High intensity swimming outlay in Olympic distance triathlon does not affect cycling performance and cycling economy

Daniel Schiodt

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Author/s: Daniel Schiødt
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Abstract

Aim: The purpose of the present study was to investigate to what extent swimming at 100% of 500m velocity the first 500m of a 1500m swim will influence on average 1500m velocity and the intensity and economy during a 22.5km TT (time trial) on a cycle ergometer.

Method: Six triathletes (n = 6) five male and one female, age (34.8 ± 6.4) years, and VO2max: (63.2 ± 5.7)ml-kg⁻¹·min⁻¹), performed baseline testing on two different days: 1) an isolated 500m maximal swim test and a VO2max cycle test, 2) Cycle lactate threshold (LT) -, and a cycle 22.5km time trial test with continuous VO2max measurements. After resting for one day, two consecutive tests, separated by 24 hours, were performed: a) 500m maximal swimming outlay continued by 1000m swimming at competitive velocity (S500max TT), further continued by transition (T1) and a 22.5km cycle time trial (TT). b) 1500m swim at competitive velocity (S1500 TT), transition T1, and a 22.5km cycle TT.

Parameters (VO2, VO2 scaled, %VO2, VE, R, HR, La-, Brake power, CAD, Cc and WE) were measured.

Results: The results showed a significant difference (3.3%) between mean power produced during the 22.5km time trial and the S500max TT, accompanied by a 4% reduction in mean VO2 and a 2.5% increase in HR. No difference in mean power produced, VO2 or HR between the 22.5km TT and S1500 TT was apparent.

Conclusion: Although suffering from gradual fatigue and suspected hyperthermia in both consecutive tests, there were no significant differences in between S500max TT and S1500 TT. These triathletes may thus benefit strategically, and in an indirect manor physiologically, from a 500m maximal swimming outlay in an Olympic triathlon.

Advice: The advice to these triathletes is to implement a 500m maximal swimming outlay in their strategy in order to gain the best possible position for further benefits of drafting, and thereby maintaining the potential of optimizing their race performance in Olympic Triathlons.
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Abbreviations

ATP  Adenosinetriphosphate
Ca2+  Calcium Ion
CAD  Cadence
Cc  Cost of cycling
CHO  Carbohydrate
CO2  Carbon Dioxide
DPS  Distance Per Stroke
EC  Economy
FFA  Free Fatty Acids
H+  Hydrogen Ion
H2PO4-  Di-hydrogen tetra phosphate
HCO-3  Bicarbonate Ion
HR  Heart Rate
HRpeak  Peak Heart Rate
K+  Potassium
La-  Lactate
[LA-]b  Blood lactate concentration
LT  Lactate Threshold
MAP  Maximal Aerobic Power
MCT  Monocarboxylate transporter
MHC  Myosin Heavy Chain
Mg2+  Magnesium Ion
OBLA  Onset of Blood Lactate Accumulation
PCr  Phosphocreatine
Pi  inorganic Phosphorus
PO  Power Output
Qc  Cardiac output
R  Respiration exchange ratio (CO2expired/ O2 inspired)
RPM  Revolutions Per Minute
SV  Stroke Volume
VE  Volume of Expiration
VO2  Oxygen consumption
VO2max  Maximal Oxygen Uptake
%VO2max  Fractional utilization of maximal oxygen uptake
W  Watt (Brake Power)
WE (ή)  Work Efficiency
Introduction

Olympic Triathlon
Olympic triathlon is an endurance discipline consisting of continuous swimming, cycling and running at distances 1500m, 40km and 10km, respectively. The change in between disciplines, the so called transfers T1 and T2, is undertaken as fast as possible as a part of the competition. A successful completion of an Olympic triathlon by elite male competitors typically lasts approximately two hours but may vary according to weather conditions and terrain. The time proportions of swimming, cycling and running in Olympic triathlon are approximately 15%, 48% and 37% respectively.¹

The Energetics of Triathlon
Skeletal muscle requires chemical energy to contract and undertake mechanical work. The contractions are enabled through the breakdown of ATP (adenosine triphosphate) via myosin ATPase. ATP is present as intramuscular stores (approximately 5 mmol/kg wet weight) which is a relatively small amount to comply for mechanical work. Other metabolic pathways to resynthesize ATP must therefore be activated in order to maintain muscle contractions.² There are three distinct energetic pathways that ensures the generation of ATP to comply for mechanical work of the skeletal muscle. These are the phosphocreatine (PCr) breakdown, glycolysis, and aerobic oxidation of carbohydrates (CHO) and Free Fatty Acids (FFA). The intensity of the exercise is an important determinant for which of the energetic processes that are recruited. These closely integrated energy processes, or metabolic pathways, are limited in two inherent ways: by the maximum rate (power) and the amount of ATP (capacity) that can be produced.³ ⁴

Medbø & Tabata¹¹⁴ have demonstrated that the amount of both aerobic and anaerobic energy releases in bicycling to exhaustion in 0.5, 1 and 3 minutes, respectively, are equal after 60 seconds. Aerobic energy release becomes increasingly important as the duration of activity increases. According to Aastrand & Rhodal (1986),¹⁹ the energy release during a marathon is 99% aerobic. Olympic triathlon performance is thus heavily dependent on aerobic endurance capacity.
**Aerobic Endurance Performance**

Several investigators have indicated that the main requisites for successful triathlon performance are high maximal oxygen uptake (VO2max), lactate threshold, and maximal sustainable percentage of VO2max, as well as a low energy cost of exercise for each discipline. In a heterogeneous group of endurance athletes the performance factor VO2max is properly the singular most important one. When these variables are measured in the single disciplines and in simulated triathlons however, the relation between them is lower as when compared to the respective single sports disciplines. The prior exercise affects the strength of the correlation between physiological variables specific to one discipline and performance in it under conditions characteristic of triathlon competition.

**Maximal Oxygen Uptake (VO2max)**

Maximum oxygen uptake (VO2max) is defined as the highest rate at which oxygen can be taken up and utilized by the body during severe exercise. Minute volume is a measure for the heart’s pumping capacity and is often named cardiac output (Qc). Qc is measured in litres of blood per minute and is dependent on stroke volume (SV) and heart frequency (Hf). Stroke volume is the amount of blood ejected from the heart with each heart beat. Stroke volume is equal to the difference between the amount of blood in the heart after completion of filling (end-diastolic volume) and the amount of blood remaining after ejection (end-systolic volume) (Åstrand & Rhodahl 1986). At rest an average person has Qc of 5 liters. At maximal exercise the amount may rise to above 30 L/minute (ibid, Åstrand & Rhodahl 1986). The rise in Qc depends on a linear increase in both SV anf Hf up to a certain point among untrained to moderately trained individuals (approximately 40-60% of Hfmax) but increases up to approximately Hfmax among elite endurance athletes (Zhou et al 2001).

Oxygen uptake can be expressed as cardiac output Qc multiplied by the systemic oxygen extraction, or the arteriovenous oxygen difference across active tissues, as:

\[ \text{Vo2 (l/min) = Qc (l/min) X (arterial O2 content- venous O2 content)} \]  

(Andersen & Saltin 1985). As such, maximal oxygen uptake can also be determined by measuring maximal cardiac output and the arteriovenous oxygen difference. Whereas the maximal cardiac output is the product of maximal heart rate and maximal stroke volume, the maximal arteriovenous oxygen difference is the difference between the maximal arterial - and minimal venous blood oxygen content.
There are two main factors that limit VO2max. These are the rate of O2 utilization by the muscles and the O2 supply to the muscles (Åstrand & Rodahl 1986).\textsuperscript{19} When vigorous exercise by large muscle groups as in bicycling is performed, the addition of extra muscle groups only produces a small increase in cardiac output and VO2max. If the critical factor were muscle O2 usage, the additional contracting muscles would use proportionally more O2 to meet the enhanced O2 requirements.\textsuperscript{22}

When exercising, the VO2max seem to be limited by the capacity of the cardiorespiratory system in that it fails to deliver enough oxygen to the exercising muscles (Di Prampero, 2003).\textsuperscript{118} The O2 delivery failure of the cardiorespiratory system in humans can be observed by three lines of evidence: 1) When manipulating with the oxygen delivery, (by blood doping, hypoxia, or beta-blockade), VO2max changes accordingly;(Basset. et al., 2000).\textsuperscript{17} 2) The increase in VO2max with training results stem primarily from an increase in maximal cardiac output not an increase in the a-vO2 difference; (Helgerud et al., 2007).\textsuperscript{119} 3) When exercising with a small muscle mass, as in one leg cycling or knee extension, this muscle mass has a VO2 two to four-fold the VO2 in the same muscle mass during whole body exercise (Andersen & Saltin 1985, Powers et al., 1989).\textsuperscript{120,23}

**Fractional utilization of maximal oxygen uptake (%VO2max)**

Fractional utilization may only be a function of time if the race lasts for 30 minutes or shorter.\textsuperscript{24} If the duration is longer, fractional utilization is very similar to the term LT, measured as the % of VO2max, and determined by the same factors.\textsuperscript{24}

**Substrate availability and hydration**

An increase in pre-event glycogen stores can prolong the duration for which moderate-intensity exercise can be undertaken before fatiguing. It may also enhance the performance of a set amount of work (i.e. a set distance) by preventing the decline in pace or work output that would otherwise occur as glycogen stores decline towards the end of a task.\textsuperscript{25} Typically, CHO loading will postpone fatigue and extend the duration of steady-state exercise by as much as 20%, and improve performance over a set distance or workload by 2-3%. Increased CHO availability either by muscle glycogen loading prior to exercise or CHO ingestion before and during exercise, is associated with
enhanced endurance performance.\textsuperscript{26,27} Also enhanced ability to utilize fat at a given intensity should logically give the same postponement of fatigue as glycogen loading. According to Norby et al. (2006) individuals with a higher VO2max will have the highest ability to oxidise fat at different submaximal intensities.\textsuperscript{28}

Performance can be impaired when athletes are dehydrated. Adding carbohydrates and natrium Na(+) in beverages favours consumption and retention, respectively. Endurance athletes are thus recommended to maintain a proper hydration before, during, and after training and competition since it will help replace fluid loss and will lower the submaximal exercise heart rate by maintaining plasma volume and evidently, maintain performance.\textsuperscript{29} Athletes are in several studies recommended to consume 500ml of fluid solution 1-2 hours prior to competition, and to hydrate with cold fluid solutions in regular intervals. For prolonged intense exercise lasting more than 1 hour, athletes are recommended to drink between 600ml to 1200ml of a 4-8% carbohydrate solution per hour.\textsuperscript{24,30} However, sweat rates vary greatly between individuals, even when exercising at the same metabolic rate.\textsuperscript{31}

\textbf{Lactate Threshold}
At rest and during easy, steady rate swimming, lactate is produced and consumed at equal rates. As a result, there appears to be no net increase in blood lactate concentration (Donovan & Brooks 1983; Brooks et al 1996). Lactic acid turnover is the ratio between lactic acid production and removal, but it doesn’t explain the magnitude of how much lactate that is produced and removed as maximal values. Thus, even though lactate turnover (production and consumption) may be several times higher during easy to moderate swimming, we might not observe any increase in blood lactate. If one however, at steady state swimming observes an increase in blood lactic acid concentration, the logical explanation would be that there must be more production than consumption of lactic acid and, hence, lactate accumulates in the blood.\textsuperscript{32}

Although muscle or blood lactate accumulation are good indirect indicators of increased proton release and the potential for decreased cellular and blood PH, such relationships should not be interpreted as cause and effect.\textsuperscript{33}

The metabolic acidosis is caused by an increased reliance on nonmitochondrial ATP turnover and not lactate production. Hence, as pointed out by Robergs RA et al. (2004), the best way to decrease metabolic acidosis is to decrease nonmitochondrial ATP turnover by stimulating mitochondrial respiration. For a given ATP demand, any effort to decrease lactate production without increasing
mitochondrial respiration will worsen metabolic acidosis. This seems to be in accordance with the observations of Holloszy et al. (1984).123

The basic threshold paradigm is that elevated lactic acid (HLa) production and concentration during exercise is the result of O2-limited oxidative phosphorylation.34 For most untrained individuals, the non-linear relationship between VO2 and blood lactate acid concentration results in an inflection point in blood lactate acid around 65% of maximum oxygen capacity (VO2max). However, as the individual becomes trained the onset blood lactate (OBLA) inflection point occurs at a greater percentage of maximal oxygen consumption. 35, 36, 37 Highly trained distance swimmers may exhibit OBLA at 90% or more of their VO2max. This translates to an increased ability to do work at a given workload as well as an increased time to fatigue at a given workload.38 This may be a result of an increased ability to clear lactate by consumption, or a reduced reliance upon nonmitochondrial ATP turnover during exercise, or a combination of both.39 OBLA represents the highest work intensity with a balance between the production and removal of lactate from the blood and suggests nothing about aerobic or anaerobic mitochondrial metabolism per se.40

**Ventilatory requirements of exercise**

The ability to maintain arterial blood-gas and acid-base homeostasis during exercise requires work and power from the respiratory and cardiac ‘pumps’. Since respiratory cost of work during exercise increases, enough energy must be provided by local vascular perfusion to the respiratory muscles as well as adequate amounts of stored substrates that can readily be taken into use. If these criteria are not met, respiratory muscle fatigue may occur.

At submaximal work intensities below the so called ventilatory threshold, the increase in VE curve is linear and proportional to the rate of increased work of the respiratory and skeletal muscles. When exercising near maximum VE increases rapidly, and at this stage moderately fit subjects have been shown to use 30-40 % of their respiratory muscle power, also termed Maximal Voluntary Ventilation (MVV). This difference in between MVV and the VE at near maximal exercise has been termed the ‘breathing reserve’. The MVV is largely independent of fitness and training status, opposite that of VE in so that well trained subjects can attain a high VE and maintain a high percentage of MVV. Because of this, well trained subjects have a low breathing reserve.41
**Metabolic cost of respiratory work**

A high VE in itself requires energy. From rest and to an intensity corresponding to VO2max, the relative oxygen cost of breathing increases to approximately 10% (Aaron et al 1992).121

**Work Economy and Work Efficiency**

The energy cost of locomotion (C) may be defined as the quantity of energy spent per distance unit for example expressed in ml O2/ kg/km or as J/kg/km.42

Cycling economy (EC) is often defined as the rate of energy expenditure per unit of brake power output, or in other words; the amount of oxygen per Watt (Cc).43

Cooper and Storer (2001) define work efficiency (η) as a measure of the metabolic cost of performing external work. Hence, work efficiency is calculated by dividing the caloric value of the external work performed by the metabolic cost of the work in terms of the caloric value of the oxygen uptake.44

Work efficiency has been debated by Moseley et al. (2001). In the debate, Moseley points out that it is difficult to interpret the results of studies that make use of the term efficiency without knowing the reliability of the measure. The WE has a baseline of energy cost of unloaded cycling (0W) typically about 5 kJ/min-1. The method has been criticised, since exercise changes gastro intestinal blood flow, splanchnic processes, cardiac output, and ventilation rates that again will increase the energy needed to maintain homeostasis. Therefore, it is unlikely that either of the baseline energy expenditure measurements remain constant during exercise with changes in oxygen uptake (VO2), cadence, or environmental conditions.49

Miura, H. et al. (1997) found significant correlations between both Cc and overall triathlon performance and between running economy and overall triathlon performance during a simulated laboratory test triathlon and an Olympic distance triathlon. Swimming economy correlated poorly (r = 0.208, ns).
Cycling economy and efficiency

Horowitz et al. (1994)\textsuperscript{51} showed that in a group of cyclists that were divided according to the proportion of muscle-type 1 fibres, the subgroup that was high in the proportion of type 1 fibres (73\% VS. 48\%), produced a significantly higher power for a given VO\textsubscript{2} during a one-hour test, indicating a higher efficiency or economy.

Fibre recruitment may relate to the intensity-dependent increase in oxygen uptake and total energy requirement during constant load dynamic exercise in several ways, either by recruitment of additional fibres of both main types, a gradual shift in fibre recruitment towards less efficient FT fibres, and/or a specific decrease in FT fibre efficiency over time due to metabolic and ionic changes.\textsuperscript{52} Sunde et al. (2009)\textsuperscript{53} has shown that 8 weeks of maximal strength training improved Cc (5\%) and WE (4.7\%) in cycling at 70\% VO\textsubscript{2max} and in time to exhaustion (17.2\%) at maximal aerobic power (MAP) among competitive road cyclists with no changes in freely chosen cadence, and with no concurrent increase in body weight or maximal oxygen uptake. Maximal strength training may thus be an effective enhancer of performance in cycling.

Hansen et al. (2002)\textsuperscript{54} studied the variation in freely chosen pedal rate between subjects and its possible dependence on percentage myosin heavy chain I (%MHC I) in m. vastus lateralis, maximum leg strength and power, as well as efficiency. The investigators found that there was a considerable variation in freely chosen pedal rate between subjects: 56-88 r.p.m. at low and 61-102 at high, during cycling at submaximal power outputs (low 40 and high 70\% of the power output at which maximum oxygen uptake (VO\textsubscript{2max}) was attained at 80 r.p.m.). This variation was only partly explained by percentage MHC I (21-97\%) as well as by leg strength and power. Subjects with high percentage MHC I chose high pedal rates close to the pedal rates at which maximum peak crank power occurred, while subjects with low percentage MHC I tended to chose lower pedal rates, favouring high efficiency. In the same study it is outlined that in general the subjects had a freely chosen pedal rate of 24 and 14 r.p.m. higher, at low and high submaximal power output respectively, than what elicited the highest gross efficiency: 50 and 66 r.p.m. at low and high submaximal power output, respectively.

If one were to decrease the pedal rate correspondingly, the savings corresponds to 60 and 20 seconds, respectively on a 40km distance, which is the distance travelled on bicycle in Olympic triathlon.
Fatigue

Fatigue is defined as a reduction in the force or power-generating capacity of muscle. The sites of fatigue include the central nervous system and motor outflow, and peripheral sites such as the sarcolemma, t-tubule system, sarcoplasmatic reticulum (SR) and myofilaments. It is unlikely that a single mechanism can explain fatigue under all circumstances, but possible mechanisms include ionic disturbances, impaired excitation-contraction coupling, accumulation of metabolites, and substrate depletion. Possible mechanisms for the impaired excitation-contraction coupling may include a reduced calcium release from the SR and impaired myofibrillar calcium sensitivity. This impaired sensitivity could also be due to increased metabolite/ion (e.g. Ca2+, Mg2+, H+, Lactate and Pi, inorganic phosphate, also present as H2PO4-) accumulation or modification by free radicals however, recently the role of acidosis as an important cause of fatigue has been challenged.

Bangsbo et al. (1996) has shown in a study where intense exhaustive leg exercise, with- and without a proceeding intense intermittent arm exercise, that muscle PH was inferior to that of femoral blood potassium K+ in indicating fatigue because the level of potassium was equal at fatigue, unlike the level of lactate. Furthermore, the lowered muscle PH did not affect the rates of glycogen breakdown and production of lactate. Nordsborg et al. (2003) showed in an in vitro study that if you take a muscle and incubate it in a chamber and then slowly increase the level of K+, the muscle will reduce its contractibility and finally stop to contract. In this study however, potassium levels was different at fatigue thus bringing doubts to the findings of Bangsbo (1996). Pedersen et al. (2007) argues that it is not muscle acidification that makes muscle contraction deteriorate because at high levels of potassium where muscle contraction deteriorates, adding levels of acidification and adrenalin made muscles regain their contractibility. They argue that acidification helps protect the muscles ability to contract.

It is extremely difficult to identify the exact mechanisms involved in a fatiguing voluntary exercise protocol. Identifying causal relationships for parameters examined in relation to fatigue is difficult since a lot of our current knowledge has been put together from in vitro and in vivo studies. In vitro studies explaining fatigue may not represent the dynamical changes that follow from an in vivo exercise protocol where individuals become fatigued voluntarily. Thus, one should be very careful when extrapolating the results of such studies to definitive fatigue mechanisms.
Swimming in Triathlon

In contrary to pool-based swim competitions, triathlon swimming takes place in a river, a lake or in the sea. The swimming often begins as a mass start with as much as 300 competitors, and in contrast to the uniform conditions for regular swim competitions, the triathlon swim sequence is performed under varied conditions, types of water, turbulence and temperatures.65

Gonzales-Haro et al. (2005)105 describes in his study of physiological adaptations in a simulated Olympic triathlon swim to cycle sequence, how the pool length has an important effect on metabolic and biomechanical variables which one has to consider when compare studies undertaken in pools of different length. When compared to a 50meter pool, swimming in a 25meter pool results in more effective swimming. The reason for this is the many turns one takes in a 25meter pool compared to a 50meter pool over a given distance, and it results in significantly lower blood-lactate and heart rate at the same relative velocity. It is underlined that the differences in variables gets more significant as the intensity rises, with the exception of HR, and that there at maximal velocity where no differences.

Technique and physical capacity are important factors with respect to performance in front crawl swimming.66 Swimming in Olympic distance triathlon consists of a 1500m effort. Front crawl swimming is the preferred style owing to the fact that it is the more effective in regards to energy expenditure speed of propulsion and biomechanical efficiency. Since water has a molecular density that is almost a 1000 times greater than that of atmospheric air, the physical element in which one exercises when swimming, presents a large resistance that has to be overcome, and the forces or power required to obtain propulsion through the water, is large as compared to the physical element of air in cycling or running.

One could argue that by applying more force and thereby increasing the muscular power output, these forces can be overcome and good performance would be eminent. For a lot of sports disciplines, this could be the case. However, in the case where physical performance requires a large component of technical ability, as in the case of swimming, the importance of adding more power to increase physical performance becomes less evident.68 As speed increases, water resistance increases to the same amount raised to the power of 2.69

There are two central tenets of swimming performance. One is the ability to decrease the hydrodynamic resistance that the body represents in water. A second ability of performance is to make propulsive forces, as when arm-stroking and leg-kicking, as effective as possible.70
One of the constraints of being partially submerged in water is that the water’s density exerts a hydrostatic pressure on the swimmer’s chest wall once the inspiratory muscles are relaxed. This pressure eventually has to be overcome by elevating the inspiratory muscle work and those muscles may become fatigued. One may speculate that a wetsuit, which has to fit the triathletes’ body tightly, may also elevate the resistive forces in inspiratory muscle work. Also, since the swimmer’s face is submerged in the water to improve horizontal body position and decrease resistive forces, the swimmer has to coordinate the breathing pattern with the biomechanical stroking of the arms. The arm-coordination and a part-time submerged face leaves the swimmer no choice but to make a rapid and forceful inspiration once the face is clear of the water, which may be fatiguing to the inspiratory muscles. In relation to breathing pattern, a swim-bench study has shown that bilateral breathing, as when breathing for every 3rd or 6th armstroke, creates a more balanced power output (PO) in between the arms when stroking, and that this pattern decreases resistive forces as compared to unilateral breathing.

Elite swimmers are known to be more effective than elite triathletes. The energy cost during front crawl is 21 to 29% lower, and propelling efficiency 36.4% higher among elite swimmers as when compared to triathletes. Some of these differences may be due to an increase in the propulsive phases and a shortening of the recovery phase for the swimmers at high velocities. In contrast, the triathletes seem to increase their recovery phase at maximal velocities which leaves them with less time to produce power with the submerged arm during an arm-cycle. Also, since the stroke rate has shown to be equal between swimmers and triathletes at high velocities, the arms of swimmers are likely to be moving faster during the propulsive phase thereby producing more power per stroke and a higher velocity.

A study by Roels et al. (2005) compared VO2max and ventilatory threshold in free swimming and cycle ergometry between swimmers and triathletes. Since there is a peripheral component to VO2max, the study wanted to examine how the mode of activity and total muscle mass being used during maximal exercise relates to the level of oxygen uptake. Vo2max in the cycling test was significantly (p<0.01) higher in the triathletes than in the swimmers. In the swimming test however, VO2max was significantly (p<0.05) higher in the swimmers than in the triathletes. VO2max was also significantly (p<0.05) higher in the cycling test than in the swimming test in triathletes. The opposite happened to the swimmers, showing a significantly (p<0.05) higher VO2max in swimming than in the cycling test. The study confirmed that different testing modes lead to different VO2max
values, and that swimmers have very specific training adaptations even when compared to triathletes.

**Wetsuit usage.**
It is widely accepted that the use of a wetsuit during the swimming section of a triathlon leads to reductions in energy cost and gains in performance by improving buoyancy and reducing drag.\(^78\)\(^79\)\(^80\)\(^81\)\(^82\)\(^83\)\(^84\). Core temperature elevations of 0.89 ± 0.13 degrees Celsius caused by 30 minutes of wetsuit swimming has shown to occur in water temperatures of 25.6 degrees. Although the triathletes in the mentioned study (Trappe, TA. 1995) experienced a decrease in core temperature after swimming, the core temperature rose to a level significantly above pre-immersed core temperature after 15 minutes of cycling\(^85\). Another study\(^86\) indicated that wearing a wetsuit during the swimming stage of an international distance triathlon in 25.4 degrees C water does not have adversely affect on the thermoregulatory responses of the triathletes on the subsequent cycling and running stages.

**Drafting during swimming.**
As in contrast to the cycle sequence of a triathlon, drafting is always legal when swimming. Drafting, leads to a decreased energy cost at a given speed and can be obtained either behind or to the side of another swimmer.\(^87\) Chatard (2003)\(^88\) found that the better drafting position was directly behind the lead swimmer (0 and 50cm) which led to a 21% and 20% reduction in passive drag, respectively. At this drafting distance, oxygen uptake was reduced by 11%, HR by 6%, blood lactate by 38%, RPE by 20%, and stroke rate by 6%, whereas stroke length was increased by 6%. Lateral drafting (0 to 50cm) and 50 to 100cm behind that of the lead swimmers hands led to a 6% and 7% reduction in drag, respectively. This energy saving strategy makes it possible for the triathletes to conserve energy for the cycle and running sequence. Delextrat (2003)\(^89\) showed in her study that cycling efficiency, when cycling 15 minutes at 75% maximal aerobic power and a freely chosen cadence, was improved by 4.8%, p<0.05. when swimming 750m at competition speed in a drafting position, as compared to swimming alone. Taken together, the above mentioned effects of wearing a wetsuit and drafting has shown to reduce the exercise intensity at the same relative swimming velocity and, although at a shorter distance (750m) than in Olympic distance, to improve cycle efficiency in the acute phase of the cycle stage.
Olympic triathlon swimming outlay

Recent research by Vleck et al. (2006)\textsuperscript{90} has shown that in an elite Olympic distance triathlon the Top50\% of overall finishers (n=12) swam significantly (p<0.05.) faster at 222m and 496m of a 1500m swim. The average swimming velocity was not significantly different (1.31 ± 0.02m/s-1 vs. 1.29 ± 0.04m/s-1) between the Top50\% and Bottom50\% of overall finishing position. The research concludes that the position at 400-500m into the swimming phase seems to reflect the overall finishing position during this elite Olympic distance triathlon.

To swim faster during the first 400-500m, and thus be among the lead swimmers, seems to be a major tactical advantage. The reasons for this may be due to the disadvantage of cuing, loss of arm stroke, power, speed and time as swimmers encounter the first buoy where the field of swimmers narrows. These disadvantages may quickly lead to a 5 to 10 seconds loss which seems impossible to regain\textsuperscript{91}. Secondly, surpassing slower swimmers in an outlay and then draft on slightly faster swimmers can be critical for performance, as drafting has shown to lower the relative intensity and cost of swimming.\textsuperscript{87,88} Thirdly, due to World Cup seating-rules, the fields’ best triathletes are seated in so that the better the seating, the more centrally triathletes will be located on the pontoon at race start. If the field and pontoon is large enough, one would be slightly disadvantaged by having to travel further in order to get to the first buoy, when starting more laterally on the pontoon. That positioning during the swim phase is of major importance is supported by Landers et al.(2000)\textsuperscript{92} who showed that thru 18 world cup competitions, 90\% of the elite male triathletes and 70\% of the elite female triathletes, the final winner of the competitions left the water with the first swimming pack.

Cycling in triathlon

Much in line with triathlon endurance performance factors, Atkinson et al. (2003), suggests that the key physiological factors for success in cycling are maximal aerobic power, muscle fibre type, cycling efficiency and lactate threshold.\textsuperscript{93} There is a variety of internal and external factors however that determines cycling velocity. When categorized these are divided into two groups; the physiological factors which influence mechanical power production (internal factors), and mechanical and environmental factors that affect power
demand (external factors), among which air resistance is recognized as the most important one at competition velocity (approximately 40 km/h).  

**Maximal aerobic power (MAP)**

It is common for researchers to refer to the aerobic power of cyclists in terms of maximal aerobic power (MAP). The reason for this is that most research has found major differences in the power produced at VO2max between highly trained cyclists and elite professional performers while there were minor differences in VO2max itself.  
The maximal power outputs typically range from 350 W to 525 W (5.5 to 7.6 W kg⁻¹) for top cyclists.  

**Drafting**

Drafting in triathlons during the cycling section is illegal in Norway, but allowed in most international races. When addressing drafting, one focuses on the decrease in frontal air resistance (pressure drag) when cycling in the slipstream of a leading rider. By slipstreaming, the drafting triathlete benefits from riding into a lower pressure vortex created by the triathlete in front. At similar speed the following rider may therefore experience a 30 % reduction in the required power output compared to that of the triathlete in front. It has also been shown that cycling behind a pack of 8 riders results in a much larger decrease in energy cost than does drafting behind one, two or four riders. 

More importantly, based on two studies (Hausswirth et al. 1999, Hausswirth et al. 2001) it is indicated that drafting and the decrease in metabolic cost of cycling is beneficiary in that it has influence on the VO2 that can be maintained during the subsequent running sequence.  

**Cadence (rpm)**

The optimal pedalling frequency is generally defined as the rate at which the efficiency is maximal. There seem to be disagreement about the optimal cadence. According to Gaesser & Brooks (1975) and Marsh et al. (2000), the rates producing the highest efficiencies are approximately 50-60 rpm, regardless of which definition of efficiency is used. Other researchers have found the optimal cadence to be between 60 to 80 rpm. Nevertheless, during steady-state cycling over level ground, cyclists choose pedal rates faster than the optimal one (80-100 rpm), even if this may lead to substantially larger energy expenditure. 

Hansen et al. (2008) found similar characteristics between chosen pedalling cadence and another voluntary motor rhythm, unloaded finger tapping with regards to steadiness and individuality. They
indicate that freely chosen cadence primarily is a robust innate voluntary motor rhythm, likely under the influence of central pattern generators that are minimally affected by internal and external conditions during submaximal cycling.

Hansen EA, et al., (2007)\textsuperscript{104} observed that heavy strength training for 12 weeks had subjects choose a 9 rpm lower pedal rate and improved their energy expenditure by lowering it by approximately 3%. This is in opposition to Sunde et al (2009)\textsuperscript{49} who did not find any changes in cadence after 8 weeks of maximal strength training.

**The effect of prior swimming on cycling.**

A study by Kreider, et al. (1988)\textsuperscript{7} showed that in a simulated triathlon (0.8km swim, 75-min cycling, and 40-min run) prior swimming lead to a decreased (p<0.05) power output in cycling (191± 4.2 to 159 ± 7.6 W) and mean differences (p<0.05) in oxygen uptake (3.18 ± 0.1 to 3.01± 0.1 L/min-1), ventilation (84.7± 4 to 80.4±4.2 L/min-1), stroke volume (128± 7.1 to 118± 3.5 ml/min-1) cardiac output (20.7±1.2 to 18.9 ± 0.8 L/min-1) mean arterial pressure (105 ± 3.8 to 96 ± 7.9 mmHg) and rectal temperature (38.2 ± 0.2 to 38.4 ± 0.3 degrees C) to that of control cycling.

**Residual effects from swimming to cycling sequence.**

Gonzales-Haro et al.\textsuperscript{105} has shown in a swimming, transfer and cycle simulation, that when elite triathletes swim 1500m at 1.29 ± 0.07m/s-1 (98 ± 2% MAS), transfers, and cycle on an ergo meter at competition speed for an hour, the mean power and VO2 drops to 266 ±34 watt, representing 77 ± 10% maximum aerobic power (MAP) and 82 % VO2max, respectively. At time intervals 08-10\textsuperscript{th} and 57-59\textsuperscript{th} minute the VO2 increases from 80 % VO2max to 87 % VO2max. At same time intervals, the power increased from 77% to 82%MAP, although showing a drop from 12-14\textsuperscript{th} minute. During the 1500m swim mean HR was 162 ± 15. Lactic acid (mMol) after T1 was 6.6 ± 1.8. The pedaling rate during the cycling sector averaged 99 r.p.m.

Delextrat et al.\textsuperscript{106} has studied the gross efficiency when cycling 30minutes on a cycle ergo meter at 95 r.p.m. is preceded by a 1500m swim. Results showed that when national triathletes swam 1500m in a 50m pool at mean 1.2m/s-1 in T 20m51s ± 3m2s, the continued ergometer cycling from 3\textsuperscript{rd} to 5\textsuperscript{th} minute changed significantly (p<0.05). The VO2 changed from 55.4 ± 3.5 to 58.2 ±
3.8ml/kg/min-1, and %VO2max from 77.1 ± 4.9 to 80.9 ± 5.3, gross efficiency (%) from 18.4 ± 2.2 to 16 ± 1.9. Heart rate changed from 150 ± 14 to 164 ± 7 beats/min-1, VE from 92.1 ± 9.9 to 106.6 ± 12.4L/min-1, and finally, respiratory frequency changed from 31.7 ± 5.1 to 38 ± 7.2 breath/min-1, from control cycling to the cycling preceded by swimming, respectively.

Aim of the present study

Since race position in the first 500m swimming during an Olympic triathlon is shown by Vleck et al (2006) to influence on overall finishing position, the aim of this study was to assess to what extent swimming at 100% of 500m velocity at the first 500m of a 1500m swim will influence on the average 1500m velocity and the intensity maintained on a cycle ergometer during a 22.5km time trial.
Methods

Subjects
Six triathletes, five males and one female, were included in this study, after each having received and signed consent forms approved by the Human Research Committee. The triathletes had all been participating in triathlon competitions and were considered well trained. Their mean age was $34 \pm 6$ years, height $181.5 \pm 4.7$ Cm, and weight $75.3 \pm 7.3$ Kg.

Protocol
The subjects were tested on four different days as depicted in table 1. Tests were organized according to the schedule below. Test days 3 and 4 were double blinded.

<table>
<thead>
<tr>
<th>Table 1.</th>
<th>Time organization of different test variables on different days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day</td>
<td>AM</td>
</tr>
<tr>
<td>1</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td></td>
</tr>
</tbody>
</table>

Anthropometry: measurements of height and weight, ISOS500max: time performance test in 500m maximal swimming, LT: measurements of lactate threshold, 22.5 km TT: time performance in 22.5 km time trial on the test ergometer cycle, S500max: time performance test in 1500m where the first 500m equals ISOS500max result, T1: transition between swimming and cycling, S1500m: time performance in 1500m swimming. Half of the triathletes were randomly assigned to complete the tests of day 4 the day before the tests of day 5.
**Experimental procedures**

VO2 was measured using the metabolic test system, Sensor Medics Vmax Spectra (SensorMedics 229 California, USA). Blood lactate \([\text{La}]_b\) measurements were performed using an Arkray Lactate Pro LT-1710 analyser (whole blood) (Arkray Inc. Kyoto, Japan). Cycling tests were performed on a modified ergometer cycle; Lode Corival V2, Groningen, the Netherlands. The cycle ergometer displays watt and RPM and was equipped with the triathletes own pedals prior to testing. Horizontal and vertical adjustments on sitting position were also done prior to all cycle ergometer testing.

**Testing procedures**

Swim tests were performed in a 25m indoor pool with water temperature of 27 degrees Celsius. Triathletes were, dressed in their wetsuit and, prior to entering the water, measured for resting blood \([\text{LA}]_b\). Post warm-up and post 500m maximal swim test \([\text{LA}]_b\) were also measured. The warm-up and 500m maximal swim test were interspersed with a 5 minute rest. The triathletes resting- and post warm-up heart rate was measured and were recorded continuously throughout the maximal swim test. Time for the warm-up and Time and split times for the 500m maximal swim test were measured by using a stop watch.

Cycling VO\textsubscript{2\text{max}} tests were performed using an incremental protocol with individual increments in watts to reach VO\textsubscript{2\text{max}} between 5 to 10 minutes of cycling. The criteria used for evaluation of VO\textsubscript{2\text{max}} were: a plateau in VO\textsubscript{2} despite an increase in power output, a heart rate (Hf) \(\geq 98\%\) of the predicted age related maximal heart rate, \(R \geq 1.05\), \([\text{LA}]_b \geq 8.0 \text{mMol}\cdot\text{l}^{-1}\), and voluntary exhaustion.

LT on ergometer cycling was defined as the warm up \([\text{La}]_b\) value (i.e. measured after the lowest brake power in day one) + 2.3 mmol. This is in line with the protocol proposed by Helgerud et al.
The test consisted of several five minute steps, increasing the power output by approximately 30 W after each step. The oxygen cost of cycling was measured at the same 5-minute steps.

The 22.5km time trial (TT) was immersed after a 10 minute warm up and a 1 minute rest where a lactate measurement was taken. The triathletes themselves determined power output and RPM during the test, being able to change the number of watts whenever they chose, with exception of the last 200 meters. During the TT, Hf, watts and RPM was recorded at minute 1, 2:30 and every 2:30 (min/sec) hence forth until test completion. VO2 measurements were collected at minute 3:30, 8:30 and every 10th minute from there on until test completion. The noted VO2 was calculated as the mean value of three consecutive readings. Within the first minute of test completion a second lactate measurement was taken.

The 1500 maximal swimming test with first 500m equal to personal best 500m time(S500max), was a replication of the 500m maximal swim test, only this time, [LA−]b was taken once, within one minute after the warm-up. The triathletes then swam as fast as possible, replicating their 500m maximal swim time ±5 seconds and continued with another 1000m swimming at self selected competition speed, completing the 1500m as fast as possible. Time, split times and Hf were recorded during the swim test. Completing the swim, triathletes went through transfer T1; changed
to cycling clothes and ran a distance of 190m with an elevation of 10m outdoors and entered the laboratory where ergometer cycling were immersed at the same wattage as during the isolated TT. Seated on the ergometer cycle, a second lactate measurement was taken. Triathletes then completed the TT as fast as possible, adjusting wattage and RPM freely. VO2, HR, wattage and RPM was recorded as in the isolated TT. On test completion a final lactate measurement were taken within the first minute.

Another 1500m swimming test was performed, only this time the 1500m swimming were done at self selected competition speed. This test was also followed by the ergometer cycle TT as described above.

Half of the triathletes were randomly assigned to complete the tests of day 4 the day before the tests of day 5.

**Allometric scaling**

Energy cost for movement does not increase in the same rate as body weight (Bergh et al. 1991). Allometric scaling per kg body weight raised to the power of 0.75 and metre has been reported to decrease the SD in cost of running Cr between subjects (Helgerud, 1994, Helgerud et al., 2001, Hoff et al., 2005). According to Åstrand & Rodahl (1986), VO2 values expressed per kg body weight raised to the power of 0.67 and metre is most appropriate in cycling. VO2 values are thus mainly expressed in ml·kg⁻⁰.⁶⁷ in the present study.

**Statistical analysis**

Statistical analyses were performed using the software program SPSS, version 13.0 (Statistical Package for Social Science, Chicago, USA). In all cases, p<0.05 was taken as the level of significance in two-tailed tests. Descriptive statistical analyses were made to display means and standard deviations. To compare means, T-tests were used. Correlations were calculated by the Pearson correlation test.
Table 2. Physical and physiological characteristics of triathletes

<table>
<thead>
<tr>
<th>Variables</th>
<th>n=6; 5 males and 1 female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>34.8 ± 6.4</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>75.3 ± 7.3</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>181.5 ± 4.7</td>
</tr>
<tr>
<td>VO₂max</td>
<td></td>
</tr>
<tr>
<td>ml·kg⁻¹·min⁻¹</td>
<td>63.2 ± 5.7</td>
</tr>
<tr>
<td>ml·kg⁻⁰·⁶⁷·min⁻¹</td>
<td>264 ± 32</td>
</tr>
<tr>
<td>HRpeak</td>
<td>180± 5</td>
</tr>
<tr>
<td>LT</td>
<td></td>
</tr>
<tr>
<td>%VO₂max</td>
<td>80.6 ± 3.3</td>
</tr>
<tr>
<td>W</td>
<td>278 ± 49</td>
</tr>
</tbody>
</table>

Values are mean ± SD
VO₂max: Maximal oxygen consumption, HRpeak: Peak heart rate at VO₂max, LT: Lactate threshold, W: Watt.
The mean and standard deviation of swimming time and heart rate for trials ISOS500max, S500max and S1500 are depicted in table 3.

### Table 3. Swimming time and heart rates

<table>
<thead>
<tr>
<th>Variables</th>
<th>n=6; 5 males and 1 female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time: (seconds-1) ISOS500max</td>
<td>489 ± 57</td>
</tr>
<tr>
<td>Time: (seconds-1) S500max</td>
<td>1558 ± 141</td>
</tr>
<tr>
<td>Time: (seconds-1) S1500</td>
<td>1568 ± 174</td>
</tr>
<tr>
<td>HR (beats ·min⁻¹) ISOS500max</td>
<td>151 ± 8</td>
</tr>
<tr>
<td>HR (beats ·min⁻¹) S500max</td>
<td>152 ± 12</td>
</tr>
<tr>
<td>HR (beats ·min⁻¹) S1500</td>
<td>150 ± 9</td>
</tr>
<tr>
<td>HRpeak (beats ·min⁻¹) ISOS500max</td>
<td>164 ± 9</td>
</tr>
<tr>
<td>HRpeak (beats ·min⁻¹) S500max</td>
<td>163 ± 9</td>
</tr>
<tr>
<td>HRpeak (beats ·min⁻¹) S1500</td>
<td>165 ± 8</td>
</tr>
</tbody>
</table>

Values are mean ± SD
Time: seconds-1, HR: mean heart rate during respective swim tests, HRpeak: Peak heart rate during respective swim tests, ISOS500max: time performance test in 500m maximal swimming, S500max: time performance test in 1500m where the first 500m equals ISOS500max result, S1500m: time performance in 1500m swimming.

The mean and standard deviation of swim trials time ISO500max, S500max, S1500 and transfer times S500max T1 and S1500 T1 is depicted in table 4.

### Table 4. Swim and Transfer (T1) times

<table>
<thead>
<tr>
<th>Variables</th>
<th>n=6; 5 males and 1 female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time: (seconds-1) ISOS500max</td>
<td>489 ± 57</td>
</tr>
<tr>
<td>Time: (seconds-1) 500m outlay S500max</td>
<td>492 ± 49</td>
</tr>
<tr>
<td>Time: (seconds-1) 500m outlay S1500</td>
<td>501 ± 04</td>
</tr>
<tr>
<td>Time: (seconds-1) Total S500max swim</td>
<td>1557 ± 141</td>
</tr>
<tr>
<td>Time: (seconds-1) Total S1500 swim</td>
<td>1567 ± 174</td>
</tr>
<tr>
<td>Time: (seconds-1) S500max T1</td>
<td>194 ± 25</td>
</tr>
<tr>
<td>Time: (seconds-1) S1500 T1</td>
<td>206 ± 37</td>
</tr>
</tbody>
</table>

Values are mean ± SD
ISOS500max: time performance test in 500m maximal swimming, S500max: time performance test in 1500m where the first 500m equals ISOS500max result, S1500m: time performance in 1500m swimming. T1: transition between swimming and cycling.
Time: seconds-1.
Physiological and time performance results obtained during swimming and cycling are depicted in table 5.

**Table 5. Physiological variables and cycling characteristics during 22.5km time trial sessions**

<table>
<thead>
<tr>
<th>Variables</th>
<th>n=6: 5 males and 1 female</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO2 22.5km TT (ml·kg(^{-1})·min(^{-1}))</td>
<td>53.6 ± 5.3</td>
</tr>
<tr>
<td>VO2 S500max TT (ml·kg(^{-1})·min(^{-1}))</td>
<td>51.9 ± 5.5*</td>
</tr>
<tr>
<td>VO2 S1500 TT (ml·kg(^{-1})·min(^{-1}))</td>
<td>51.5 ± 5.7*</td>
</tr>
<tr>
<td>VO2 ml·kg(^{0.67})·min(^{-1})</td>
<td>223 ± 28</td>
</tr>
<tr>
<td>%VO2max 22.5km TT</td>
<td>84.9 ± 2.8</td>
</tr>
<tr>
<td>%VO2max S500max TT</td>
<td>82.9 ± 3.9</td>
</tr>
<tr>
<td>%VO2max S1500 TT</td>
<td>81.6 ± 6.1</td>
</tr>
<tr>
<td>VE 22.5km TT (L·min(^{-1}))</td>
<td>133± 21</td>
</tr>
<tr>
<td>VE S500max TT (L·min(^{-1}))</td>
<td>130± 20</td>
</tr>
<tr>
<td>VE S1500 TT (L·min(^{-1}))</td>
<td>129± 23</td>
</tr>
<tr>
<td>R 22.5km TT (L·min(^{-1}))</td>
<td>0.95 ± 0.05</td>
</tr>
<tr>
<td>R S500max TT (L·min(^{-1}))</td>
<td>0.92 ± 0.06</td>
</tr>
<tr>
<td>R S1500 TT (L·min(^{-1}))</td>
<td>0.94 ± 0.06</td>
</tr>
<tr>
<td>HR 22.5km TT (beats·min(^{-1}))</td>
<td>162 ± 6</td>
</tr>
<tr>
<td>HR S500max TT (beats·min(^{-1}))</td>
<td>166 ± 4*</td>
</tr>
<tr>
<td>HR S1500 TT (beats·min(^{-1}))</td>
<td>167 ± 7*</td>
</tr>
<tr>
<td>Brake power 22.5km TT (W)</td>
<td>275 ± 58</td>
</tr>
<tr>
<td>Brake power S500max TT (W)</td>
<td>266 ± 53*</td>
</tr>
<tr>
<td>Brake power S1500 TT (W)</td>
<td>265 ± 52</td>
</tr>
<tr>
<td>CAD 22.5km TT (RPM)</td>
<td>104 ± 11</td>
</tr>
<tr>
<td>CAD S500max TT (RPM)</td>
<td>102 ± 12</td>
</tr>
<tr>
<td>CAD S1500 TT (RPM)</td>
<td>103 ± 11</td>
</tr>
<tr>
<td>CC 22.5km TT (ml·kg(^{0.67})·W(^{-1}))</td>
<td>0.752 ± 0.098</td>
</tr>
<tr>
<td>CC S500max TT (ml·kg(^{0.67})·W(^{-1}))</td>
<td>0.741 ± 0.091</td>
</tr>
<tr>
<td>CC S1500 TT (ml·kg(^{0.67})·W(^{-1}))</td>
<td>0.741 ± 0.085</td>
</tr>
<tr>
<td>WE 22.5km TT (%)</td>
<td>21.2 ± 2.2</td>
</tr>
<tr>
<td>WE S500max TT (%)</td>
<td>21 ± 1.5</td>
</tr>
<tr>
<td>WE S1500 TT (%)</td>
<td>20.8 ± 1.0</td>
</tr>
</tbody>
</table>

Values are mean ± SD
VO2: Oxygen consumption, % VO2: Percentage of maximal oxygen consumption, VE: Volume of Ventilation, R: Respiratory Quotient, HR: Heart rate, W: Watts, CAD: Cadence, CC: oxygen cost of cycling, WE: Work Efficiency, 22.5 km TT: 22.5 km time trial on cycle ergometer, S500max TT: 22.5 km time trial on cycle ergometer after time performance swimming test in 1500m where the first 500m equals ISOS500max result, S1500m TT: 22.5 km time trial on cycle ergometer after time performance swimming test in 1500m.
*p<0.05 difference from 22.5km TT value
Lactate responses to the isolated Time Trial 22.5kmTT, the ISO500max, the S500maxTT and S1500TT are depicted in table 6.

**Table 6. Blood Lactate concentration \([\text{L}a^-]\) pre- and post tests**

<table>
<thead>
<tr>
<th>Variables</th>
<th>n=6; 5 males and 1 female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-test mMol La- 22.5km TT</td>
<td>1.9 ± 0.9</td>
</tr>
<tr>
<td>Post-test mMol La- 22.5km TT</td>
<td>9.9 ± 4.5</td>
</tr>
<tr>
<td>Pre-test mMol La- ISO500max</td>
<td>2.8 ± 1.0</td>
</tr>
<tr>
<td>Post-test mMol La- ISO500max</td>
<td>7.2 ± 1.8</td>
</tr>
<tr>
<td>Pre-test mMol La- S500max TT</td>
<td>5.1 ± 1.2</td>
</tr>
<tr>
<td>Post-test mMol La- S500max TT</td>
<td>8.1 ± 1.5</td>
</tr>
<tr>
<td>Pre-test mMol La- S1500 TT</td>
<td>4.7 ± 1.7</td>
</tr>
<tr>
<td>Post-test mMol La- S1500 TT</td>
<td>8.4 ± 3.3</td>
</tr>
</tbody>
</table>

Values are mean ± SD
La⁻ : Lactate in Millimoles per Litre, ISOS500max: test in 500m maximal swimming, 22.5 km TT: 22.5 km time trial on cycle ergometer, S500maxTT: 22.5 km time trial on cycle ergometer after time performance swimming test in 1500m where the first 500m equals ISOS500max result, S1500m TT: 22.5 km time trial on cycle ergometer after time performance swimming test in 1500m.

Time performance results from the 22.5 km cycle ergometer time trial with and without the prior swimming tests are depicted in table 7.

**Table 7. Time for completion of various Time Trials**

<table>
<thead>
<tr>
<th>Variables</th>
<th>n=6; 5 males and 1 female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time: (seconds-1) ISO22.5km TT</td>
<td>2183 ± 514</td>
</tr>
<tr>
<td>Time: (seconds-1) S500max TT</td>
<td>2264 ± 583</td>
</tr>
<tr>
<td>Time: (seconds-1) S1500m TT</td>
<td>2272 ± 486</td>
</tr>
</tbody>
</table>

Values are mean ± SD
Time: minutes: seconds-1.
ISO22.5 km time trial on cycle ergometer, S500maxTT: 22.5 km time trial on cycle ergometer after time performance swimming test in 1500m where the first 500m equals ISOS500max result, S1500m TT: 22.5 km time trial on cycle ergometer after time performance swimming test in 1500m.

The results showed a significant difference (3.3%) between mean power produced during the 22.5km time trial and the S500max TT, accompanied by a 4% reduction in mean VO2 and a 2.5% increase in HR. No difference in mean power produced, VO2 or HR between the 22.5km TT and S1500 TT was apparent.
Discussion

**Swimming outlay**
The main result in this master thesis is that no significant differences between the two sequential trials S500max and S1500 were apparent. As such, the results showed no detrimental effects of a maximum 500 meter swimming outlay on the following cycling performance, supporting that a fast swimming outlay, where the triathletes surpasses the slower field of swimmers, has a tactical advantage.

On the other hand, the group of triathletes participating in this study showed a rather poor swimming capability with mean 1500m swimming time performance approximately 10 minutes slower than world class triathletes. It is possible to imagine that the S500max outlay, because of a higher maximal swimming velocity, would result in a larger degree of fatigue during the last 1000m of the 1500m swim. The tactical benefit of a maximal 500m outlay may thus be reduced.

In Norway, there exists a non-drafting rule that gives triathletes with a large cycling capability an advantage so that a strong swimmer with lesser cycling capacity cannot draft on the stronger cyclists during the cycling part. The strong swimmer may thus loose position in the field to stronger cyclists. When Norwegian triathletes are to participate in competitions outside Norway where drafting is legal, the benefits of a strong swimming outlay becomes even more evident.

One could argue that because drafting in swimming has been found to lower La- concentration\textsuperscript{108} and furthermore, improve cycling efficiency\textsuperscript{109} the benefit of a strong swimming outlay where the triathletes are positioned so that he/she may draft on slightly better swimmers, is present, even in a non-drafting race.

Mean HR during swimming in the present study was 152 ±12 and 150 ± 9beats/min during the S500max and S1500, respectively. At first hand one might expect the mean S500max HR to be significantly higher than S1500 HR. However, it seemed that although the fast outlay in S500max significantly increased HR compared to the outlay in S1500, the mean HR in the respective swim trials is approximately the same since HR towards the end of S1500 is elevated as the strategy, spontaneously chosen by the triathletes, is to increase power output towards the end of swimming. This is also indicated by the speed attained during the last 1000meters of S500max and S1500 (1.07 and 1.06) m/sec-1., respectively, which is actually higher than the speed obtained in their outlays (0.98 and 1.0) m/sec-1., respectively.
The better swimmers in the present study had the highest relative HR during the swim. This indicates that a high swimming capacity enables the triathletes to elevate their relative HR more than that of triathletes with inferior swimming capacity. This is in accordance with Gonzales-Haro et al., (2005)\textsuperscript{105} where good swimmers had a mean HR during swimming close to mean HR during cycling (162 ± 15) VS. (162 ± 13)beats/min-1. The triathletes participating in Gonzales-Haro (2005) had an average 1500m swimming speed of 1.29 m/sec.

**Transfer one (T1)**

The transfer T1 lasted 03:14 min and 03:26 min for S500max and S1500, respectively. The time spent on T1 is approximately three times that of the elite male triathletes during the 1997 and 1998 Triathlon World Championship (01:12 ± 00:16 SD) equivalent of 1.0 ± 0.2 SD percentage of total race time. Since there is no standardisation in the length of T1 the time spent often varies between events.\textsuperscript{110} However, in the present study, one could speculate that the undulating course of running during T1, and hence, the comparatively long time spent on T1, could alter the physiological premises for the ergometer cycling. As compared to the study by Gonzàles-Haro et al.\textsuperscript{105} who had triathletes run 100m during T1 and who showed that at the end of their 1500m swimming sequence, triathletes [La-] was 6.8 ± 2.1 mM, similar to that at the end of T1 (6.6 ± 1.8 mM). Delextrat et al.\textsuperscript{106} had a standardized T1 transfer time of 3 minutes, but had all cycling tests performed near the swimming pool. Their average [La-] after T1 was 6.9 ± 2.6 mM. This is higher [La-] than what was observed in our present study (5.1mM ± 1.1 and 4.7mM ± 1.6) for the S500max and S1500, respectively.

**Ergometer cycling**

The watt produced during TT (275 ± 57) decreased significantly (p>0.05) to 266 ± 53 (S500max TT), which in turn was almost identical to the wattage during S1500 TT. These changes co-existed with a significant increase in HR from 162 ± 6 to 166 ± 4 and 167 ± 7, respectively.

The increase in heart rate during S500max TT and S1500 TT may be explained by a drop in mean arterial pressure, as shown by Kreider (1988)\textsuperscript{7} in a somewhat similar study. The increased heart rate could also be a symptom of hyperthermia, elevated muscle- and core temperature, as discussed in Gonzales et al. (2003, 2008).\textsuperscript{111, 117}
During all swimming trials the triathletes wore wetsuits. With a water temperature of 27 degrees Celsius, a warm up and an intense exercise trial of approximately 25 minutes, it is likely that core temperature may have been elevated, although this was not tested in the present study. A simultaneous elevation of muscle- and core temperature (hyperthermia) has been recognized to be affecting performance by changing glycogen utilization, blood glucose concentration, noradrenaline and heart rate (Gonzales, et al 2003).\textsuperscript{111} Hyperthermia has previously been observed to lower oxygen consumption with the reductions primarily being a function of hyperthermia related lowering of cardiac output and mean arterial pressure and their associated effects on skeletal muscle blood flow and oxygen uptake and delivery.\textsuperscript{111} Dehydration causing lowering of blood volume facilitates an increase in heart rate (Frank-Sterling mechanism and Tachycardia).\textsuperscript{112} Other investigators found that during intense cycling, hyperthermia alone (+1 degree esophageal and +6 degrees C. skin Temp.), or in combination with dehydration (4%), did not alter the initial VO2 kinetics but reduced VO2max by 16% and performance time by 51-53%. The reduction in VO2max lead to a proportional reduction in O2 pulse and significantly elevated maximal heart rate from 190 at normal state, to 195 at hyperthermia.\textsuperscript{113} Based on these studies, it is reasonable to suspect that our triathletes might have been exposed to elevations in core temperature and dehydration. However, core temperature and body weight post tests was not measured.

Glycogen depletion may be implicated in a reduced endurance performance. Since the trials in our study approximates 66 minutes of high-intensity work-load, and that most of the trials S500max and S1500 were done within 48 hours, the triathletes may have been exposed to glycogen depletion, and could possibly have benefited from CHO loading. The present study did not include a protocol for hydration and CHO loading, except that the triathletes were free to hydrate whenever they wanted post swimming. The contents of their beverages were not included in this study either. Thus, the differences in supplements of the triathletes’ beverages could have an effect on the triathletes’ performance in this study which is unaccounted for.
According to results from Gonzales-Haro (2005)\textsuperscript{105}, there is reason to suspect that triathletes, if they had not been aware of their present power output, would naturally select a mean power output at- or close to their individual lactate threshold. However, Kenefick (2002)\textsuperscript{122} showed that a 20km TT could be finished with a $[\text{La}^-]_b$ of approximately 250\% LT. In the present study, the triathletes completed 22.5kmTT with a $[\text{La}^-]_b$ of approximately 240\% LT, S500max TT and S1500 TT with a $[\text{La}^-]_b$ of approximately 215\% LT. One has to keep in mind, however, that our triathletes were aware that there was no consecutive running stage which partly explains the increase in their intensity towards the end of the TT, knowing that the physical strain were near an end.

The freely chosen cadence among the triathletes in this study was much higher (approximately 100-105 rpm) than what has previously been reported as the most energetically optimal one (Hansen et al 2002).\textsuperscript{54} As mentioned by Hansen et al. (ibid, 2002) subjects with a majority of MHC I might select these high pedalling rates to let fast twitch fibres work in an energetically optimal manor in order not to exhaust slow twitch fibres. Since our study did not examine the subjects relative proportion of fast and slow twitch muscle fibres, we can only speculate that a high MHC I content and a high pedalling rate, lowers neuromuscular fatigue and that this might be the reason for the adapted high pedalling rate. The high freely chosen cadence may also reflect the triathletes robust innate voluntary motor rhythm, minimally affected by internal and external conditions as proposed by Hansen et al. (2008).\textsuperscript{103}

The mean fractional utilisation of VO\textsubscript{2} max during time trials did not significantly decrease when preceded by swimming. However, the results indicate a lowering in VO\textsubscript{2} in accordance with the lower power output after prior swimming; from 22.5km TT (84.9 ± 2.8) VS. S500max TT (82.1 ± 3.9) VS. S1500 TT (81.6 ± 6.1). This is in contrast to Delextrat et al., (2005)\textsuperscript{106} who, during the 3\textsuperscript{rd} to 5\textsuperscript{th} minute, showed significant differences in %VO\textsubscript{2}max (77.1 ± 4.9) VS. (80.9 ± 5.3) in their control cycle trial and swim-to-cycle trial, respectively.

Taken together, the differences in VO\textsubscript{2} and fractional utilisation of VO\textsubscript{2}max during the 3\textsuperscript{rd} to 5\textsuperscript{th} minute the two studies in between, could be due to methodological differences and/or the triathletes physiological capacity. For instance, Delextrat wanted to examine the effects of a preceding 1500m swim on a cycle trial of 30 minutes at 75\%MAP and 95 r.p.m. In our study, we examined the effects of two different swimming outlays on two maximal cycling time trials with a freely chosen r.p.m. and wattage. As for the physiological capacity, Delextrat et al., (2005)\textsuperscript{106} used triathletes that had
competed at the national level for at least three years and had a VO2max of 71.9 ±7.8 ml/kg1/min. One also has to take into account, that in the present study, the number of subjects studied was six (n= 6). If we were to increase the number of subjects by two (n= 8) multiplied with mean decline in %VO2max for each time trial during the 3rd to 5th minute, respectively, the differences in %VO2max from 22.5km TT to S500max TT and S1500 TT, respectively, would become significantly different. This indicates that the low number of triathletes included in this study presents a statistical problem in that it under-represents the changes in %VO2max between the different time trials.

Table 8. Comparison chart of 3 simulated Olympic triathlons, modalities swimming and cycling, and 1 Olympic triathlon, their parameters and results.

<table>
<thead>
<tr>
<th>Wetsuit</th>
<th>1500m speed (m/sec-1.)</th>
<th>Pre-cycle La- (mMol)</th>
<th>Post-cycle La- (mMol)</th>
<th>%VO2max</th>
<th>Watt</th>
<th>CAD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gonzales-Haro (2005) yes 1.29</td>
<td>6.6 ±1.8</td>
<td>8.8 ±1.5</td>
<td>82.8</td>
<td>266</td>
<td>99</td>
<td></td>
</tr>
<tr>
<td>Delextrat (2005) no 1.2</td>
<td>6.9 ±2.6</td>
<td>5.9 ±1.3</td>
<td>81.2</td>
<td>275</td>
<td>95</td>
<td></td>
</tr>
<tr>
<td>Schiødt (2010) S500max TT yes 1.04</td>
<td>5.1 ±1.2 /</td>
<td>8.1 ±1.5 /</td>
<td>82.9 /</td>
<td>266 /</td>
<td>102 /</td>
<td></td>
</tr>
<tr>
<td>S1500 TT yes 1.04</td>
<td>4.7 ±1.7</td>
<td>8.4 ±3.3</td>
<td>81.6</td>
<td>265</td>
<td>103</td>
<td></td>
</tr>
<tr>
<td>WC 2000 Sydney, Australia</td>
<td>263</td>
<td>70</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are mean ± SD. Wetsuit: wearing, m/sec-1: speed in meters per second, La-: Lactate in Millimoles per Litre, %VO2: Percentage of maximal oxygen consumption, Watt, CAD: Cadence.

Delextrat (2005)\textsuperscript{106} also showed that VE (L/min-1) from the 3rd to 5th minute when cycling changed significantly from (92.1 ± 9.9) to (106.6 ± 12.4) when preceded by swimming. She also mentions that this variable was the only one significantly different (p< 0.05) (101.3 ± 13.1) to (109.3 ± 11.1) at the end of cycling (28th to 30th minute). In the present study mean VE (L/min-1) during the 3rd to 5th minute changed significantly (p= 0.01) in between the 22.5km TT (120 ± 14) and the S500max TT (128 ± 14) with almost the same VE as the S500max TT in the S1500. Interestingly the VE increases while the VO2 decreases after swimming. This indicates in one way or another, that the prior work done during swimming has an effect on the ventilation during the first 5 minutes of subsequent cycling. The triathletes in the present study still showed a significant difference in VE when completing the trials in so that VE 22.5km TT (142 ± 25) was significantly different from VE at completion of S500max TT (134 ± 25). The VE at completion of 22.5km TT was not significantly different from VE when completing S1500 TT (134 ± 29).
In the present study no changes in $C_C$ were apparent from 22.5km TT to both S500max TT and S1500 TT. The same applies for WE, which isn’t surprising since the two variables represent almost the same.

VCO2 during time trials showed significant differences ($P= 0.02$) between 22.5km TT and S500max and S1500 TT. This may indicate that a greater contribution of energy during the 22.5km TT was derived from the TCA cycle (buffering) than that of the S500max TT and S1500 TT, and that the triathletes completed the 22.5km TT at a higher relative intensity.
The mean respiratory exchange ratio (R) for the 22.5km TT was $0.95 \pm 0.05$ versus the S500maxTTs’ $0.92 \pm 0.06$.

**Conclusion**

In a non-drafting Olympic triathlon, triathletes of mediocre swimming capacity, may benefit strategically and physiologically from a 500m maximum swimming outlay with no detrimental effects, compared to a more moderate swimming outlay.

**Limitations of the present study**

Further studies on this subject could benefit from including more subjects, using a flume pool with a lower water temperature, and include triathletes of a larger swimming capacity. The present study did not take into account measurements of efficiency like Distance Per Stroke (dps) during swimming, which would have been beneficiary for our understanding of the triathletes swimming performance. Also, one would benefit from replicating dietary intake 24-hours previous to S500max and S1500 and standardizing the amount and content of a consumed carbohydrate-protein beverage. Furthermore, one should also weigh athletes at completion of all trials. To observe changes in core temperature (Ctemp) and Qc, measurements of core temperature and Qc (ecg readings) during ergometer cycling could be beneficiary as well.
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Conversation with the Danish national triathlete head-coach Michael Krüger.


