improve their competition performance.

Triathlon Experience

Millet and Bentley (2004) investigated whether the quantity of competition experience affected triathlon performance by comparing the energy cost of running after cycling between male and female senior and junior elite triathletes. Subjects performed a consecutive run-cycle-run bout, during which both physiological and performance data were collected. One physiological variable measured was the net energy cost (EC) of running both before and after the cycle bout. Net EC is similar to the mechanical cost of running after a cycling bout, as examined by Millet et al. (2001). However, in contrast to Millet et al. (2001), the net change in EC (Δ EC) between the first and second mean run bout times were significantly different only between junior and senior females. The authors concluded that senior female triathletes were distinguishable from their junior contemporaries by their significantly lower ΔEC between the two running bouts. The conclusion is weakly supported by the data, due to the large standard deviation and small sample size. In contrast, the senior male triathletes were distinguished from their junior contemporaries by a significantly higher ventilatory threshold, which occurs at approximately the same exercise intensity as VO_{2AT} .

The differences in net EC (between female senior and junior triathletes) and VO_{2AT} (between male senior and junior triathletes) could also be attributed to increased muscular efficiency that occurs as an athlete matures. Coyle (2005) conducted a longitudinal study that investigated the adaptations seen in cyclist Lance Armstrong over a seven year period. Interestingly, Armstrong's VO_{2AT} did not improve during this

period; in fact, it decreased from 85% VO_{2max} to 76% VO_{2max} from 1992 to 1997. However, the author attributed Armstrong's continued success to constant physiological adaptation to training that lead to improved muscular efficiency, which was defined as the ratio of the amount of work produced to the energy used to produce it. Both gross and delta efficiency increased from 21.18% and 21.37% respectively in 1992 to 23.05% and 23.12% respectively in 1999. This efficiency was reflected in the power produced when consuming 5.0 L O₂ min⁻¹, which increased from 374 W to 404 W during the study period. However, it is interesting that the exact physiological mechanisms responsible for this athlete's 8% increase in efficiency remained unclear. As a side note, the author commented that it was 'remarkable' that these improvements were achieved during a period where Armstrong developed and overcame advanced cancer, which required both surgery and intensive chemotherapy.

Equipment Mechanics

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The mechanics of an athlete's bicycle also influence overall triathlon performance. Garside and Doran (2000) investigated the influence of varying the bike seat tube angle on subsequent run-bout performance. It was found that a more upright or 'forward' angle (81° 'steep' vs. 73° 'shallow') significantly improved the run performance during the first 5 km of the race-simulating 10 km run. They proposed that the adopted riding position more closely mimicked the natural running position, thus reducing the effect of the transition phase.

Bicycle wheel diameter also positively influences overall race times (Olds et al., 1995). Cyclists often configure their bicycles so the front wheel is smaller than the back, which is said to have a two-fold advantage. The first is that a smaller front wheel

diameter facilitates closer drafting to a leading athlete, and the second is that it tips the rider slightly forward, reducing the surface area presented to wind resistance. Indeed, Olds et al. (1995) did find that bicycles configured with smaller front wheels resulted in a reduced wind resistance over a 6.5 km time trial course. When drafting was permitted during comparison trials, the same distance was covered up to 1.5% faster. Although these improvements seem small, they could positively influence elite competition results (Olds et al., 1995).

Gonzalez and Hull (1989) investigated the optimal bicycle configuration for a given rider by identifying which design resulted in the lowest movement cost. They chose the bicycle crank arm length, seat height, longitudinal foot position (i.e., the distance between cleat attachment point on the pedal and the lateral malleolus), cadence, and seat tube angle as bicycle variables, and divided subjects into three height categories: the 'small', 'average', or 'tall' man. They found that as the height of the rider increased, crank length, seat height and foot position should also increase, whereas seat tube angle and pedaling rate should decrease. It was stressed that because all geometric variables (with the exception of foot position) are comparable in terms of statistical sensitivity, each should be given careful consideration when assembling and adjusting bicycle equipment.

Race Strategy

Triathlon literature is abundant with 'optimal' race strategy and tactics, many of which flatly contradict each other. Vleck et al. (2006) found that a reduced pace during the swim leg results in the athlete attempting to catch up during the cycle, thus negatively affecting run time and overall race position. Sleivert and Wenger (1993) indirectly

agreed, saying that because the run leg is the biggest overall triathlon indicator for both men and women, it is important to conserve energy for this leg. Conversely, a study by Peeling, Bishop and Landers (2005) concluded that swimming below time trial intensity could significantly improve cycling time and overall triathlon performance.

Drafting is a widely-used racing strategy, which involves using the drafting-zone of a leading athlete to lessen the effect of wind resistance during cycling. The drafting athlete cycles in the leading athlete's slipstream, which is an artificial tailwind within which air is already moving forward when the drafting athlete reaches it. Drafting reduces wind resistance, which constitutes over 90% of the mechanical resistance against a bicycle when traveling over 8.9 m·s⁻¹ (19.9 mph) (Kyle, 1979). The smaller this resistance, the easier it is to cycle at a higher intensity, which improves subsequent run time (Vercruyssen et al., 2005). Drafting has been legalized for professional International Triathlon Union (ITU) World Cup triathlons since 1996 (C. Elford, personal communication, June 27, 2006), but continues to be illegal for age-group races.

Drafting has been a topic of interest for some time. Some studies have attempted to identify optimal drafting strategies to ease the cardiovascular demand of maintaining a high cadence, and thus reduce the transition phase during draft-legal races. A study by Kyle (1979) investigated the reduction of wind resistance while athletes ran and cycled in groups. He found that total wind resistance decreased by an average of 44% if there was no gap between the leading cyclist's back wheel and the drafting cyclist's front wheel. Increasing the wheel gap to two meters decreased the drop in total wind resistance to an average of 27%. Kyle (1979) also investigated the effect of body position during cycling. Tailing subjects found a greater drafting benefit when the leading cyclist assumed the upright riding position rather than the crouched over racing position, which seems obvious given that the former position would present a greater surface area and generate a larger slipstream. Olds et al. (1995) stated that there is no drafting benefit if the wheel gap exceeds three meters, which makes it interesting that the ITU considers riders to be drafting if they follow within six meters of a leading cyclist.

A study by Hausswirth, Lehenaff, Dreano, and Savonen (1999) investigated the benefits of drafting on several physiological factors including energy expenditure, heart rate and ventilation, as well as subsequent run performance time. National level triathletes performed a baseline 5 km isolated run, which was compared to running after both a drafting and a non-drafting cycle bout. One of the interesting findings was that preferred cycling cadence increased significantly when drafting, but this was accompanied by a 14% reduction in VO₂. Drafting may have allowed the athlete to maintain his/her cycling velocity using a lower power output. This was indicated by the reduced VO₂ and the reduction in post-cyclé blood lactate measures, suggesting that the athlete was conserving energy for the more demanding run leg of the test. Thus, the ability to draft during the triathlon cycling leg may result in an increased energy reserve that can be utilized during the transition phase and possibly result in an improved run performance time.

Cycling Cadence

Another important triathlon race strategy is cadence manipulation, which is also one of the more popular areas of investigation in recent triathlon studies. The physiological effect of cycling cadence plays a primary role in the development of triathlon competition strategy. Power output (PO), which in cycling translates directly to

velocity, is a function of torque and cadence. These two variables may be manipulated when achieving a set PO, and the decision to emphasize either torque or cadence to achieve that output depends on the strength and fitness of the athlete, respectively. MacIntosh, Neptune, and Horton (2000) chose to vary cadence to identify the minimum level of muscle activation necessary to achieve a power output equivalent to 50-55% of each subject's VO_{2max}. The EMG amplitude of seven lower limb muscles (soleus, medial gastrocnemius, tibialis anterior, VM, RF, long head of BF and gluteus medius) was measured during these tests. It was found that the minimal level of muscle activation to achieve a given PO occurred at a unique or 'optimal' cycling cadence, and that there was a positive correlation between PO and optimal cadence. This information would be particularly useful to higher level triathletes, who race at a level of competition where the difference between athletes tends to be their ability to perform at high intensities for a long duration (Coyle, 1995; Roalstad, 1989), rather than superior strength.

Effect of Cadence on Subsequent Running

The effect of cycling cadence on ensuing running performance has been the focus of several recent studies. The resultant literature is equivocal, implying that there are both effective and detrimental ways of manipulating cycling cadence. Bernard, Vercruyssen, Grego, Hausswirth, Lepers, Vallier, and Brisswalter (2003) investigated how cycling at 60, 80 or 100 rpm affected a subsequent 3 km run bout. These cadences were selected as they are close to those previously shown to represent the energetically optimal cadence (EOC), the freely chosen cadence and typical drafting cadence, respectively, demonstrated by study subjects. Subjects' performance during a subsequent

run was compared to an isolated run trial that served as the control. Bernard et al. (2003) found that, although there was no significant effect of cycling cadence on ensuing run performance, cycling at the two higher cadences increased stride rate and running velocity during the first 500 m of the run. They also found that higher cadences were associated with higher HR and VO₂ values during the run. The authors concluded that the elevated metabolic cost associated with high cadence strategies lead to an unstable running pattern; hence, it was unwise to adopt such a strategy. However, their data showed that cycling at the low cadence significantly elevated VO₂ after the first km of the run and VO₂ continued to rise until completion of the run trial. This rise in VO₂ was not discussed by the authors, but it seemed to signify the appearance of the slow component of oxygen uptake kinetics, indicating that adopting a lower cadence strategy could negatively influence run performance. Additionally, the use of an isolated run trial as a baseline seemed questionable, especially when investigating the effect of cycling on subsequent run performance.

Gottschall and Palmer (2002) supported a fast cadence strategy after they investigated the effect of cycling 20% faster or slower than an established preferred cadence. The study protocol had subjects complete a 30 min cycling bout prior to running 3.2 km. Subjects initially completed a baseline control trial, where they were asked to maintain a cadence that simulated racing conditions, and then performed two experimental trials that altered their baseline cadence by $\pm 20\%$ for the duration of the cycle bout. In line with the Bernard et al. (2003) findings, initial stride frequency increased during the $\pm 20\%$ trial. Run times also improved by 4% in comparison to the baseline run bout, and by 7% in comparison to the -20% run bout. Their study design

ensured heart rates were constant throughout the three cycling trials in an effort to ensure that cadence was the only influencing factor on subsequent run performance.

Vercruyssen et al. (2002) investigated the effect of different cycle cadences on the appearance of the VO_2 slow component during a subsequent running performance. Three different cadences were utilized, which were either freely chosen by the athlete (FCC), or mathematically calculated (mechanically optimal cadence: MOC and energetically optimal cadence: EOC). The study protocol consisted of three 30 min cycle run bouts followed by a 15 min run, and each subject performed all trials. A 45 min isolated run trial, which was broken into 30 and 15 min portions in order to standardize all four tests, served as a control. After each cycling bout, subjects performed a run bout at an intensity that was controlled across all tests, during which oxygen consumption was measured. Results showed that performance of the MOC and FCC, which were the highest cadence tests $(90.2 \pm 0.8 \text{ rpm} \text{ and } 81.2 \pm 7.2 \text{ rpm} \text{ respectively})$, coincided with the appearance of the VO₂ slow component during the run bout, while the EOC (72.5 \pm 4.6 rpm) led to a stable VO₂ during running. By definition, the slow component of oxygen uptake kinetics, or a delayed steady state, appears at an exercise intensity higher than steady state. This exercise intensity is generally above VO_{2AT}, and is associated with physiological changes that include elevated muscle temperature and the recruitment of Type II muscle fibers (Vercruyssen et al., 2002). These changes indicate that the subject would be unable to maintain that intensity for the duration of an event such as the 10 km triathlon run, therefore resulting in suboptimal running performance. Thus, these authors suggested that a cadence that elicits the VO₂ slow component during the subsequent run is unsuitable for triathlon competition.

Vercruyssen and his colleagues followed up this study with another in 2005, which investigated the effect of different cycling cadences on subsequent run time to fatigue. The cycling protocol consisted of a baseline 30 min time trial to establish FCC, and two experimental trials that varied the FCC by $\pm 20\%$ during the last 10 min of the 30 min bout. Each cycle bout was followed by a run to fatigue, where subjects were instructed to maintain a pace equivalent to 85% VO_{2max}. Each subject performed both experimental tests. It was found that performance of the -20% trial saw a significantly improved run time to fatigue in comparison to the baseline and +20% tests. These findings solidify the results of the Vercruyssen et al. (2002) study, which found that a +20% strategy is detrimental to running performance, but contradicted the Gottshall and Palmer (2002) study that indicated a +20% strategy resulted in faster run performance over 3.2 km. The Vercruyssen et al. (2005) study differs from Gottschall and Palmer (2002) in that Vercruyssen et al. (2005) ensured PO was consistent across the baseline and the two trial tests, possibly allowing tighter control over the effect of cycling cadence on ensuing running performance. In contrast, Gottschall and Palmer (2002) kept HR constant, meaning that the absolute PO generated during the increased cadence trial may have been less than that generated during the decreased cadence trial (PO was not reported in this study). A decreased PO could have resulted in reducing muscular fatigue during the higher cadence trial, possibly lessening the influence of cadence on subsequent run performance." However, Lepers, Millet, Maffiuletti, Hausswirth, and Brisswalter (2001) found that HR and VO₂ were not affected by a $\pm 20\%$ cadence strategy during a 30 min cycle bout at 80% maximal aerobic power. Nevertheless, the fact that Vercruyssen et al. (2005) measured run time to fatigue rather than run time over a set distance may

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render their results as less relevant to triathlon competition. As such, an investigation into the influence of different cadence strategies on run performance within an environment more reflective of actual triathlon competition may shed light on this matter.

Summary

This literature review discussed variables that influence cycling technique, the appearance and severity of the transition phase, and its affects on subsequent run performance in triathlon. As is appropriate for all endurance sports, triathletes must possess a strong foundation of aerobic fitness, which recent research shows is better indicated by the VO_{2AT} in conjunction with VO_{2max}, rather than just the latter. However, because triathlon is a multi-sport event, it provides a transitional challenge not seen in regular sports (i.e., the cycle-run transition). Studies show that fatigue caused by a previous exercise bout plays a major role in the transition phase, but such fatigue is not the only source of the problem. When performing a cycle-run bout, the athlete must shift from a non-weight bearing, somewhat crouched cycling posture to the weight bearing and upright running posture. This change also requires the athlete to shift from concentric contraction of the hip and knee that predominates during cycling to the eccentric contraction of the knee required during running. Many studies have found these shifts to be difficult for the body to make. As such, they should be trained for, possibly by the inclusion of 'multi-block' training, to expedite the required physiological adaptation.

Other research found that sheer experience and training volume, which increases muscle efficiency, is a critical aspect of successful triathlon performance. Racing strategies such as drafting, along with cycling cadence selection and optimal bicycle configuration also play a critical role.

The conflicting reports regarding cycling cadence provide the basis for this current investigation. A study by Gottschall and Palmer (2002) found that cycling at a cadence above that freely chosen by the athlete improves subsequent running performance. However, this conclusion may be questioned considering it was not clear whether PO generated during the high cadence bout was equivalent to that produced during their other tests. Other studies, including that by Bernard et al. (2003), Vercruyssen et al. (2002) and Vercruyssen et al. (2005), support a lower than preferred cadence strategy due to the improved performance seen during a subsequent running bout. However, whether the protocols employed by these studies would accurately translate to triathlon competition is unclear. This study hopes to shed light on this matter by setting a protocol similar to that utilized by Vercruyssen et al. (2005), but differs in that it will reflect Olympic triathlon distances.

Chapter 3

METHODS

In this study, the effects of manipulating cycling cadence during the final third of a 40 km cycle bout upon the physiological effort of the cycle-run transition and 10 km run performance were investigated. The following chapter outlines the methods used in this study. This chapter is divided into the following sections:

1. Subjects

2. Design

3. Equipment

4. Performance Trials

5. Statistical Analysis

<u>Subjects</u>

All testing protocols were approved by the Ithaca College's All-College Review Board for Human Subjects Research. Following a recruitment presentation to the Ithaca Triathlon Club and subsequent word-of-mouth, 15 (13 male, 2 female) triathletes who had performed at least one Olympic distance triathlon within the previous six months volunteered to participate in this study. Each subject signed an informed consent form (Appendix A) after being made aware of the study protocol, potential risks and benefits. They also completed a medical history form (Appendix B) detailing potential events or conditions, such as heart problems, that may exclude them from the study. Performance exclusion criteria for subjects consisted of achieving a maximal oxygen consumption (VO_{2max}) value of less than 45 ml·kg⁻¹·min⁻¹ for males and 40 ml·kg⁻¹·min⁻¹ for females.

Of the original 15 subjects, three were unable to consistently maintain the required cadence during the high cadence trial, consequently invalidating their data. Therefore, 12 (11 male, 1 female) subjects completed all elements of the study and were included in data analysis. Subject characteristics can be seen in Table 1.

<u>Design</u>

Each participant reported to the laboratory four times during the course of the study, and completed one test per visit. There was at least a four day rest period between each test. In the first test, subjects' VO₂ during treadmill running was measured. The second test was a baseline cycle-run bout, where subjects cycled 40 km immediately followed by a 10 km treadmill run. Each subject's preferred cadence (PC) was determined by his or her average cadence during the baseline cycle bout, as was average power output (PO). In the third and fourth tests, which were randomized to prevent an order effect, subjects were required to maintain PC during the first 27 km cycling, then altered their cadence by $\pm 20\%$ during the last 13 km. PO was to be kept constant throughout the 40 km bout. Once the cycle bout was complete, subjects performed a 10 km treadmill run, which they were instructed to perform at race pace. Subjects were given written instructions on how to prepare for both the VO_{2max} test, as outlined in Appendix D.

Equipment

All experimental cycle tests were performed on a Computrainer indoor trainer (Pro Model 8002, RacerMate, Seattle, WA), which was controlled by CompuTrainer Coaching Software 1.5 (CS) installed on a Dell Optiplex G260 computer. The

	Age	Height	Weight	PC	PO	VO_{2max}	HR _{max}
	(y)	(cm)	(kg)	(rpm)	(W)	(ml·kg ⁻¹ ·min ⁻¹)	(b·min ⁻¹)
Males $(n = 11)$	37.5	179.2	76.0	85.3	176.3	62.2 	184.0
	± 6.6	± 5.4	± 5.0	± 6.2	± 25.8	± 6.4	± 7.0
Female (n=1)	47	167.6	59.1	79	134	50	153
All	38.3	178.2	74.6	84.8	172.8	61.2	181.4
(n = 12)	± 6.9	± 6.1	± 6.8	± 6.2	± 27.5	± 71.2	± 11.2

Descriptive Characteristics of Subjects by Group and Gender

Note: data (mean \pm *SD*) are age, height, weight, maximal oxygen consumption (VO_{2max}), and maximal heart rate during an incremental treadmill running test. Preferred cycling cadence (PC) and average power output (PO) were determined during the baseline cycle – run trial. CompuTrainer allows a bicycle's rear wheel to be suspended against a magneticallybraked roller. The CompuTrainer was set to operate in the general exercise mode, where resistance on the roller is determined by rider weight and speed to replicate outdoor cycling. This software was used to create a user data file that included age, height, body weight and gender for each subject, and also to collect performance data during all tests. . Subjects were fitted with a Polar heart rate monitor (S120, Polar Electro Oy, Kempele, Finland) so HR could be monitored telemetrically during each test.

All VO_{2max} and 10 km running trials were carried out on a commercial treadmill (Precor USA C954, Woodinville, WA). During the running portion of the experimental trials, subjects used the treadmill in manual mode, and could adjust running speed autonomously. The treadmill was controlled by the researcher during the VO_{2max} trials to adjust both treadmill speed and incline as required.

As per the trial protocol, subjects were periodically fitted with a mouthpiece that directed expired air into a gas analyzer (ParvoMedics TrueMax 2400, Sandy, UT) to measure VO₂ at set intervals throughout the cycling and running bouts. During these intervals, blood was also drawn from each subject, and blood lactate was measured by the Acutrend® Lactate Analyzer (Roche, Mannheim, Germany), as described by Bassett, Merrill, Nagle, Agre and Sampedro (1991).

Performance Trials *

Maximal Tests

Each subject underwent a maximal running test to determine peak VO_2 during each test. The maximal running test was incremental, and subjects first completed a 3

min warm up phase at their estimated triathlon 10 km race pace. Once the warm up time had elapsed, the test was begun. The first 2 min of the test was performed at 0.5 mph faster than race pace, and the next 2 min at 1 mph faster than race pace. This velocity was maintained for the remainder of the test. Each subsequent 2 min stage saw an increase in treadmill grade by 2.5% until the subject reached volition exhaustion. During these maximal tests, VO₂ was recorded at 30 s intervals, and HR was recorded at minute intervals. Volitional exhaustion was determined in accordance with Howley et al. (1995) (i.e., the subject reached a plateau in VO₂ despite an increase in power output, recorded an RER of 1.15, or decided he or she could no longer maintain the exercise at the current intensity). Peak VO₂ was recorded as the average of the four highest consecutive VO₂ (ml·kg⁻¹·min⁻¹) values (Vercruyssen et al., 2005).

When the test was completed, subjects cooled down at a self-determined 'easy' pace for 2 min. At the end of the cool-down, a fingertip was sterilized with an alcohol prep pad, and a sterile lancet used to make a puncture so a blood sample could be obtained for blood lactate analysis.

Cycle-Run Bouts

All subjects performed three cycle-run bouts, during which they completed a 40 km cycle immediately followed by a 10 km treadmill run. Subjects were requested to complete each trial at their race pace to simulate competition performance. Subjects supplied their own cycling and running equipment and a fan was available to cool them during each test. Subjects who stated they routinely consumed energy supplements during a triathlon were permitted to do so during trials, but under stipulation that: a) they bring enough product to the baseline trial for all three cycle-run trials; and b) they

consume the same quantity of the same supplement at the same point/s during all cyclerun bouts. This information was recorded during each trial.

During the baseline and experimental tests, outlined in Figure 1, expiratory gas data was collected twice during both cycling and running. This required subjects to wear a gas analyzer mask to allow measurement of VO₂, which was removed when data was not being collected. During the cycle leg, respiratory measurements were taken from 27 km to 28 km (27 km being the cadence transition point) and from 38 km to 40 km. A blood sample was also taken during these times for lactate analysis, and subjects were asked for their RPE. During the experimental trials, subjects were required to change gears after 27 km in order to keep PO as close as possible to the average PO maintained during the baseline trial. Upon completion of the cycle bout, subjects were instructed to prepare for the run bout as quickly as possible, but were not to remove the gas analyzer mask as expired gases were also measured during the first 8 min of the run bout. This transition time was recorded. After 8 min of running, subjects removed the analyzer head gear themselves while they continued running, and blood was again drawn for lactate analysis. Each subject was responsible for determining his or her own running speed on the treadmill throughout the entire 10 km, with the instruction that they alter speed based on how strong they were feeling. In order to avoid motivation based on previous performances, elapsed time on the treadmill was hidden from subjects after the first 8 min of running. Subjects ran without head gear until the 8 km mark, at which point head gear was again donned and gas data collected until the end of the 10 km. Time to completion was recorded. Subjects removed the head gear, were immediately asked for their RPE, and then decreased the treadmill speed to an easy walking recovery pace for 1-2 min.

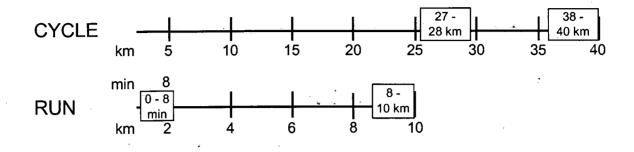


Figure 1.

Linear representation of all cycle / run trials. Text boxes indicate where physiological variables were measured.

The treadmill was then stopped, subjects were seated, and another blood sample was taken for lactate analysis.

Statistical Analysis

Once data collection was complete, a series of ANOVA analyses were performed. Firstly, two one-way ANOVA analyses were performed to identify trial differences in 1) cycle time, and 2) run time. Data were also collected on six physiological variables (heart rate, oxygen consumption, minute ventilation, respiratory exchange ratio, rating of perceived exertion, and blood lactate concentration) at four time points during each cycle-run trial. These data were analyzed in three stages as each time point was measured to meet a particular objective of the study. The time points were organized and analyzed as follows:

1. Time 1 (27 – 28 km cycle). The study protocol across all trials was identical until the 27 km point of the cycle bout. As such, none of the physiological data collected at Time 1 would be expected to exhibit a significant difference between trials at this time point. To verify physiological response consistency between trials at Time 1, a one-way ANOVA was conducted for each physiological variable. If significance was detected, post-hoc dependent t-tests were performed. Time 2 and 3 (38 – 40 km cycle and 1 – 8 min run). This time period represented the cycle-run transition phase, which was the main focus of interest for this study. Data were collected at both time points to identify if physiological effort differed between trials. As such, a 3 x 2 ANOVA (trial x time) was performed on all physiological data collected. If an interaction was found, post-hoc analysis was performed using a series of dependent t-tests. A Bonferroni correction was not

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performed on these analyses for two reasons: 1) we were not assuming the null hypothesis was true, and 2) we did not wish to increase our chances of producing a Type II error (i.e., failing to reject the null hypothesis when it is false). If no interaction was identified, significant main effects were followed by appropriate dependent t-tests.

2. Time 4. This paper notes previous studies that did not reflect realistic triathlon distances of either cycling, running, or both. As such, the Time 4 portion of the analysis determined whether a cycling strategy had any lasting influence on a full Olympic-distance triathlon run leg. To identify differences between trials, a one-way ANOVA was performed on physiological data collected. A significant finding for any physiological dependent variable at Time 4 was followed by a series of post-hoc dependent t-tests.

Statistical analyses were performed using SPSS version 13.0 for Windows (SPSS Inc., Chicago, IL). An alpha level of 0.05 was used to denote statistical significance of ANOVA measures.

Chapter 4

RESULTS

This study was performed to determine if altering cadence during the last third of a 40 km cycling bout affected physiological effort of both the cycle – run transition and overall triathlon performance, as well as 10 km run time in trained triathletes. Subjects performed three trials of 40 km cycle / 10 km run bouts. The first was a baseline trial to identify PC, average cycling PO, and subsequent 10 km run time. PO was held constant during the second and third trials, while cadence was either increased or decreased by 20% from PC during the final 13 km of cycling. This chapter describes the statistical analyses of collected data, and is divided into the following sub-sections: 1) Run Time; 2) Cycle Time; and 3) Physiological Variables, including (a) Heart Rate; (b) Oxygen Consumption; (c) Ventilation; (d) Respiratory Exchange Ratio; (e) Rating of Perceived Exertion; and (f) Lactate. These dependent physiological variables were measured at four time periods during each trial: (Time 1) 27 – 28 km cycling; (Time 2) 38 – 40 km cycling; (Time 3) 1 – 8 min running; and (Time 4) 8 – 10 km running.

Run Time

A one-way ANOVA was performed on run time data to determine whether the cycling cadence intervention had an effect on 10 km run time. The results of this analysis are outlined in Table 2. The significant difference ($F_{(2,22)} = 21.22$; p < 0.05) seen in run times between trials resulted in post-hoc dependent t-tests, which were completed to identify specific difference. The post-hoc analysis showed that baseline 10 km run time was significantly longer than HC and LC 10 km run time (Figure 2). There was no difference between HC and LC run times.

	SS	DF	MS	F	р
Trial	39.56	2	19.78	21.220	0.000*
Error (Trial)	20.51	22	0.93	·	.

Run Time ANOVA Summary Table

Note. * *p* < 0.05; n = 12.

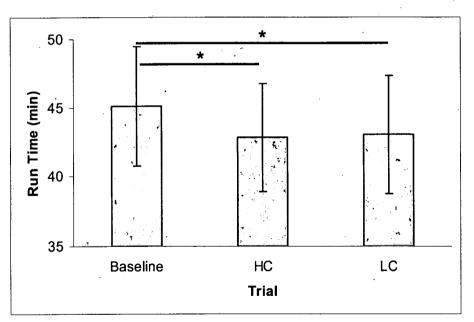


Figure 2

Mean and standard deviation for 10 km run times during all trials. Run time during the baseline trial was significantly (* p < 0.05) greater than run time for either the HC or LC trials.

Cycle Time

A one-way ANOVA was performed on cycle time to determine whether subjects demonstrated a difference in cycling performance among trials. Table 3 displays the results of this analysis, and shows that cycling time was similar for all trials ($F_{(2,22)} = 0.813$; p = 0.457). This was expected as the study protocol required a constant power output, and therefore speed, throughout all trials. As such, time taken to complete the 40 km cycle should not have varied between trials. Figure 3 shows mean cycle times.

Physiological Variables

Heart Rate

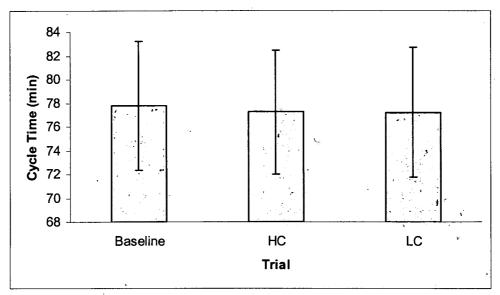
Time 1. A one-way ANOVA was performed on HR data at Time 1 to determine whether a significant between trial difference was seen for HR at this point. Table 4 shows the results of this analysis, which indicate that a significant difference existed between trials ($F_{(2,22)} = 14.702$; p < 0.05). Follow-up dependent t-tests identified that HR during the baseline trial was significantly greater than during the HC and LC trials at Time 1 (Figure 4).

Time 2 and 3. A 3 x 2 ANOVA (trial x time) with repeated measures on both factors was performed to detect statistically significant differences in HR for trials at Time 2 and Time 3. These results are outlined in Table 5, which shows a significant interaction ($F_{(2,22)} = 17.208$; p < 0.05). Post-hoc dependent t-tests indicate that, at Time 2, HR was significantly greater during both the baseline and HC trials when compared to the LC trial. At Time 3, HR during the HC trial was significantly greater than the LC trial. In other words, HR was lower throughout the transitional phase (i.e., Time 2 and

	SS	DF	MS	F	р
Trial	2.45	2	1.23	0.813	0.457
Error (Trial)	33.18	22	1.51		

Cycle Time ANOVA Summary Table

Note. n = 12.





Mean and standard deviation for 40 km cycle times during all trials.

The Trans And A Summary Table	HR	(Time 1)) ANOVA Summary Tab	le
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	SS	DF	MS	F	р
Trial	1109.06	2	554.53	14.702	0.000*
Error (Trial)	829.78	22	37.72		

Note. * p < 0.05; n = 12.

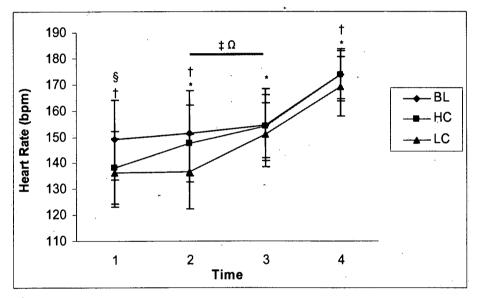


Figure 4

Mean HR values across four time points during each cycle-run trial. Significant differences (p < 0.05) between Trials are denoted as follows:

§ p < 0.05 between baseline and HC trials.

[†] p < 0.05 between baseline and LC trials. * p < 0.05 between HC and LC trials.

Significant differences (p < 0.05) between Time 2 and Time 3 are denoted as follows:

p < 0.05 during HC trial.

 $\Omega p < 0.05$ during LC trial.

HR (Time 2 and 3) 3 x 2 ANO	VA Summary Table
-----------------------------	------------------

			· · · · · · · · · · · · · · · · · · ·			
	SS	DF	MS	F	р	
Trial	1079.25	2	539.62	9.534	0.001*	
Error (Trial)	1245.16	- 22	56.60			
Time	1169.06	· 1 ·	1169.06	23.763	0.000*	
Error (Time)	541.17	11	49.20			
Trial*Time	366.64	2	183.32	17.208	0.000*	
Error (Trial*Time)	234.37	22	10.65			

Time 3) during LC compared to HC. Time-based post-hoc comparisons revealed that Time 2 HR was significantly lower than Time 3 during the HC and LC trials (Figure 4), meaning that running elevated HR above that measured during HC and LC cycling.

Time 4. A one-way ANOVA was performed on HR data at Time 4 to determine if a significant between trial difference was seen for HR at this point. The results of this analysis are displayed in Table 6, and indicate that a significant difference existed between trials ($F_{(2,22)} = 6.154$; p < 0.05). Follow-up dependent t-tests were performed and identified that HR during the baseline trial was significantly greater than during both the HC and LC trials (Figure 4). This was similar to the HR results at Time 1.

Oxygen Consumption

Time 1. A one-way ANOVA was performed on the VO₂ data at Time 1 to determine whether subjects experienced significant between trial differences at this point. The results of this analysis are displayed in Table 7, and indicate there was no significant difference in VO₂ between trials at Time 1 ($F_{(2,22)} = 2.500$; p = 0.105).

Time 2 and 3. A 3 x 2 ANOVA (trial x time) with repeated measures on both factors was performed to detect statistically significant differences in VO₂ between trials at Times 2 and 3. These results, outlined in Table 8, identify a significant interaction $(F_{(2,22)} = 5.785; p < 0.05)$. Post-hoc dependent t-tests indicated that VO₂ was significantly elevated at Time 2 during the HC trial when compared to the LC trial. At Time 3, VO₂ during the HC trial was significantly greater than the baseline trial, but was significantly lower than Time 3 during all trials (Figure 5). As with HR, VO₂ during running was greater than when measured during cycling.

	SS	DF	MS	F	р
Trial	161.23	2	80.61	6.154	0.008*
Error (Trial)	288.18	22	13.10		

HR (Time 4) ANOVA Summary Table

Note. * p < 0.05; n = 12.

Table 7

VO₂ (Time 1) ANOVA Summary Table

	SS	DF	MS	F	р
Trial	23.03	2	11.52	2.500	0.105
Error (Trial)	101.35	22	4.61	• 	

VO₂ (Time 2 and 3) 3 x 2 ANOVA Summary Table

	SS	DF	MS	F	, p
Trial	52.90	2	26.45	3.106	0.065
Error (Trial)	187.34	22	8.52		
Time	684.52	1	684.52	46.898	0.000*
Error (Time)	160.56	11	14.60		
Trial*Time	34.69	2	17.34	5.785	0.010*
Error (Trial*Time)	65.95	22	3.00		

Note. * p < 0.05; n = 12.

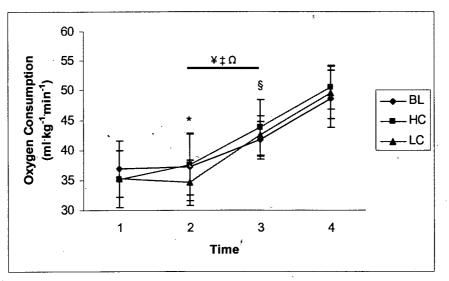


Figure 5

Mean VO_2 values across four time points during each cycle-run trial. Significant differences (p < 0.05) between Trials are denoted as follows:

§ p < 0.05 between baseline and HC trials.

* p < 0.05 between HC and LC trials.

Significant differences (p < 0.05) between Time 2 and Time 3 are denoted as follows:

¥ p < 0.05 during baseline trial.

p < 0.05 during HC trial.

 $\Omega p < 0.05$ during LC Trial.

Time 4. A one-way ANOVA was performed on VO₂ data at Time 4 to determine whether a significant between trial difference existed for VO₂ at this point. The results of this analysis are shown in Table 9, and indicate no significant difference for VO₂ between trials at Time 4 ($F_{(2,22)} = 3.176$; p = 0.061).

Ventilation

Time 1. A one-way ANOVA was performed on VE data at Time 1 to determine whether a significant between trial difference existed at this point. The results of this analysis are displayed in Table 10, and indicate there was no significant difference for VE between trials at Time 1 ($F_{(2,22)} = 3.440$; p = 0.051).

Time 2 and 3. A 3 x 2 ANOVA (trial x time) with repeated measures on both factors was performed to detect statistically significant differences in VE between trials at Times 2 and 3. Table 11 outlines this analysis, which identified a significant interaction $(F_{(2,22)} = 7.503; p < 0.05)$. Post-hoc dependent t-tests indicated that VE was significantly greater during the baseline and HC trials when compared to the LC trial at Time 2. At Time 3, however, the LC and HC VE were similar, although the HC VE was greater than baseline. The time-based post-hoc comparisons revealed that Time 2 VE was significantly lower than Time 3 during all trials (Figure 6), indicating that VE drifted upward during running at Time 3 compared with cycling at Time 2, as did HR and VO₂.

Time 4. A one-way ANOVA was performed on VE data at Time 4 to determine whether subjects experienced significant between trial differences in VE during the final 2 km of the three cycle-run trials. The results of this analysis are displayed in Table 12, and indicate there was no significant difference in VE at Time 4 ($F_{(2,22)} = 2.663$; p =0.092).

VO₂ (Time 4) ANOVA Summary Table

· · · · · · · · · · · · · · · · · · ·	SS	DF	MS	F	р
Trial	22.61	2	11.30	3.176	0.061
Error (Trial) [·]	78.29	22	3.56		

Note. * p < 0.05; n = 12.

Table 10

VE (Time 1) ANOVA Summary Table

	SS	DF	MS	F	р
Trial	108.44	2	54.22 ²	3.440	0.051
Error (Trial)	346.75	22	15.76		

	SS	DF	MS	F	р
Trial	252.30	2	126.15	3.838	0.037*
Error (Trial)	723.03	22	32.87		
Time	2488.88	1	2488.88	16.297	0.002*
Error (Time)	1679.97	11	152.72		
Trial*Time	189.72	2	94.86	7.503	0.003*
Error (Trial*Time)	278.14	22	12.64	• •	.

VE (Time 2 and 3) 3 x 2 ANOVA Summary Table

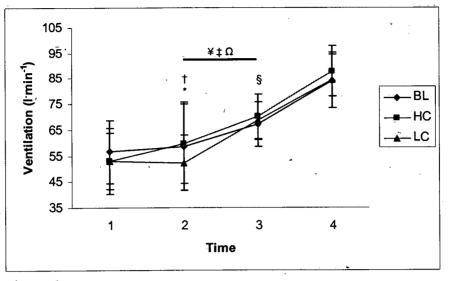


Figure 6

Mean VE values across four time points during each cycle-run trial. Significant differences (p < 0.05) between Trials are denoted as follows:

§ p < 0.05 between baseline and HC trials.

 $\frac{1}{p} < 0.05$ between baseline and LC trials.

* p < 0.05 between HC and LC trials.

Significant differences (p < 0.05) between Time 2 and Time 3 are denoted as follows:

 $\frac{1}{2} p < 0.05$ during baseline trial.

p < 0.05 during HC trial.

 $\Omega p < 0.05$ during LC trial.

Table 12

	SS	DF	MS	F	p
Trial	111.33	2	55.66	2.663	0.092
Error (Trial)	459.86	22	20.90		L

VE (Time 4) ANOVA Summary Table

Respiratory Exchange Ratio

Time 1. A one-way ANOVA was performed on RER data at Time 1 to determine whether a significant between trial difference existed at this point. The results of this analysis are displayed in Table 13, and indicate there was no significant difference in RER between trials at Time 1 ($F_{(2,22)} = 0.391$; p = 0.681).

Time 2 and 3. A 3 x 2 ANOVA (trial x time) with repeated measures on both factors was performed to detect statistically significant differences in RER between trials at these time points. These results, outlined in Table 14, identify a significant interaction $(F_{(2,22)} = 6.994; p < 0.05)$. Post-hoc dependent t-tests indicated that, at Time 2, RER was significantly greater during the HC trial than the baseline and LC trials. At Time 3, however, there was no significant difference in RER between trials. Time-based post-hoc comparisons revealed that RER at Time 3 was significantly greater than Time 2 during the baseline and LC trials (Figure 7). This result is consistent with the findings of other physiological variables (i.e., HR, VO₂ and VE).

Time 4. A one-way ANOVA was performed on RER data at Time 4 to determine whether a significant between trial difference existed at this point. These results are shown in Table 15, and indicate that a significant difference existed between trials ($F_{(2,22)}$ = 5.849; p < 0.05). Post-hoc dependent t-tests indicated RER was significantly greater during HC than baseline and LC at Time 4 (Figure 7).

Rating of Perceived Exertion

Time 1. A one-way ANOVA was performed on RPE data at Time 1 to determine whether a significant between trial difference was experienced at this time. These results are displayed in Table 16, and indicate that a significant difference existed between trials

RER (Time 1) ANOVA Summary Table

· ·	SS	DF	MS	F	p
Trial	0.000	2	0.000	0.391	0.681
Error (Trial)	0.008	22	0.000		

Note: * p < 0.05; n = 12.

Table 14

RER (Time 2 and 3) 3 x 2 ANOVA Summary Table

	F		· · ·		
	SS	DF	MS	F	ŕ
Trial	0.001	2'	0.001	3.151	0.063
Error (Trial)	0.005	22	0.000		·
Time	0.018	1	0.018	8.593	0.014*
Error (Time)	0.023	11	0.002		
Trial*Time	0.002	2	0.001	6:994	0.004*
Error (Trial*Time)	0.003	22	0.000		

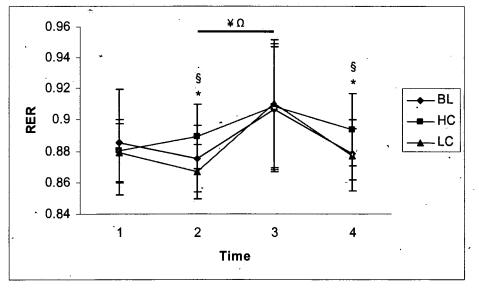


Figure 7

Mean RER values across four time points during each cycle-run trial. Significant differences (p < 0.05) between Trials are denoted as follows:

* p < 0.05 between baseline and HC trials.

§ p < 0.05 between HC and LC trials.

Significant differences (p < 0.05) between Time 2 and Time 3 are denoted as follows:

¥ p < 0.05 during baseline trial. $\Omega p < 0.05$ during LC trial.

Table 15.

······································	SS	ĎF	MS	F	р
Trial	0.002	2	0.001	5.849	0.009*
Error (Trial)	0,004	22	0.000		

RER (Time 4) ANOVA Summary Table

.t ₂ .	SS	DF	MS	F	р
Trial	5.06	2	· 2.53	4.529	0.023*
Error (Trial)	12.28	22	0.56		

RPE (Time 1) ANOVA Summary Table

 $(F_{(2,22)} = 4.529; p < 0.05)$. Follow-up dependent t-tests identified that RPE during the baseline trial was significantly greater than during HC and LC trials (Figure 8).

Time 2 and 3. A 3 x 2 ANOVA (trial x time) with repeated measures on both factors was performed to detect statistically significant differences in RPE between trials at these time points. Results are outlined in Table 17, and show a significant interaction $(F_{(2,22)} = 4.013; p < 0.05)$. Post-hoc dependent t-tests indicated that, at Time 2, RPE was significantly greater during the baseline and HC trials than during the LC trial. At Time 3, however, RPE during the HC trial was significantly greater than the baseline trial. In this regard, it can be seen that RPE results are consistent with several other physiological variables (i.e., HR, VO₂, VE and RER). Time-based post-hoc comparisons revealed Time 2 RPE was significantly lower than Time 3 during the LC trial (Figure 8), which mimics the physiological drift reported for most variables measured during running.

Time 4. A one-way ANOVA was performed on RPE data at Time 4 to detect a significant difference in RPE at this point. The results of this analysis are displayed in Table 18, and indicate there was no significant difference in RPE between trials at Time 4 $(F_{(2,22)} = 0.846; p = 0.443)$. In other words, subjects felt similarly during all conditions despite some physiological differences between trials by Time 4 (e.g., HR and RER).

Lactate

Time 1. A one-way ANOVA was performed on lactate data at Time 1 to determine whether subjects experienced significant between trial differences at this point. The results of this analysis are displayed in Table 19, and indicate there was no significant difference in lactate between trials at Time 1 ($F_{(2,22)} = 1.182$; p = 0.326).

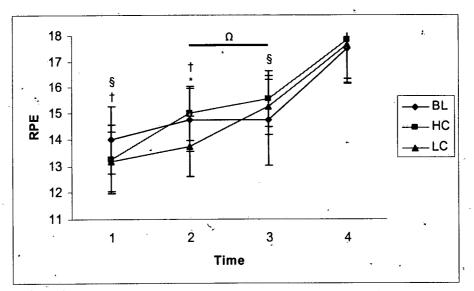


Figure 8

Mean RPE values across four time points during each cycle-run trial. Significant differences (p < 0.05) between Trials are denoted as follows:

§ p < 0.05 between baseline and HC trials.

† p < 0.05 between baseline and LC trials. * p < 0.05 between HC and LC trials.

Significant differences (p<0.05) between Time 2 and Time 3 are denoted as follows:

 $\Omega p < 0.05$ during LC trial.

Table 17

RPE (Time 2 and 3) 3 x 2 ANOVA Summary Table

	SS	DF	, MS	· F	р
Trial	7.86	2	3.93	5.712	0.010*
Error (Trial)	15.14	22	0.69		
Time	8.68	1	8.68	9.405	0.011*
Error (Time)	10.15	11	0.92		
Trial*Time	6.86	2	3.43	4.013	0.033*
Érfor (Trial*Time)	18.81	22	0.86		

Note. * p < 0.05; n = 12.

· <u>• • •</u>	SS	DF	MS	F	р
Trial	0.67	2	0.33	0.846	0.443
Error (Trial)	8.67	22	0.39		

RPE (Time 4) ANOVA Summary Table

Note. * p < 0.05; n = 12.

Table 19

Lactate (Time 1) ANOVA Summary Table

	SS	DF	MS	F	р
Trial	11.01	• 2	5.51	1.182	0.326
Error (Trial)	102.52	22	4.66		

Note. * *p* < 0.05; n = 12.

۶. ج *Time 2 and 3.* A 3 x 2 ANOVA (trial x time) with repeated measures on both factors was performed to detect statistically significant differences in lactate between trials at times 2 and 3. These results are outlined in Table 20, and show no significant interaction ($F_{(2,22)} = 1.343$; p = 0.282). However, a significant main effect difference was found for both trial ($F_{(2,22)} = 4.299$; p < 0.05) and time ($F_{(2,22)} = 8.995$; p < 0.05). Posthoc dependent t-tests on the trial-based main effect indicated significantly lower lactate values during the LC trial than measured during the baseline trial (Figure 9). The timebased main effect indicated a significantly greater lactate during the run (i.e., Time 3) than during cycling (i.e., Time 2) (Figure 9).

Time 4. A one-way ANOVA was performed on lactate data at Time 4 to determine whether a significant difference in lactate existed at this point. The results of this analysis are displayed in Table 21, which indicate no significant difference in lactate values between trials at Time 4 ($F_{(2,22)} = 0.872$; p = 0.432).

Lactate (Time 2 and 3) 3 x 2 ANOVA Summa	arv Table
--	-----------

•				••	
•	SS	DF	MS	F	р
Trial	55.09	2	27.54	4.299	0.027.*
Error (Trial)	140.95	22	6.41	•	
Time	57.96	1	57.96	8.995	0.012*
Error (Time)	70.88	11	6.44		
Trial*Time	14.68	2	7.34	1.343	0.282
Error (Trial*Time)	120.19	22	5.46		

Note. * p < 0.05; n = 12.

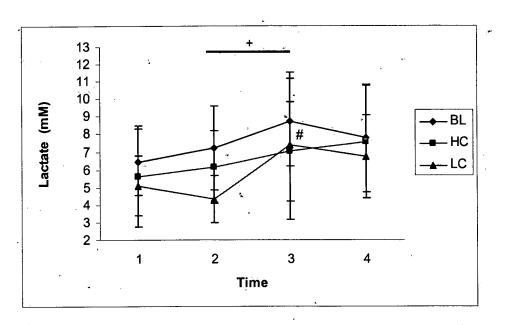


Figure 9

Mean lactate values across four time points during each cycle-run trial.

- # Significant main effect (p < 0.05) between baseline and LC Trials.
 - + Significant main effect (p < 0.05) between Time 2 and Time 3.

٤.

Table 21

	· · · · · · · · · · · · · · · · ·			•	
	SS	DF	MS	F	р.
Trial	7.59	2	3.79	0.872	0.432 .
Error (Trial)	95.67	22	4.35	,	•
-					

Lactate (Time 4) ANOVA Summary Table

Summary

The results of these analyses, outlined in Table 22, indicate many differences in physiological variables between trials across time. A close look at these data reveals several trends that can be identified from this study. With the exception of two instances (HR and RPE at Time 1), the direction of significant differences for all variables at all time points was consistently HC > baseline > LC. This indicated that utilization of the HC strategy was generally more physiologically demanding than the baseline trial, which itself was more physiologically demanding than the LC strategy. Additionally, timebased comparisons saw all physiological variables significantly increase during the transition phase (i.e., from Time 2 to Time 3) during the LC trial, while the HC and baseline trials saw significant increases at these times in only three physiological variables. This may indicate that the LC strategy is a less physiologically demanding, and therefore more energetically efficient cycling strategy, but also that the HC and baseline strategies require the athlete to make a smaller physiological 'jump' to begin running. Lastly, all physiological variables exhibited significant differences between Time 2 and Time 3, while Time 1 and Time 4 each saw differences in only two physiological variables. This may provide a physiological basis for the common complaint that transitioning from cycling to running is a particularly challenging aspect of triathlon.

Summary of Significant Findings.

	<u> </u>	T1	, T2	T3	T4
Heart Rate	Trial	· §†	+ *	*	† *
	T2 - T3		‡	Ω	
Oxygen Consumption	Trial		*	Ş	
	T2 - T3		¥ţΩ		
Ventilation	Trial		† *	§	
	T2 - T3		¥‡	Ω	
RER	Trial		§ *		§ *
	T2 - T3		¥	Ω	
RPE	[′] Trial	§ †	† *	§	
	T2 - T3		2	2	
Lactate	Trial	<u> </u>	i	4	
	T2 – T3		-	+	14 64

Note: Differences across Trials are denoted as follows:

§ HC > baseline (p < 0.05);

§ baseline > HC (p < 0.05)

- † baseline > LC (p < 0.05);
- * HC > LC (p < 0.05).

Differences across Time 2 and Time 3 are denoted as follows:

 $\pm T3 > T2 (p < 0.05)$ during baseline trial;

- $T_3 > T_2 (p < 0.05)$ during HC trial;
- Ω T3 > T2 (p < 0.05) during LC trial.
- # Lactate main effect difference where baseline > LC (p < 0.05);

+ Lactate main effect difference where T3 > T2 (p < 0.05).

Chapter 5

DISCUSSION

The primary focus of this study was to investigate the effect of increasing or decreasing cycling cadence during the final third of a 40 km cycling bout on physiological effort during the cycle-run transition of simulated duathlon trials, as reflected by differences in physiological responses recorded during trials. The secondary purpose was to determine if this cadence alteration influenced subsequent 10 km run time. The principle finding was that utilizing a higher than preferred cadence (HC)during the last third of the cycle bout minimized the increase in physiological response during the cycle-run transition period. However, when compared to cycling at a preferred (PC) or lower than preferred cadence (LC) during the final third of a 40 km cycling bout, HC elevated both the cycling energy requirement and perceived work effort (RPE, which may be interpreted as both a physiological and psychological variable). In other words, the LC intervention improved economy during the last third of the cycle bout, but resulted in a greater increase in physiological effort during the cycle-run transition relative to the HC. Neither cadence strategy, however, significantly influenced 10 km run time.

Run Performance

Deviating from PC during the final stages of the triathlon cycle bout can potentially exert both positive and negative effects on subsequent run performance. Gottschall and Palmer (2002) found that a higher than preferred cadence strategy improved run performance, whereas Bernard et al. (2003), Vercruyssen et al. (2002), and

Vercruyssen et al. (2005) saw run performance improve only after a lower cycling cadence strategy. However, these studies differed in how they operationally defined run performance. The first method, utilized by Gottschall and Palmer (2002) and Bernard et al. (2003), measured the time to run a given distance. The second, used by Vercruyssen et al. (2002), measured the distance run in a given time frame, and the third, used by Vercruyssen et al. (2005), measured run time to fatigue. The latter protocol is openended, whereas the others are close-ended. Close-ended tests have been shown to better simulate racing performance by realistically approximating the stress of competition (Jeukendrup, Saris, Brouns, and Kester, 1996). As such, this study utilized a close-ended. Olympic distance run protocol, and is fairly compared to studies using a similar closedended protocol. The present data showed that baseline (i.e., PC) run time was slower than HC and LC run times, which were similar. As such, these data do not corroborate the findings of either Gottschall and Palmer (2002) or Bernard et al. (2003). The inconsistency in data among the studies may be attributed to a number of factors related to protocol design.

Influential Factors

Subjects' lack of familiarity with the present study protocol as well as performing in laboratory conditions may have influenced study results. Although all subjects had prior notification of the study protocol both verbally and in writing, many said they did not know what to expect during the baseline trial. As such, several subjects later claimed they ran slower than race-pace during baseline due to anxiety. Although no data were collected to quantify anxiety, this mindset may have caused the average baseline trial run time to be more than two minutes slower than the other two 10 km trial runs. Several

studies recommend familiarity trials to ensure subjects feel comfortable using laboratory equipment. Lauren, Shing, and Jenkins (2003) found well-trained cyclists could perform a consistent 40 km time trial on a stationary wind-trainer if they first performed a familiarization trial. Similarly, Lavcanska, Taylor, and Schache (2005) found 6 min of treadmill running was adequate for subjects to produce a consistent running pattern (their operational definition of treadmill familiarity). However, these findings are based simply on equipment familiarity, whereas the subjects in this study were also anxious about performing in a laboratory environment. As such, they may have benefited from an entire cycle-run familiarity trial. However, the protocol already required subjects to commit to 10 hours of laboratory testing, and further commitment may have negatively influenced participation.

Treadmill pacing may have provided another influence on run performance in a way not possible in an actual race. Many subjects described themselves as highly competitive. Some mentioned that as they became familiar with the trial protocol, they chose to keep with the set treadmill pace (as opposed to manually decreasing treadmill speed) when it was likely they would have slowed down during a race.

It is also possible that subjects realized a noticeable training effect as a result of participation in this study, which may further explain slower baseline trial run time. Subjects were requested to maintain their current level of training throughout data collection, which ranged from two to four weeks. However, many did not routinely perform an Olympic distance cycle-run bout as part of their regimen. As such, the run time posted during the third cycle-run trial was significantly faster (p < 0.05) than the second. Timing of data collection could also have been a contributing factor, as it was

carried out from the beginning of October to mid-December, which is the post-season for those who participate in summer triathlon events.

In summary, the present study showed poorer run time on the first trial compared to either the HC or LC, although there was no difference between the HC and LC run times. A number of reasons related to familiarity or training may explain that finding, but unlike previous studies (Bernard et al., 2003; Vercruyssen et al., 2002) the present study did not find differences between HC and LC. Although altering cycling cadence did not influence overall 10 km run time, it appeared that cycling at HC was more physiologically and psychologically demanding than PC, and PC was more physiologically and psychologically demanding than LC.

Cycle Performance

Lack of protocol familiarity may have been an influential factor in baseline run performance, but as cycling work was kept constant for all three trials, the influence of cycle protocol familiarity is neither known nor of consequence. Each cycle bout was controlled to ensure a similar average PO for all three trials; hence, time to perform each cycle bout did not differ. As such, subjects had performed a similar amount of work by the beginning of each run bout. Consistency in overall cycling PO was also ensured by Vercruyssen et al. (2005) and Bernard et al. (2003), although Gottschall and Palmer (2002) did not clearly specify controlling PO.

The physical response presently seen in the various cadence conditions was comparable to that reported during cycling by Bernard et al. (2003), Vercruyssen et al. (2002) and Vercruyssen et al. (2005). This is logical because all these studies varied cycling cadence in a similar fashion. Bernard et al. (2003) and Vercruyssen et al. (2005)

found that higher cadence trials were more physiologically stressful than lower cadence trials. Bernard et al. (2003) found high cadence elevated VE, HR and blood lactate, whereas Vercruyssen et al. (2005) reported significantly greater VO₂, VE, HR and blood lactate during the final two minutes of cycling. Similarly, Vercruyssen et al. (2002) found VO2 during both high and preferred cadence bouts to be greater than a low cadence bout. The results of these three studies are consistent with the present study. During the final 2 km of cycling, five of the six physiological variables (all but lactate) assessed during the HC cycle bout were significantly greater than measured during LC. PC trial HR, VE and RPE were also higher than during the LC bout, and RER was higher during the HC bout than the baseline bout. No other known studies have evaluated RPE, which is an individual's subjective evaluation of work effort. As such, this study provides the novel finding that PC and HC strategies may be perceived as more difficult to perform than LC. Therefore, regardless of objective evaluation of the physical response to either cadence intervention, a LC strategy may be preferable unless a HC or PC strategy results in an improved triathlon run time. Cycling efficiency was also evaluated at Time 2, and was calculated in terms of PO / VO2. It was revealed that efficiency at the end of the LC cycle bout was significantly greater (p < 0.05) than at the end of the HC cycle bout. Therefore, to maintain a given cycling PO (and therefore cycling speed), utilizing a higher than preferred cadence was more physiologically demanding, and therefore less energetically efficient, than utilizing a less than preferred cadence. This finding agrees with results reported by Bernard et al. (2003), Vercruyssen et al. (2002) and Vercruyssen et al. (2005). Based on these findings, it may be further hypothesized that a lower than preferred cadence strategy may allow the athlete to maintain a greater PO (i.e., PO is not

held constant), and thereby achieve a faster 40 km cycle time. However, the consequence of implementing such a strategy on subsequent run performance is unknown. It is speculated that the local muscle fatigue that can accompany such a low cadence strategy may negatively influence the triathlete during the run.

Cycle-Run Transition

The present physiological data related to running after cycling at varying cadences agree with the findings of Vercruyssen et al. (2002), but not with those by Vercruyssen et al. (2005) and Bernard et al. (2003). In the current study, HR, VO₂, VE and RPE measured during the first 8 min of running (approximately 2 km) subsequent to the HC cycle bout were significantly greater than at the same time during the other two trials. Similarly, Vercruyssen et al. (2002) reported that cycling at either a preferred or higher than preferred cadence resulted in a significantly greater VO_2 during the final 7 min of a 12 min run bout than seen during the same period following a lower than preferred cadence cycling bout. Conversely, Vercruyssen et al. (2005) found no difference between high, preferred or low cadence trials in overall VO₂, VE, HR or lactate during a run to fatigue. However, Vercruyssen et al. (2005) stated that running VO_2 at the beginning (between minutes 3 and 10) of each run bout was significantly greater than overall running VO_2 during each run bout. In other words, VO_2 decreased after 10 min of running. This was surprising, as it implies that trials allowed subjects to reach steady state oxygen uptake, which would not be expected during a high intensity (85% maximum velocity) run to fatigue.

According to the present results, lower cycle cadence never yielded more physiologically or psychologically demanding responses than preferred or higher cycle

cadence. In fact, the LC trial was never more demanding than either the PC or HC trials at any measured time point. This is in direct contrast to Bernard et al. (2003), who found greater running VO₂ after the lower than preferred cadence cycle bout (20 min at 60 rpm) than the two other trials after subjects had run 1 km. The basis of this difference may lie in cycling intensity. Average (mean \pm *SD*) cycling PO maintained during the Bernard et al. (2003) study ranged from 275.4 \pm 19.1 W for the 60 rpm trial, to 277.2 \pm 17.2 W for the 100 rpm trial, whereas the subjects in this study averaged 172.75 \pm 27.48 W across all trials. Although the cycle time in Bernard et al. (2003) was less than a third of that required presently, their subjects worked at a much greater intensity. As such, they would have used a higher force during the 60 rpm trial, which may have resulted in greater local muscle fatigue. This fatigue would have required the recruitment of additional muscle fibers to perform the same running work, thus elevating running VO₂.

However, the LC trial in this study was less physiologically demanding, and therefore more energetically efficient, than the HC trial. Thus, it may be fair to state that a LC strategy may provide a physiological advantage, as it is desirable to conserve energy while cycling before the demanding run stage. Given this, it is logical to speculate that 10 km run time would be faster following cycling bouts utilizing slower cadences, however, this did not occur in the present study. A number of factors may have influenced this result. Lack of protocol familiarity may have caused the average cycling PO during the first trial to be lower than normal race-pace. Average VO₂ measured at Time 2 was 60% of average VO_{2max}. This percentage is considerably lower than a similar comparison made by Zhou, Robson, King, and Davie (1997), who found average HR while cycling in a competitive triathlon was 92% of cycling HR_{max}. It is acknowledged that the competitive triathlon was shorter (30 km cycle, 8 km run) than present protocol, which would potentially facilitate an elevated average cycling PO. Regardless, the difference in cycling intensity between the present study and Zhou et al. is substantial. Later cycle bouts would also have been performed at this lower intensity, which may have attenuated the physiological effect of the cycling interventions during both cycling and the subsequent run bout. The study protocol also may have allowed subjects to be paced by the treadmill, rather than requiring subjects to alter treadmill pace in response to the physiological effort required by the preceding cycle bout.

Practical Application

It is proposed that the triathlon cycle-run transition may be analyzed using two different approaches. Further, the determination of the most suitable approach should depend, in part, on the goal of implementing a cadence manipulating strategy. The first approach is from an energy conservation standpoint. Because an LC strategy was more energy efficient than HC, it may favor lesser-trained competitors who may be more likely to fatigue toward the end of the event. However, the LC strategy did result in a larger jump in energy demand once running commenced. In fact, once subjects had run for 8 min after the LC cycle bout, only one variable (HR) remained lower when compared to the same period during the HC trial. In other words, after running for 8 min following the LC cycle bout, physiological demand had caught up to that required after the HC cycle bout. This leads to the second approach, which, in keeping with the purpose of this study is from a 'transition minimization' standpoint. While it is acknowledged that the HC cycle bout was not as economical as the LC bout, it could be hypothesized that it more closely mimicked the energy requirements of running. This was graphically

demonstrated in Figures 4 - 9, where it can be seen that the slope of the line connecting Time 2 to Time 3 (for all physiological variables) during the HC trial is always smaller than during LC trial. As such, a smaller difference in the physiological effort required to cycle and run during the HC trial could justifiably be interpreted as smaller, possibly less intense, cycle-run transition. However, it is also possible that rather than choosing to conserve energy while utilizing a LC strategy, triathletes may opt to increase PO and cycle faster, resulting in elevated physiological and psychological effort. As such, the physiological jump in energy required to begin running may be attenuated, although the increased muscle fiber recruitment necessary to generate this PO may result in quicker muscle fatigue during running. However, energy efficiency is not the only aspect involved in minimizing the cycle-run transition. Although not measured in this study, several studies have quantified changes in stride length and frequency following variedcadence cycle bouts. Gottschall and Palmer (2002) attributed an improvement in 3200 m run performance after higher cadence cycling to increased stride frequency. Conversely, Bernard et al. (2003) did not see an overall increase stride frequency subsequent to cycling at 60, 80 or 100 rpm, but did report that stride frequency was significantly higher during the first 500 m following the 80 and 100 rpm bouts. This was attributed to the existence of a direct relationship between cycling cadence and initial stride frequency. Although these additional findings may confuse the matter, to assume that energy efficiency is the only influence on the cycle-run transition would, at best, be an oversimplification. As such, further research on these topics is required.

Lay triathlon publications advocate maintaining cycling cadence between 85 – 95 rpm (Cycling Cadence, n.d.; Scott, 2006; Mierke, 2005), as this mimics average running

cadence of approximately 90 rpm (Mierke, 2005). The proportion of fast and slow twitch muscle fibers possessed by an athlete may influence whether they prefer to cycle at a low (e.g., 60 rpm) or a high (e.g., 95 rpm) cadence, respectively. However, by definition, most novice athletes do not possess the aerobic capacity to maintain high cadences while generating race-pace PO. Conversely, experienced athletes generally have a more developed cardiovascular system which can deliver oxygen and remove metabolic waste products more effectively. As such, it is speculated that an optimal triathlon cadence strategy may vary according to training status and maybe only highly trained triathletes would benefit from using a high cadence strategy. Future studies should be designed to examine the interaction between training status and cadence strategies in triathlon.

Summary

The results of this study indicated that manipulating cycling cadence may not be an effective means of improving triathlon 10 km run time. However, adopting a higher or lower than preferred cycling cadence during the final third of a 40 km cycle bout was shown here to influence physiological and psychological variables during the cycle-run transition and some for the duration of the trial. However, the most profound influence was seen during the cycle-run transition, where all variables were affected across trials, across time, or both. Whether the influence of each cadence strategy is ultimately a positive or negative influence is debatable. This paper presents two methods of assessing the influence of cadence on the cycle-run transition, and the strategy that best suits each athlete may depend on factors such as genetics, training status, current preferred cadence, and the goal of implementing such a cadence intervention.

Chapter 6

SUMMARY, CONCLUSIONS & RECOMMENDATIONS

Summary

This study investigated the effect of HC and LC cycling during the final third of a 40 km cycle bout on the physiological effort of cycling, the physiological response during the cycle-run transition, and subsequent 10 km run performance. Several previous studies have investigated the effect of HC and LC strategies on cycle and run performance, but did not utilize protocols that reflected realistic triathlon distances. Twelve trained triathletes, 11 males and 1 female, were recruited from the Ithaca Triathlon Club. Subjects completed a VO_{2max} running test to assess aerobic capacity, followed by three 40 km cycle / 10 km run trials, with at least four days separating each trial. The first cycle-run trial was a baseline performance to quantify preferred cycling cadence (PC) and average cycling power output (PO), as well as subsequent 10 km run time. The second and third cycle-run trials, performed in a counter-balanced order, either increased (HC) or decreased (LC) cycling cadence by 20% from PC during the final third of the cycle bout. PO was kept constant during HC and LC trials. The effect of cadence interventions was measured by 10 km run time and the response of six physiological variables (HR, VO₂, VE, RER, RPE and lactate), which were collected four times (twice while cycling and twice while running) during each of the three trials. Data collected at Time 1 (27 - 28 km cycling) were to assess whether energy expenditure until then was consistent across trials. One-way repeated measures ANOVA analysis of Time 1 data found that two physiological variables (HR and RPE) were significantly greater during the baseline trial than during the HC and LC trials. The period between Time 2 (38 - 40)

km cycling) and Time 3 (1 – 8 min running) was defined as the cycle-run transition. A 3 x 2 (trial x time) ANOVA with repeated measures on both factors was used to analyze all dependent variables to evaluate the effect of the cycling intervention during the cycle-run transition. Five of the six physiological variables (all but lactate) measured during HC were significantly greater than LC during Time 2, but only HR during HC remained significantly greater than LC by the end of Time 3. During LC, five physiological variables measured during the final 2 km of cycling were significantly lower than when measured after the first 8 min of running. During baseline and HC, three physiological variables (VO₂, VE, and RER; and HR, VO₂ and VE, respectively) were significantly lower the final 2 km of cycling than after the first 8 min of running, however, the difference between these two time points for each variable was always greater during LC than either baseline or HC.

Conclusions

Data from this study support the following conclusions:

- Cycling at a LC during the final third of a 40 km cycle bout while maintaining PO requires less physiological and psychological effort, and is more energy efficient, than cycling at PC or HC.
- Cycling at a HC during the final third of a 40 km cycle bout while maintaining PO results in smaller physiological differences between the cycling and running legs of a triathlon. This may be interpreted as lessening the physiological effort of the cycle-run transition.
- 3. Cycling at a HC or LC during the final third of a 40 km cycling bout while maintaining PO does not differentially influence 10 km run performance time.

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4. Cycling at HC and LC both resulted in improved run time when compared to the baseline trial. However, this finding was believed to be an artifact related to subject lack of familiarity with the testing protocol during the first (i.e., baseline) trial.

Recommendations

Study in the following areas may further explain the influence of cadence manipulation on both subsequent run performance and the cycle-run transition:

- 1. The effect of varying cycle cadence on the cycle-run transition and running performance over an Olympic triathlon distance should measure elite and recreational triathletes separately, as the physiology of these groups differs, and different cadence manipulation strategies may be better suited to each group.
- 2. The effect of various familiarization techniques, with the intention of identifying strategies facilitating protocol and laboratory familiarity, while considering economy of time.
- 3. The effect of manipulating cadence for a lesser period of time (e.g., during the last 8th of a 40 km cycle bout), as this may still provide the athlete with a physiological advantage, and is a closer reflection of current competition strategy. The cycle protocol utilized in this study (i.e., varying cadence during the final third of the cycle bout) was chosen due to its similarity to protocols utilized in previous comparable studies. However, altering cadence for this length of time may not be necessary for providing a transition-reducing or energy-saving benefit.
- The effect of utilizing a LC strategy for more than one third of the cycle bout on both the cycle-run transition and subsequent 10 km run performance, as this

strategy has consistently been shown to be more energetically efficient than the HC strategy.

- 5. The effect of cadence strategy to preferred cadence, to potentially identify upper and lower boundaries of effective cycling cadence. It is likely that there are upper and lower boundaries for cycling cadence efficiency, however, these boundaries have not been identified by quantitative research. An athlete who already maintains a high cycling cadence may not benefit from a faster strategy, in a similar way that one who naturally maintains a low cadence may not benefit from a slower strategy.
- 6. The effect of allowing cycling PO to be freely-selected, while varying cycle cadence as per the present manipulation strategy, on the time to cycle and run fixed distances.
- 7. The effect of allowing cycling PO to be freely-selected, while varying cycle cadence as per the present manipulation strategy, on the time taken and distance run during the cycle-run transition.

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APPENDIX A

Informed Consent Form

The Effect of Cycling Cadence on the Cycle-Run Transition in Triathletes

1. Purpose of the Study: The cycle-run transition ('transition phase') in triathlon is a source of much awkwardness and inefficiency for competitors. In an effort to minimize this stage, the effect of altering cycling cadence toward the end of the cycle leg has recently been investigated. Altering cadence is said to influence the activation of the working musculature (primarily the quadriceps), thus affecting running performance. However, the best way to manipulate cadence is unknown. Some studies have found that decreasing cadence resulted in the best subsequent run performance, whereas others saw better results from an increased cadence. The purpose of this study is to identify the better approach.

2. Benefits: You will benefit from participating in the study because you will become familiar with current research into triathlon racing strategy, as well as be informed of your maximal effort test results. You will also find which racing strategy (either increasing or decreasing cadence during the final 13km of cycle leg) is more effective in improving overall race time.

3. Your Participation requires you to be between 18 and 45 years old, and have performed a 40km cycle and 10km run during the previous three months. You will report to the lab on four non-consecutive days. On Day One you will perform a running VO_2 max test, and you will be given written instructions on how to come prepared for this test. For the warm-up, you will run on a treadmill for 5 minutes. During the warmup and the test, a fan will cool you. After the warm-up, you will be fitted with headgear, which will hold a mouthpiece that is attached to a hose from which expired ventilatory gases (VO_2) will be measured. You will also wear a nose clip. These are maximum effort tests that have you exercising less than 20 minutes. The initial running warm-up pace will be gender relative, and the test will see the treadmill speed or grade periodically increase until you request that the test ends. When each test is complete, you will cool-down for 5 minutes. After this, one of your fingertips will be sterilized and pricked to obtain a blood sample for lactate analysis.

The baseline test will require you to complete a 40km bike ride immediately followed by a 10km treadmill run. This is to determine, as realistically as possible, your preferred cycling cadence (PC) under race conditions, as well as the cycle and run split times, and overall performance time. HR will be continually recorded. Your rating of perceived exertion (ie indicating on a scale how hard you think you are working: RPE), blood lactate and VO₂ will be measured at several points throughout the trial. During the running bout, HR will be continually recorded, and RPE and VO₂ will be periodically recorded. Another blood sample will be obtained for lactate analysis as described above. Time to complete the entire cycle-run bout will be recorded, and the cycle and run split times will be noted.

The experiment will also require you to perform two trials in random order. Both trials will require you to complete a 40km bike ride immediately followed by a 10km treadmill run. Only the cycle portion of the trials differ, such that trial 1 requires you to increase your PC by 20% (PC+20%) during the last 13km of the cycle leg, and trial 2 requires a decrease in PC to PC-20% during the last 13km. The order in which you will perform these trials will be randomized. The baseline and trial tests are each estimated to last $2 - 2\frac{1}{2}$ hours. The VO₂max tests will last approximately 45 minutes. Total participation time for the project is 6-7 hours.

4. Risks of Participation: The risks involved in this project are probably no greater than the risks you freely assume when you train or race, especially during maximal efforts. These risks include skeletal muscle injury and possibly a cardiac event, which could be fatal. The chances of a cardiac event are low in your fitness group. The fingertip that is lanced may be tender for a few days. To minimize the risks, you will warm-up and cool-down before and after each test and training session. If you feel poorly during the test or training session, you may terminate it at any time. In the event that there is an injury or cardiac event, standard first aid procedures will be promptly administered. I will call 911 to seek additional assistance if warranted.

5. Compensation for Injury: If you suffer an injury that requires any treatment or hospitalization as a direct result of this study, the cost of such care is your responsibility. If you have insurance, you may bill your insurance company. Ithaca College and the investigator will not pay for any care, lost wages, or provide other compensation.

6. If you would like more information about this study at anytime prior to, during, or following the data collection, you may contact Sharon Fitzgerald at sfitzge1@ithaca.edu or 607-351-5759, or Dr Tom Swensen at tswensen@ithaca.edu or 607-274-3114.

7. Withdrawal from the study: Participation in this study is voluntary and you may withdraw at any time if you so choose. You will not be penalized for withdrawing.

8. Confidentiality: Information gathered during this study will be maintained in complete confidence. Only Dr Swensen and I will have access to this information, which will be stored in a locked cabinet in room 320 in the Center for Health Sciences at Ithaca College or on password protected computer. You and your name will never be associated with this information in any future disclosures.

I have read and understood the above document. I agree to participate in this study and realize that I can withdraw at anytime. I also understand that I can and should address questions related to this study at any time to Sharon Fitzgerald. I also verify that I am at least 18 years of age.

Your Name (please print)

Your Signature

Date

APPENDIX B

Medical History and Health Habit and 24 hour Recall Questionnaire

 Name:

 Weight:

Age:_____ Sex:_____

1. Medical/Health History: Check if you ever had?

Heart disease/ Stroke	
Heart Murmur	
Skipped, rapid beats, or irregular	
heart rhythms	
High blood Pressure	
Lung Disease	
Epilepsy	
Injuries to back, hips, knees, ankles,	
or feet	

Other conditions/comments:

Present Symptoms: Check within the box if you have you had these symptoms within the last 6 months?

Chest Pain	
Shortness of Breath	
Lightheadedness	
Heart Palpitations	
Loss of Consciousness	
Illness, surgery, or hospitalization	
Ankle/Leg swelling	
Joint/muscle injury requiring medical	
treatment	
Allergies (if yes please list under	
comments)	

Other conditions/comments:

2. Exercise habits:

What kind of exercise do you do? (circle one)

Aerobic Strength Training

How hard do you exercise? (circle one)

Easy Moderate (can carry on a conversation)

How many times a day do your work out?

How many days a week do you work out?

3. Have you consumed alcohol in the last 12 hours? (circle one)

Yes

No

4. Have you used caffeine (e.g., coffee) or nicotine (e.g., cigarettes) in the last 30 min? (circle one)

Yes

No

5. Did you eat any food in the last 30 min? (circle one)

Yes

No

6. Did you exercise before coming in to be tested? (circle one)

Yes

No

Both

Hard (can't carry on a conversation)

APPENDIX C

Maximal Oxygen Consumption Test: Pre-test Instructions

Test date:_____

Test Time:

You are scheduled to complete a maximum effort exercise test; your performance depends upon adherence to these instructions:

- 1. Do not perform heavy exercise in the 24 hours preceding your test.
- 2. Do not drink alcohol for 12 hours preceding your test.
- 3. Do not use caffeine (e.g. coffee) or nicotine (e.g. cigarettes) for 3 hours preceding your test.
- 4. Do not eat for 3 hours preceding the test.
- 5. Do not eat any food that may cause you discomfort the day of the test.
- 6. Avoid over-the-counter medications for the 12 hours preceding the test. (However, cancel appointment if you are ill and treat yourself accordingly; we can always reschedule).
- 7. Bring your running and cycling gear.
- 8. Bring a change of clothes and food and sport drink for after the test.

Thank you for your cooperation.

APPENDIX D

Triathlon Study Information

Where Do I Go?

All testing will take place at Ithaca College – 953 Danby Rd, Ithaca NY. The testing lab is in the Center for Health Sciences (CHS) Exercise Physiology lab (Rm 303a – level 3).

CHS is building 24 on the below link. http://www.ithaca.edu/map/index.php

Parking

If your trial is between 8am and 5pm Monday – Friday, please read on – parking is restricted only during these times. You're free to park in Lot F at any other time.

If you're scheduled between 8am-5pm M-F and you've passed on your license plate number to me, you may park in the CHS parking lot, which is Parking Lot F on the below link. Otherwise, please park in the Visitors lot, which is also on the below link. Please keep in mind that it's about a 10 minute walk from the Visitors lot to CHS. http://www.ithaca.edu/map/parking.php

Pre-Test Instructions

VO_{2max} Test

A VO_{2max} test is a high intensity treadmill test, which is designed to fatigue you within 8-12 minutes. It measures how much oxygen your body uses when you're working maximally, which for my purposes indicates your level of aerobic fitness.

This makes it a hard test to do properly, as while it's a short workout, it is not easy as it is physically and mentally challenging. For these reasons, **please make sure you are well rested prior to your test** – **ie, avoid doing a high intensity** / **long duration training workout the day before** – and limit your consumption of food/nicotine/alcohol etc as per the 'Pre Test Instructions' document. If these guidelines aren't followed, your test results may be inaccurate.

What Do I Bring/Wear?

You need to wear your usual running attire – shorts / t-shirt / broken in sneakers. There are also shower facilities available, so bring a change of clothes need to head straight out after your test.

Cycle/Run Tests

These are also difficult workouts, as you will be asked to replicate your competitive race pace during both cycling and running. Due to this, again, please ensure you are well rested and avoid performing a hard training session the day before.

What Do I Bring/Wear?

- Your bike. Please don't forget it!
- Your usual cycling attire, including shoes. You don't have to worry about a helmet!
- Your usual running attire, as per the VO_{2max} test
- You may bring music / an iPod / whatever gets you through a long training workout.
- If you wish to shower afterwards, bring your change of clothes, etc.

Can I take supplements (eg carb gel, Power Bars, etc) during the trial?

I have spoken to some of you about this, and it seems to be a mixed group – some do use these kinds of supplements during a long workout, and quite a few don't. So here's the ruling:

- If you don't use supplements, that's fine. Don't bring any.
- If you do wish to use supplements, **please bring enough to get you through all 3 cycle-run workouts to your first cycle-run bout**. I will label and store them at IC, and you will have access to them for your subsequent cycle-run trials.
 - If you don't bring them to your first trial, you can't use them for any subsequent trials.
 - I will be recording what you do use during your baseline trial, and when you use it. This routine needs to be replicated through all trials.

The main gist of this is that I need you guys to be consistent for all cycle-run trials. Use them if you want, but using the same products at the same times thoughout.

If there any questions about this, please don't hesitate to ask.

Training During Your Trial Period

Where possible, please maintain your regular triathlon training schedule - in both intensity and frequency. This helps ensure that any test results are due to the trials themselves, rather than because your training schedule changed.

Additionally, please record your workouts – either in the attached 'Training Log', or your own document/system. An entry similar to '10km run (hard) – 42mins' is sufficient.

Thanks, and I'll see you soon! Sharon

<u>APPENDIX E</u>

<u>Raw Data</u>

Table E1

D	•	· •		1 4
Des	crin	tive	raw	data
200	•••P		1.00.1.1	

Subj ID	Gender	Age (y)	Weight (kg)	Height (cm)	Trial Order	VO _{2max} (ml·kg ⁻¹ ·min ⁻¹)	PO _{avg} (W)	PC (rpm)
1	М	42	80	175	- / +	48.1	130	85
2	М	30	72	175	+ / -	71.0	169	89
3	F	47	59	168	- / +	50.0	134	79 [°]
4	М	36	76	183	- / +	63.1	176	75
5	М	42	86	188	, /·+	61.2	176	74
6	M	37	72 ·	175	-/+	61.2	167	91
7	М	45	80	188	- / +	62.2	• 221	82
8	Μ	29	70	183	+/-	59.2	171	93
9	М	40	79	175	+./-	66.6	211	90
10	М	47	73	173	+/-	57.9	181	85
11	Μ	37	80	178	+/-	62.2	191	88
12	M	27	70	178	+/-	71.6	146	86

Note: + / - indicates trial order was High Cadence then Low Cadence

- / + indicates trial order was Low Cadence then High Cadence

PO_{avg} = average Power Output during baseline trail

PC = Preferred Cadence during baseline trial-

 VO_{2max} = Highest rate of oxygen consumption reached during maximal treadmill

test.

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Table E2

				-		•
Subj ID	Baseline Cycle Time (min)	HC Cycle Time (min)	LC Cycle Time (min)	Baseline Run Time (min)	HC Run Time (min)	LC Run Time (min)
1	86.62	86.53	86.28	54.92	51.18	52.05
2	80.08	77.33	74.80	42.12	39.20	38.70
3	85.20	84.47	87.07	49.45	45.30	46.62
4	75.75	76.13	76.40	42.78	39.28	40.12
5	76.42	77.60	76.35	44.40	43.57	45.68
6	77.00	78.02	76.85	42.47	39.73	39.95
7 .	69.92	68.07	68.90	50.17	47.70	47.43
8	82.68	78.33	78.23	41.03	38.97	38.70
9	70.11	71.12	71.15	45.75	43.35	41.55
10	74.80	74.85	74.83	45.90	45.23	45.18
11	73.70	73.42	73.80	42.48	40.32	39.28
12	81.10	80.97	81.98	40.35	40.13	41.23

Cycling and running performance raw data

Note: HC = High Cadence trial; LC = Low Cadence trial

-7

Table E3a

Subj ID	VO _{2avg} (ml·kg ⁻¹ ·min ⁻¹)	VE (l·min ⁻¹)	RER	HR 27 km (bpm)	HR 28 km (bpm)	·Blood Lactate (mM)	RPE
1	29.38	50.13	0.87	155	157	8.1	14
2	35.68	40.13	0.83	128	137	5.3	13
3	32.65	38.88	0.85	128	130	3.1	14
4	36.20	51.17	0.92	156	161	5.6	14
5	32.83	66.73	0.94	143	142	4.7	14
6	36.48	57.51	0.88	151	150	6.4	12
7	46.80	79.57	0.85	170	169	6.9	15
. 8	37.40	50.39	0.89	150	147	5.2	14
9	43.30	66.85	. 0.90	164	162	9.7	15
10	39.43	69.22	0.90	171	171	7.2	17
11	34.53	56.59	0.90	124	131	6.2	13
12	38.10	51.60	0.90	138	139	9.0	13

Baseline trial raw physiological data at Time 1 (27 km – 28 km cycling)

Note: VO_{2avg} = average oxygen consumption

VE = Ventilation

RER = Respiratory exchange ratio

HR = Heart rate

Table E3b

Subj ID	VO _{2avg} (ml·kg ⁻¹ ·min ⁻¹)	VE (1·min ⁻¹)	RER	HR 38 km (bpm)	HR 39 km (bpm)	HR 40 km (bpm)	Blood Lactate (mM)	RPE
1	29.78	49.06	0.87	153	154	157	4.7	÷15
2	36.02	41.57	0.83	129	131	131	8.0	• 15
3	30.90	37.65	0.84	130	130	128	2.1	- 14
4	38.81	55.32	0.90	164	167	167	9.3	16
5	33.20	63.40	0.90	145	151	151	5.4	÷15
6	36.26	58.53	0.88	155	154	153	8:0	- 13
7	46.24	80.63	0.86	172	175	174	19:5	-16
8	31.43	43.68	0.88	147	147	144	7:7	14
9	44.57	67.01	0.89	162	162	161	· 6.7	: 15
10	44.04	97.45	0.88	173	178	178	9.9	17
11	34.77	57.11	0.88	124	135	135	[,] 5.8	14
12	40.27	53.71	0.89	142	138	149	9.6	13

Baseline trial raw physiological data at Time 2 (38 km – 40 km cycling)

Note: VO_{2avg} = average oxygen consumption

VE = Ventilation

RER = Respiratory exchange ratio

HR = Heart rate

Table E3c

Subj ID	VO _{2avg} (ml·kg ⁻¹ ·min ⁻¹)	VE (l·min ⁻¹)	RER	HR 1 min (bpm)	HR 2 min (bpm)	HR 3 min (bpm)	HR 4 min (bpm)	HR 5 min (bpm)
1	34.98	63.19	0.93	155	156	157	161	165
2	43.78	56.17	0.85	131	133	134	137	137
3	37.70	52.37	0.91	127	136	136	`137	135
*4	43.83	72.72	0.98	164	164	169	165	169
5	39.89	72.99	0.87	144	152	154	158	156
6	43.25	73.65	0.90	145	157	160	162	161
7	46.39	81.80	0.89	164	167	165	166	168
8	42.28	68.58	0.97	151	157	164	160	159
9	40.85	62.45	0.89	151	154	155	155	153*
10	42.56	75.81	0.93	169	179	177	179	179
11	40.09	67.83	0.88	135	133	137	133	139
12	44.36	60.65	0.90	137	143	138	141	139

Baseline trial raw physiological data at Time 3 (1 min – 8 min running)

Note: VO_{2avg} = average oxygen consumption

VE = Ventilation

RER = Respiratory exchange ratio

HR = Heart rate

Table E3c (continued)

Subj ID	HR 6 min (bpm)	HR 7 min (bpm)	HR 8 min (bpm)	Blood Lactate (mM)	RPE
1	162	168	169	9.7	16
2	142	141	144	9.7	13
3	135	138	138	6.1	16
4	170	168	167	11.5	16
5	157	.159	158	13.4	14
6	161	163	161	6.1	13
7	168	171	172	6.6	15
8	161	167	163	7.2	16
9	157	158	156	10.5	12
10	180	180	180	9.4	18
11	141	.142	139	8.8	14
12	145	149	149	5.4	14

Baseline trial raw physiological data at Time 3 (1 min – 8 min running)

Table E3d

				· .		3		
Subj ID	VO _{2avg} (ml·kg ⁻¹ ·min ⁻¹)	VE (1·min ⁻¹)	RER	HR 8 km (bpm)	HR 9 km (bpm)	HR 10 km (bpm)	Blood Lactate (mM)	RPE
1	39.71	83.39	0.90	184	188	191	13.4	19
2	54.41	80.52	0.87	165	166	170	10.5	17
3	42.12	63.02	0.86	150	152	156	11.9	17
4	52.66	90.04	0.91	179	182	187	9.5	18
5	48.79	96.09	0.88	168	174	179	6.8	17
6	45.69	83.38	0.87	171	171	171	4.5	15
7	50.42	96.35	0.85	177	179	183	4.7	17
8	47.29	81.81	0.89	177	178	186	7.4	20
9	50.34	76.45	0.87	170	175	176	7.2	17
10	44.91	90.66	0.87	182	183	185	8.7	19
11	54.61	96.45	0.88	156	162	166	4.5	17
12	52.25	71.55	0.87	166	169	175	4.4	17

Baseline trial raw physiological data at Time 4 (8 km – 10 km running)

Note: VO_{2avg} = average oxygen consumption

VE = Ventilation

RER = Respiratory exchange ratio

HR = Heart rate

Table E4a

Subj ID	VO _{2avg} . (ml·kg ⁻¹ ·min ⁻¹)	VE (l·min ⁻¹)	RER	HR 27 km (bpm)	HR 28 km (bpm)	Blood Lactate (mM)	RPE
1	28.30	49.31	0.87	148	153	1.5	11*
2	39.95	48.72	0.86	135	134	4.9	12
3	30.83	41.02	0.89	- 114	120	7.8	14
4	32.97	42.18	0.86	132	133	4.2	14
5	32.83	61.01	0.90	140	138	11.1	14
6	33.05	46.91	0.88	124	133	2.6	12
7	43.60	75.20	0.88	151	160	7.5	14
. 8	32.87	43.23	0.85	142	137	3.7	13
9	43.40	63.73	0.93	150	151	7.2	13
10	35.19	64.24	0.89	158	165	8.9	16
11	33.53	53.91	0.88	117	121	4.4	13
12	35.70	44.24	0.88	128	132	3.6	13

High cadence trial raw physiological data at Time 1 (27 km - 28 km cycling)

Note: VO_{2avg} = average oxygen consumption

VE = Ventilation

RER = Respiratory exchange ratio

HR = Heart rate

Table E4b

Subj ID	VO _{2avg} (ml·kg ⁻¹ ·min ⁻¹)	VE (l·min ⁻¹)	RER	HR 38 km (bpm)	HR 39 km (bpm)	HR 40 km (bpm)	Blood Lactate (mM)	RPE
1	31.03	57.28	0.89	160	160	166	6.6	15
2	39.55	49.30	0.85	144	143	149	3.9	14
3	33.49	44.83	0.88	122	120	120	3.9	16
4	34.15	45.34	0.89	140	140	148	6.7	15
5	33.66	71.06	0.93	139	141	140	. 4.0	15
6	34.65	53.58	0.88	140	140	145	9.7	13
7	45.76	84.93	0.90	167	168	167	9.6	15
8	35.38	48.37	0.88	152	. 144	148	4.3	14
9	40.78	63.31	0.91	142	153	156	7.2	15
10	47.55	89.36	0.90	173	176	175	7.2	17.
1-1	37.80	64.89	0.89	135	136	135	5.3	15
12	37.04	45.20	0.87	144	137	140	5.7	16

High cadence trial raw physiological data at Time 2 (38 km - 40 km cycling)

Note: $VO_{2avg} = average oxygen consumption$

VE = Ventilation

RER = Respiratory exchange ratio

HR = Heart rate

Table E4c

· · · · ·	<u> </u>					*		
Subj	VO _{2avg}	VE	RER	HR 1 min	HR 2 min	HR 2 min	HR 1 min	HR
ID	(ml·kg ⁻¹ ·min ⁻¹)	(l·min ⁻¹)	KEK	1 min (bpm)	2 min (bpm)	3 min	4 min	5 min
				(opin)	(opin)	(bpm)	(bpm)	(bpm)
1	35.50	71.28	0.93	158	162	162	167.	167
2	45.84	61.43	0.87	141	146	147	149	151
3	40.0Š	58.30	0.91	121	135	137	138	143
4	44.63	75.05	1.00	150 ·	156	161	164	163
5	39.69	71.06	0.86	136	144	146	148	145
6	45.02	75.37	0.91	143	152	157	157	158
. 7	55.20	79.43	0.90	153	160	162	161	161
8	45.63	73.80	0.96	155	159	167	168	169
9	42.11	66.52	0.91	136	153	151	152	153
10	45.53	85.27	0.90	170	176	175	174	175
11	42.05	69.49	0.86	125	136	132	137	137
12	43.43	56.06	0.89	132	143	143	150	147

High cadence trial raw physiological data at Time 3 (1 min - 8 min running)

Note: VO_{2avg} = average oxygen consumption

VE = Ventilation

RER = Respiratory exchange ratio

HR = Heart rate

Table E4c (continued)

Subj ID	HR 6 min (bpm)	HR 7 min (bpm)	HR 8 min (bpm)	Blood Lactate (mM)	RPE
1	170	168	172	5.3	16
2	153	154	156	6.2	16
, 3	143	142	142	5.9	17
4	163	162	163	7.1	16
5	150	151	150	4.5	15
6	160	160	157	6.9	15
7	164	164	164	9.1	15
8	169	172	173	12.9	17
9	153	156	153	7.5	14
10	175	177	177	10.7	17
11	137	136	137	2.3	<u>14</u>
12	151	150	150	6.3	15

High cadence trial raw physiological data at Time 3 (1 min – 8 min running)

Table E4d

Subj ID	VO _{2avg} (ml·kg ⁻¹ ·min ⁻¹)	VE (l·min ⁻¹)	RER	HR 8 km (bpm)	HR 9 km (bpm)	HR 10 km (bpm)	Blood Lactate (mM)	RPE
1	41.98	94.65	0.90	182	186	190	11	20
2	51.96	82.67	0.89	166	170	175	6	19
3	45.37	66.16	0.86	153	154	156	5	18.
4	53.13	93.31	0.93	173	178	182	9	17
5	52.65	103.86	0.90	167	176	180	6	18
6	51.01	83.78	0.87	172	175	180	4	15
7	50.08	95.23	0.87	. 182	180	184	5	16
8	50.27	85.50	0.88	173	177	183	14	20
9	52.26	84.58	0.93	169	177	181	12	18
10	49.18	92.67	0.90	176	179	180	7	18
11	52.72	94.52	0.91	153	157	163	6	17
12	55.87	78.45	0.88	172	175	180	Ż	18

High cadence trial raw physiological data at Time 4 (8 km - 10 km running)

Note: VO_{2avg} = average oxygen consumption

VE = Ventilation

RER = Respiratory exchange ratio

HR = Heart rate

Table E5a

Subj ID	VO_{2avg} (ml·kg ⁻¹ ·min ⁻¹)	VE (l·min ⁻¹)	RER	HR 27 km (bpm)	HR 28 km (bpm)	Blood Lactate (mM)	RPE
1	31.90	49.34	0.87	142	146	5.6	13
2	38.63	43.43	0.85	131	136	2.9	12
3	31.55	39.79	0.85	116	111	2.9	13
4	33.80	46.89	0.89	143	140	5.4	13
5	31.80	55.79	0.90	136	133	4.2	. 14
6	31.55	46.36	0.88	134	130	3.6	13
້ 7	47.60	80.91	0.90	146	154	6.2	13
8	34.03	44.93	0.88	142	141	4.2	12
9	36.95	56.60	0.89	130	132	4.9	12
10 ·	36.83	71.22	0.89	170	156	8.6	16
11	36.83	61.30	0.89	136	127	7.2	14
12	31.15	38.97	0.87	122	118	5.6	13

Low cadence trial raw physiological data at Time 1 (27 km - 28 km cycling)

Note: VO_{2avg} = average oxygen consumption

VE = Ventilation

RER = Respiratory exchange ratio

HR = Heart rate

Table E5b

e.						•	· · ·	
Subj ID	VO_{2avg} (ml·kg ⁻¹ ·min ⁻¹)	VE (l·min ⁻¹)	RER	HR 38 km (bpm)	HR 39 km (bpm)	HR 40 km (bpm)	Blood Lactate (mM)	ŔPĖ
1	30.12	52.25	0.86	.146	145	150	3.9	13
2	41.34	49.11	0.85	140	140	143	2.7	13
3	31.10	39.81	0.85	118	112	116	3.9	14
4	33.24	44.32	0.87	143	146	146	5.7	14
5	31.04	51.97	0.87	132	124	126	4.9	15
6	34.69	52.24	0.86	139	141	141	2.0	13
7	40.47	66.28	0.88	153	154	157	-5.2	13
8	35.98	50.96	0.89	139	142	147	5.7	15
9	34.00	51.32	0.88	132	133	136	4.5	12
10	38.06	77.12	0.90	157	156	159	5.8	16
11	35.00	54.64	0.86	119	125	128	2.5	13
12	30.41	38.07	0.85	112	114	113	5.2	14

Low cadence trial raw physiological data at Time 2 (38 km - 40 km cycling)

Note: VO_{2avg} = average oxygen consumption

VE = Ventilation

RER = Respiratory exchange ratio

HR = Heart rate

Table E5c

Subj ID	VO_{2avg} $(ml \cdot kg^{-1} \cdot min^{-1})$	VE (l·min ⁻¹)	RER	HR 1 min (bpm)	HR 2 min (bpm)	HR 3 min (bpm)	HR 4 min (bpm)	HR 5 min (bpm)
1	35.27	68.24	0.92	151	155	158	162	164
2	46.32	63.98	0.88	141	148	148	148	148
3	39.50	56.50	0.91	122	130	131	135	135
4	44.04	74.33	1.00	153	160	163	164	165
5	41.16	66.57	0.84	129	142	140	143	143
6	43.96	73.70	0.90	141	157	161	162	163
7	45.16	76.18	0.90	149	155	155	155	155
8	47.39	76.75	0.96	153	163	158	164	172
9	41.76	68.54	0.93	136	137	144	151	155
10	40.25	70.83	0.93	161	170	165	165	168
11	43.02	72.73	0.88	133	138	135	137	138
12	42.09	54.37	0.88	124	134	135	137	136

Low cadence trial raw physiological data at Time 3 (1 min – 8 min running)

Note: VO_{2avg} = average oxygen consumption

VE = Ventilation

RER = Respiratory exchange ratio

HR = Heart rate

Table E5c (continued)

Subj ID	HR 6 min (bpm)	HR 7 min (bpm)	HR 8 min (bpm)	Blood Lactate (mM)	RPE
· 1	162	165	165	8.1	16
2	148	148	148	3.7	15
3	133	134	135	6.0	15
4	164	165	164	12.1	15
5	147	145	144	3.9	15
6 [·]	164	164	161	3.8	14
7	157	158	158	5.0	14
8	168	169	169	17.7	17 .
9	154	154	156	5.2	16
10	169	170 .	169	8.1	17
11	136	144	141	4.7	14
12	137	142	141	10.0	15

High cadence trial raw physiological data at Time 3 (1 min – 8 min running)

Table E5d

Subj ID	VO_{2avg} (ml·kg ⁻¹ ·min ⁻¹)	VE (l·min ⁻¹)	RER	HR 8 km (bpm)	HR 9 km (bpm)	HR 10 km (bpm)	Blood Lactate (mM)	RPE
1	43.01	96.63	0.89	178	186	188	10.4	20
2	48.91	74.36	0.85	163	152	163	5.4	19
3	44.07	64.89	0.86	145	148	149	6.0	18
4	53.44	92.01	0.90	177	179	183	10.7	17
5	49.83	87.75	0.86	147	154	167	5.4	18
6	49.75	86.12	0.87	173	178	179	4.7	15
7	52.66	94.07	0.86	173	175	177	3.4	16
8	49.73	81.24	0.88	173	171	185	7.2	20
9	53.18	88.75	0.93	169	174 [°]	177	9.7	18
10	42.08	76.99	0.87	174	. 179	180	6.8	17
11	55.76	98.84	0.89	152	159	164	5.9	17
12	52.83	69.93	0.87	157	169	174 [,]	5.1	17

Low cadence trial raw physiological data at Time 4 (8 km - 10 km running)

Note: VO_{2avg} = average oxygen consumption

VE = Ventilation

RER = Respiratory exchange ratio

HR = Heart rate