



Temperature responses to exercise and performance

Jonathan Dugas

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Temperature responses to exercise and performance

By

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This thesis is dedicated to my wife, partner, and best friend, Lara.

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Declaration

I, Jonathan Paul Dugas, do hereby declare that the experiments presented in this thesis were conceived and executed by myself except where otherwise stated.

Neither the substance nor any part of this thesis has been submitted in the past, is being submitted, or is to be submitted for a degree at the University of Cape Town or any other academic institution.

This thesis is presented in fulfillment of the requirements for the degree of Ph.D.

I hereby grant the University of Cape Town free license to reproduce this thesis in part or whole for the purpose of research.

Signed: Signed by candidate _____

Jonathan Paul Dugas

Date: 22/05/06

List of publications

Dugas JP. Sodium ingestion and hyponatremia: Sports drinks do not prevent a fall in serum sodium concentration during exercise (Letter to the Editor). *Br J Sports Med.* 2006 April; 40(4):372-74.

Noakes, TD, Sharwood KA, Speedy D, Hew-Butler T, Reid S, Dugas JP, Almond C, Wharam P, Weschler L. Three independent biological mechanisms cause exercise-associated hyponatremia: Evidence from 2,135 weighed competitive athletic performances. *Proc Nat Acad Sci.* 2005 December; 102(51):18550-555.

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Abstract

The temperature responses to exercise have been a much investigated topic of intense research interest over the past 50 years. More recently, the effects of fluid ingestion on temperature regulation have been the focus of this area. The aim of this thesis is to undertake research to evaluate what has become the established dogma in this field and to determine whether a new model might better explain thermoregulation in humans during endurance exercise.

Study 1

The first study of the thesis examined fluid replacement strategies in 196 (men and women) finishers of a local one-day, 109 km cycling race. From this larger population, 60 men and 60 women were matched for finishing time and their data were compared. The major finding of this study was that although the men and women consumed similar total volumes of fluid during the race, because of the women's significantly smaller mass, they consumed more fluid on a relative basis. Although pre-race plasma $[Na^+]$ was not measured, the women had a significantly lower post-race plasma $[Na^+]$, suggesting that the drinking behavior of the women was not ideal. This raises the possibility that universal fluid replacement guidelines for athletes are perhaps misplaced, and that any drinking guidelines for exercise should differentiate between sexes. This would reduce the probability that women will overdrink and therefore be at risk of developing hyponatremia during exercise.

Although many variables were measured in this study, post-race rectal temperatures were not. This led to the next study in which temperatures were measured continuously during prolonged exercise.

Study 2

The second study investigated the rectal temperature responses during endurance running with a novel technique. Collaboration with a local technology firm (Sygade) produced 10 miniature data loggers which could log rectal temperature, heart rate, and air pressure at frequencies up to 75 times per minute. Because the data loggers were lightweight and portable, transport of the loggers on an athlete's person was possible without the equipment interfering with the athletes' performance. This new equipment produced novel data showing that 1) rectal temperatures oscillate within a narrow range during prolonged exercise, and 2) even in cool conditions recreational runners reach relatively high peak rectal temperatures ($> 39\text{ }^{\circ}\text{C}$) relatively early in the race and not at the finish. This study also showed that although subjects became more dehydrated as the race progressed, T_{re} did not rise inexorably and peak at the end of the race. Instead, the data showed that the runners regulate their temperature successfully within a two degree range even when dehydrated.

Study 3

The next study used the same data loggers from Study 2 and recorded the rectal temperature responses in six highly-trained cyclists and one recreational cyclist who completed a one-day 109 km cycle race. The findings in this study were similar to Study 2. Rectal temperatures oscillated during the race, presumably in response to changes in metabolic rate, although actual metabolic rates were not measured, and peak temperatures were below $39\text{ }^{\circ}\text{C}$. This suggested that even in highly-trained athletes who become progressively more dehydrated over the course of a race, the risk of heat stroke is small. However, one difference between these two studies was that six of the athletes in this study were competing at a high level (top 5% of finishers). In these more highly-trained athletes, peak rectal temperatures were not always achieved in the early stages of the race, and instead were reached by many at the finish. This suggests that these athletes successfully implemented a pacing strategy that allowed

them to complete the race safely, reaching the highest temperature only at the end of the race just prior to stopping exercise, after which the rectal temperature dropped rapidly without any specific intervention aimed at achieving this result. The findings from this study were confirmed in Study 4.

Study 4

Having examined the temperature responses to endurance exercise during actual endurance events, the methodology was moved to the laboratory to examine the thermoregulatory responses of highly-trained male cyclists completing self-paced 80 km time trials in hot and humid conditions. The volume of ingested fluid was manipulated to create different levels of dehydration in the same subjects at the end of each trial. Again the major findings from this study confirmed those from the previous studies, namely that similar peak rectal temperatures are achieved regardless of the exercise intensity and environmental conditions. In addition, this study showed that when an athlete is aiming for the best result possible as in an individual time trial, the rectal temperature response is different compared to when the athlete's goal is purely to complete the event. In recreational athletes in the field setting, the rectal temperature appears to oscillate frequently as the athlete changes the metabolic rate by slowing or stopping momentarily. However in more trained athletes in a time trial situation, the rectal temperature rises sharply during the initial 20-30% of the trial, before rising much less rapidly during the middle of the trial. Finally, at some point within the final 30-40% of the trial, as the athlete makes a conscious decision to increase the power output in a race for the finish, the "end spurt," and the rectal temperature again begins to rise more sharply. The peak rectal temperature is then achieved at the termination of exercise. This would seem to be the result of a carefully executed, conscious and subconscious, learned pacing strategy.

Study 5

One subject from Study 1 was found to have hyponatremic encephalopathy following the cycle race. During the course of the cycle race she gained 2.4 kg and presented to the research team with a plasma $[\text{Na}^+]$ of $129 \text{ mmol}\cdot\text{L}^{-1}$. Her weight gain and her ensuing hyponatremic encephalopathy were achieved in spite of a relatively modest fluid intake of $735 \text{ mL}\cdot\text{h}^{-1}$. This was a novel finding and warranted further investigation. Therefore the athlete was invited to undergo exercise testing in the laboratory to determine her sweat rate and the concentration of Na^+ in her sweat. The findings revealed that this individual had a relatively low rate of sweating ($520 \text{ mL}\cdot\text{h}^{-1}$) even in wind-still laboratory conditions, and a normal concentration of Na^+ in the sweat ($68 \text{ mmol}\cdot\text{L}^{-1}$). These findings helped reinforce the idea that fluid replacement guidelines should not be universal in nature, and should perhaps not even prescribe minimal hourly volumes, as this athlete was consuming fluid at a rate far below the current prescription yet she still finished the race with a dangerously low plasma $[\text{Na}^+]$.

Study 6

Due to a remarkable and unique opportunity, it became possible to investigate the rectal temperature responses to swimming in extremely cold water ($0\text{-}5^\circ\text{C}$). Data were collected with a custom built telemetry unit that measured the rectal temperature of a cold-water swimmer during record-breaking swims at 80°N (Verlegenhukun) in the Arctic Ocean and at $65^\circ 10' \text{S}$ (Petermann Island) near the Antarctic Peninsula, and during an additional one-mile swim at $62^\circ 56' \text{S}$ (Deception Island). The data illustrate the human body's remarkable capacity to defend its core temperature, whether the challenge is heat or cold. Most remarkable about these data was that during ~ 20 minutes of exposure to $0\text{-}5^\circ\text{C}$ water, this swimmer's rectal temperature changed by less than two degrees Celsius. He experienced the classic "after drop" that has been well-documented, but never reached critically low core temperatures

after either of his record swims, either training or open water. However, after swimming one mile in 30:30 at Deception Island, the swimmer reached a T_{re} of 33.6 °C in the minutes following this swim. Muscle temperature (vastus lateralis) was measured during the recovery period following the Deception Island swim, and reveals more novel findings about how the body defends its core temperature.

Summary

The original conclusions from early researchers that rectal temperature is determined primarily by metabolic rate were initially propounded in the 1930's. However approximately 30 years later the prevailing conclusion about human thermoregulation changed so that cardiovascular function was thought to be the primary predictor of rectal temperature by lowering the capacity to sweat and to conduct heat from the core to the skin. However, the studies from this thesis support the conclusions of the original researchers that metabolic rate is the largest predictor of rectal temperature, and that any effects of dehydration on rectal temperature are small in relation to the effects of metabolic rate.

List of Abbreviations

°C	Degrees Celsius	PV	Plasma volume
CHO	Carbohydrate	PPO	Peak power output
°F	Degrees Fahrenheit	Q	Heat content
g	Grams	Q _s	Heat storage
h	Hours	RH	Relative Humidity
HR	Heart rate	s	Seconds
kCal	Kilocalories	T	Temperature (Dry bulb unless otherwise indicated)
kJ	Kilojoules	T _{re}	Rectal temperature
km	Kilometers	T _{skin}	Skin temperature
L	Liters	VO ₂	Oxygen uptake or consumption
m	Meters	VO _{2max}	Maximal oxygen uptake
min	Minutes	W	Watts
mL	Milliliters		

Chapter 1

Literature Review

Introduction

Dehydration and its effects on human physiology and, in particular, thermoregulation, has been studied in much detail for the greater part of the 20th century (2; 5; 19; 21; 41; 54; 72; 118-120; 136; 138; 141; 147; 158; 159; 193; 203; 213; 219; 256) and continues to be a much investigated and debated topic. Included in this research are several different associated aspects of physiology such as thirst, heat balance, core temperature (T_c) regulation, skin blood flow, plasma osmolality, and sweat rate, among many others. (57-61; 160)

Perhaps the most important early researcher in this area was E.F. Adolph (4), who together with his associates performed a series of experiments in the 1940's on soldiers in the desert. He measured primarily weight losses and estimated sweat losses from these values, but published a volume of unique observations about fluid balance in humans. (4)

In the 1960's and early 1970's as the discipline of Exercise Physiology began to grow, a new generation of scientists accepted the challenge of studying human physiology during exercise and in varied environments. In this era researchers such as Saltin (201), Greenleaf (55; 86; 87; 89-91), Ekblom (54; 55), Pugh (191-193), Gisolfi (71; 72; 129), and Costill (37; 41) began to investigate fluid status and its associated aspects during prolonged exercise in both neutral and thermally stressful conditions, and published some of the classic studies and observations of this profession.

In 1969 Wyndham and Strydom (256) published what was considered by many to be a watershed study in this area. They examined post-race rectal temperatures after two 32 km runs in mild ambient conditions and found that the runners who lost the most weight also had the highest rectal temperatures. Although it is now known that they misinterpreted their results

(172), this article and others from that era (41; 72) laid the foundations for what I believe to be the myth that dehydration is a dangerous condition that should be avoided at all costs. This led to more research that focused on euhydration and the effects of maintaining body weight during prolonged exercise. Because some early studies found that fluid consumption during fixed-workload exercise attenuated the rise in rectal temperature (2; 41; 72), it was thought that the maintenance of body weight would therefore prevent the core temperature reaching 42° C, the temperature at which heat stroke is diagnosed and an individual's condition becomes critical, and active cooling is required to save the athlete's life.

In the 1980's the focus of the research in this area leaned towards a dehydration-rehydration oriented model to investigate different rehydration techniques that would best restore an individual's fluid status to pre-exercise levels (30; 252). These studies focused on testing the efficacy of different (hypertonic or isotonic or hypotonic) solutions in returning the subjects' plasma volumes and body masses to pre-exercise levels, or on the effect of these different rehydration techniques on selected aspects of thermoregulation in a subsequent exercise bout immediately following the rehydration. What these studies showed was that when humans begin an exercise bout in a hypohydrated state, that is, less than fully hydrated, the thermoregulatory response is indeed altered. The onset of sweating is delayed, leading to a steeper rise in core temperature during the initial period of exercise. However, although hypohydration appears to alter thermoregulation during the initial stages of exercise in the heat, it was not investigated whether or not the final or peak core temperature was altered during the post-rehydration exercise bout, or whether or not exercise performance was affected. Therefore it is possible that even though the slope of the sweating response curve is altered by hypohydration, the overall ability of the body to thermoregulate itself might not be altered as would be shown by a higher peak core temperature.

Research published in the early 1990's (147; 149) built on the foundation of earlier research and began to use different interventions *during* exercise to examine the effects of dehydration on thermoregulation in hot and humid environments. This line of research appears to have contributed greatly to the formation in 1996 of the American College of Sports Medicine's most recent fluid replacement guidelines (40). These guidelines, although based on studies with primarily young, well-trained male subjects exercising at constant and relatively high workloads, advise participants in physical activity to consume fluid in equal amounts to weight losses or to consume as much fluid as tolerable (40). These guidelines promulgated the belief that any amount of dehydration is a dangerous condition, and dehydration itself is something which is to be avoided at all costs. It is now believed that this advice led subsequently to many reported (6; 50; 64; 68; 69; 75; 97; 106; 161; 186; 220; 221; 223-226; 228; 229; 258) (and presumably many more unreported) cases of hyponatremia by encouraging recreational athletes to consume excessive volumes of fluid to prevent "voluntary dehydration." More recently it has been shown that excessive fluid intake is one of three conditions that in combination cause exercise-associated hyponatremia (EAH) (6; 98), although some questions remain concerning the prevalence of hyponatremia in women compared to men and the exact reasons why women present more frequently than do men with EAH.

Although the concept that too much fluid causes EAH is more widely accepted now, the concept that "voluntary dehydration" is to be avoided since it will cause the inevitable rise in core temperature to heat stroke levels has been widely propagated in scientific texts for many years (10; 17; 32; 42; 85; 88; 90; 171; 204; 231; 232).

This review will examine dehydration and its much researched effects on the body's thermo-regulatory abilities during sub-maximal endurance exercise, with emphasis placed on field studies and exercise during actual events. In addition, it will review critically the research design limitations and interpretation of data from these studies and examine their importance in formulating current fluid replacement guidelines. Finally, it will review original investigations performed in the 1930's through to the present and examine the history of fluid replacement research during endurance exercise.

Heat loss and thermal balance

Two primary models exist that scientists use to explain heat balance in humans. The set point model described by Cabanac (28) states that body temperature is regulated to a specific value, namely, 37 °C, and that behavioral and physiological mechanisms work to maintain this set point. A second model as described by Webb (249) states that body heat is the regulated variable, and that body temperature is a result of the body's effort to maintain the balance of heat loss and heat gain.

Cabanac addresses the nature of regulatory systems in biology and adopts an engineering-oriented approach to describe how the nature of these systems dictates that body temperature is the independent variable (28). The primary reason given for reaching this conclusion is that all humans including lower-limb amputees, newborns, and healthy adults all regulate variables such as temperature, blood pressure, and electrolyte concentrations at the same values, although their blood masses, electrolyte contents, and heat contents vary widely.

Webb however, argues that body heat content is the regulated variable (249). In his model of temperature regulation, T_{re} is a dependent variable and heat production (metabolic rate) is

the independent variable, which varies widely according to the activity level of the individual at any given moment. Webb describes a control scheme in which the “temperature controller” maintains the body temperature by sensing heat loss and altering the heat-losing mechanisms so that heat loss matches heat production. He hypothesizes that this controller maintains the body temperature within a narrow range by responding to neural signals in the transcutaneous temperature gradient(s).

However, regardless of which model is applied, T_c is maintained within a range, as this is the nature of a homeostatic system. Humans have a vast array of resources and mechanisms with which they can maintain their core temperature (T_c) within a desirable range, specifically 36-40 °C. This implies that although, at rest, humans maintain a T_c of approximately 36.5-37.5 °C, during exercise the core temperature can be maintained between 37-40 °C with no adverse effects except perhaps some effect on exercise performance in highly trained and motivated athletes. In addition, the temperature response varies among individuals (Chapters 3 and 4), and rectal temperatures as high as 41.7 °C have been recorded without symptoms in healthy individuals during competitive exercise (138; 198).

The evidence from athletes competing in recreational races indicates that humans can tolerate relatively high ambient temperatures (≥ 30 °C) and have a large capacity to cope with hot and humid environments (136-138; 158; 193), although performance has been shown to be reduced in these environmental conditions (26; 35; 108; 135; 234; 241). This tolerance is achieved by a series of behavioral and physiological responses.

First and most important, a conscious decision is made by an individual to commence exercise in a given environment. This act itself illustrates the ability of humans to regulate their tem-

perature, for if temperature regulation were an issue during physical activity in hot and humid environments then, certainly, the choice would be made not to engage in the activity. Having made this decision, the next behavioral outcome entails setting the intensity of the exercise. If the conditions are particularly harsh (for example >33 °C and 50% relative humidity), it is likely that the individual will select an exercise intensity that will prevent high levels of personal (and thermal) discomfort. In other words, the individual will choose a lower exercise intensity (power output) than would be chosen in more moderate ambient conditions. This has been shown previously by the study of Tucker et al. (241), which showed that the power outputs during 20 km cycling time trials were approximately 15% lower in the heat (35 °C, 60% RH) compared to the cold (15 °C, 60% RH), resulting in a performance difference of 2.7%. Others have described the importance of this “anticipatory” control (132).

Although other behavioral responses that contribute to thermoregulation include removing clothing and seeking shade in hot conditions and adding clothing and seeking heat sources in cold conditions, it is the conscious selection of an exercise intensity that has the most profound effect on an individual's thermal status since the metabolic rate sets the rate of heat production (168). Therefore the freely chosen exercise intensity (or lowering thereof) is the most important determinant of whether or not a person will exceed his or her heat-losing capabilities during exercise.

Behavior alone does not ensure the maintenance of the core temperature at a desirable level, however, and physiological responses are triggered to cool the body. Heat loss and gain in the body follows the first law of thermodynamics:

$$M - W = E \pm R \pm C$$

$$\text{(Gains)} = \text{(Losses)}$$

where M is the metabolic rate, W is the physical work done, E is heat lost via evaporation, R is heat lost or gained via radiation, and C is heat lost or gained via convection and conduction.

The most effective mechanism by which humans dissipate heat is evaporation. Humans have a large capacity to produce sweat, although the amount of sweat that is evaporated is highly dependent on the available and exposed surface area of the skin, and also on the water vapor pressure gradient between the skin and the ambient air (114; 164). Together with convection, these two mechanisms become the primary route of heat loss in humans. Radiation also plays a role, but when compared to the heat losses associated with convection and evaporation, the contribution of radiation is small.

Foundational thermoregulatory research

The early studies – Forgotten findings

Early investigators made simple yet profound observations about the temperature response to exercise (12; 38; 163; 165; 166; 168; 198; 201; 255), although these data appear to have been largely overlooked by modern researchers. This sub-section will highlight the main findings of these investigators that are especially relevant to the modern understanding of thermoregulation during exercise.

In 1938 Nielsen (168) was the first to show that the rectal temperature is a direct function of the exercise intensity, and therefore the metabolic rate, with a series of experiments conducted on three men in both warm (36 °C) and cold (5 °C) air temperatures with 35-55% humidity. The importance of Nielsen's early paper is that it established that the rectal temperature is regulated at a higher level during exercise. Perhaps the most remarkable figure in

this classic article is the one showing the rectal temperature during 250 minutes of exercise at 175 W. This subject reached a steady state in his temperature after approximately 50 minutes, after which point the rectal temperature remained at ~ 38.1 °C for the remaining three hours of exercise. Upon stopping exercise, the rectal temperature in this subject then decreased rapidly by approximately 0.05 °C per minute, reaching pre-exercise values within 40 minutes. In addition, Nielsen showed the rectal temperature response was the same even during exercise at different ambient temperatures. This finding held true as long as the subjects remained in thermal equilibrium with the environment. In most individuals this occurred as long as the ambient temperature was ≤ 30 °C, although this will vary depending on an individual's state of training and acclimatization (168).

Nielsen's work in 1938 (168) followed prior work published by Winslow et al. (255) in 1937. Those investigators published a comprehensive series of observations about the thermal balance in various environmental conditions in humans at rest. Their main findings, as summarized by the authors, were that in ambient temperatures between 16-24 °C their subjects achieved thermal balance so that their heat rates of heat loss matched their rates of heat production. They further noted that above these temperatures the sweating rate increases in a linear fashion so that even in very high ambient temperatures (>30 °C) "the body maintains a high degree of thermal equilibrium" (255). However perhaps the most interesting observation made by Winslow et al. was that the minimal evaporative heat loss in some of their experiments "is profoundly influenced by air movement," a concept which was later recognized and proven by other authors (33; 45; 214; 243).

Over 20 years later other investigators (12; 38) demonstrated that the increase in rectal temperature during exercise is proportional to the relative exercise intensity and not necessarily the absolute exercise intensity. For example, Astrand (12) studied subjects with different levels of maximal oxygen uptake ($\text{VO}_{2\text{max}}$) and found that after seven hours of cycle ergometry at a workload corresponding to 50% of each subject's peak power output (PPO), the mean rectal temperature in all four subjects was 38.1 °C. This despite the fact that the oxygen uptakes of the four subjects during this trial ranged from 1.1-2.7 $\text{L}\cdot\text{min}^{-1}$. These data are supported by Clasing and Laumann (38), who studied 20 trained and 20 untrained subjects at a fixed exercise intensity of 130 $\text{beats}\cdot\text{min}^{-1}$. Even though this HR represented a wide range of absolute work rates and metabolic rates, all the subjects reached almost exactly the same rectal temperature (38).

Nielsen and Nielsen (165) in 1962 demonstrated in a more profound manner what Nielsen showed in 1938 (168)—that the T_{re} response is independent of the ambient temperature in conditions of 5, 20, and 30 °C (although at 30 °C the relative humidity was low (25%)). One limitation of this study was that the exercise bouts were only 60 minutes in duration, and so the nature of the T_{re} response to much longer duration exercise in different conditions was not studied.

In 1963 Robinson (198) reviewed the findings and conclusions of Nielsen (168). He emphasized that the temperature response to exercise is a function of the exercise intensity and therefore the metabolic rate, and that at given sub-maximal workloads humans reach a temperature steady-state within 60 minutes and remain at that temperature for the duration of the exercise bout. The novel aspect of Robinson's 1963 paper, however, was the rectal temperature data of two highly trained and competitive runners after completing several time tri-

als of three miles, five km, and 10 km in both hot and cool ambient conditions. The results of those measurements are reproduced in Figure 1.

The data from Robinson (198) show that the body apparently regulates all potential variables so that the highest core temperatures are reached only at the very end of exercise. For example, when one runner completed a three-mile time trial in 14 minutes “when the air temperature was 30.6 °C and solar radiation was high” his post-run rectal temperature was 41.1 °C. However, in similar conditions of 30.0 °C, another runner ran 10 km in 31 minutes and finished with a post-run rectal temperature of 41.1 °C. Furthermore, the author stated that these two runners “made normal recoveries from the great elevations of body temperature,” indicating that they showed no adverse signs or symptoms of “heat injury.”

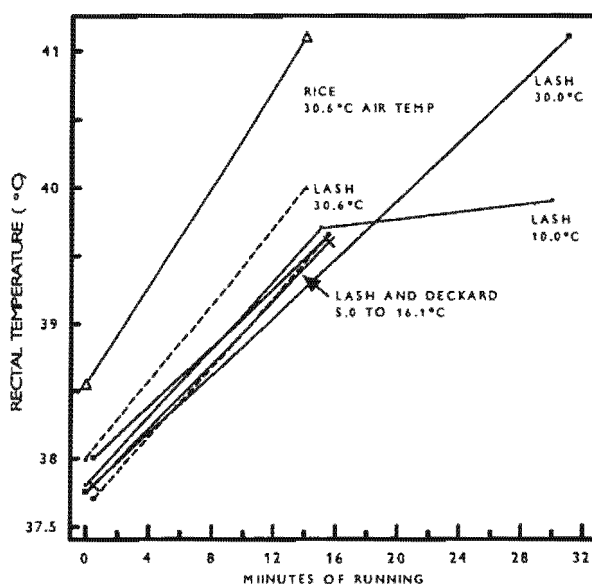


Figure 1. T_{re} responses of two world class runners immediately before and after running 3 mi (14 min.), 5 km (15 min.), and 10 km (31 min.). T_{re} was elevated by warm-up before the races. The air temperature during each race is printed under each runner's name. As noted by the original authors, the "humidity and solar radiation were high with temperatures of 30 °C and 30.6 °C." Redrawn from Robinson (1963).

Saltin and Hermansen (201) showed again that rectal temperature is regulated at a higher level during exercise. However, the primary contribution of their study and of Clasing and Laumann's work (38) was that the temperature response appears to be related more to the relative intensity of the subjects than the absolute intensity. For example, although the energy

expenditure in Saltin and Hermansen's subjects during one experiment varied between 6.3-13.5 kcal·min⁻¹, the mean rectal temperature in all subjects was 38.15 ± 0.09 °C. This workload corresponded to approximately 50% of each subject's VO₂max.

Finally, in 1969 Nielsen (163) published a thesis on thermoregulation at rest and during exercise. It is an extensive body of work and represents the prevailing concepts on thermoregulation from that era as discussed in the above studies. Nielsen discusses the common findings up to 1969 and discusses critically the limitations and concepts that could not be explained by the available evidence.

Taken together, the findings of these early researchers (12; 38; 163; 165; 165; 166; 168; 198; 201; 255) represent novel and important observations about the rectal temperature responses to exercise. Specifically their results all show that the rectal temperature is a direct function of the metabolic rate, and that it is regulated at a higher level during exercise. Yet these findings appear to have been lost and are not often acknowledged by many modern scientists. Instead, a number of studies published in the 1960's (41; 72; 90; 256) appear to have contributed to an intellectual paradigm shift so that the concept that metabolic rate is the strongest predictor of rectal temperature has been largely disregarded. In contrast, the current model identifies body fluid status, or more specifically the level of dehydration, as the most important determinant of rectal temperature during exercise.

Early fluid ingestion trials

In the 1940's the United States military tasked E.F. Adolph study the effects of various harsh environments on its soldiers. The result of this was a published volume of unique observations made by Adolph and his associates (4) in which the fluid consumption habits, tempera-

ture, and sweating responses of soldiers in an array of different conditions are presented and discussed.

One of Adolph's more relevant experiments consisted of two groups of men hiking in the desert for between three to eight hours. One group (N = 70) were prohibited from ingesting any water. The other group (N = 59) were permitted to refill their canteens after each 50 min work cycle, and therefore had *ad libitum* access to water during the hike. Adolph reported that only one subject in the water ingestion group reached exhaustion before the end of the hike while 11 in the fluid restriction group fatigued prematurely.

Adolph's conclusions from this experiment were that a shortage of water will ultimately lead to exhaustion, although not primarily from thirst which was the only the second-most frequently self-reported symptom of the men walking without water.

What Adolph's experiment above (and others) demonstrates is that fluid restriction will indeed impact performance. Furthermore, Adolph demonstrated not that replacing 100% of fluid losses is necessary and beneficial to performance, but instead that *ad libitum* access to water is sufficient to enhance performance. He also demonstrated that fluid ingestion does have an effect on rectal temperature since the subjects in his experiments had lower rectal temperatures when ingesting water compared to those who were fluid restricted.

An early researcher in this field who followed Adolph was W.S.S. Ladell, who completed extensive research on heat exposure and acclimatization in western Africa, with an emphasis on water and sodium intake. In 1955 Ladell published an intricate and detailed series of experiments in trained men who ingested water or varying concentrations of saline during intermit-

tent exercise in the heat. Ladell's primary conclusions were that T_{re} appeared to reach a steady state when fluid was ingested compared to when no fluid was ingested (Figure 2). He also proposed that humans have a "free circulating water" volume of approximately two Liters. He suggested, based on his data and others (116), that urinary and salivary output are not affected until a water loss of two Liters is reached, and that therefore up until this level of fluid loss, the function of the body will be unimpaired.

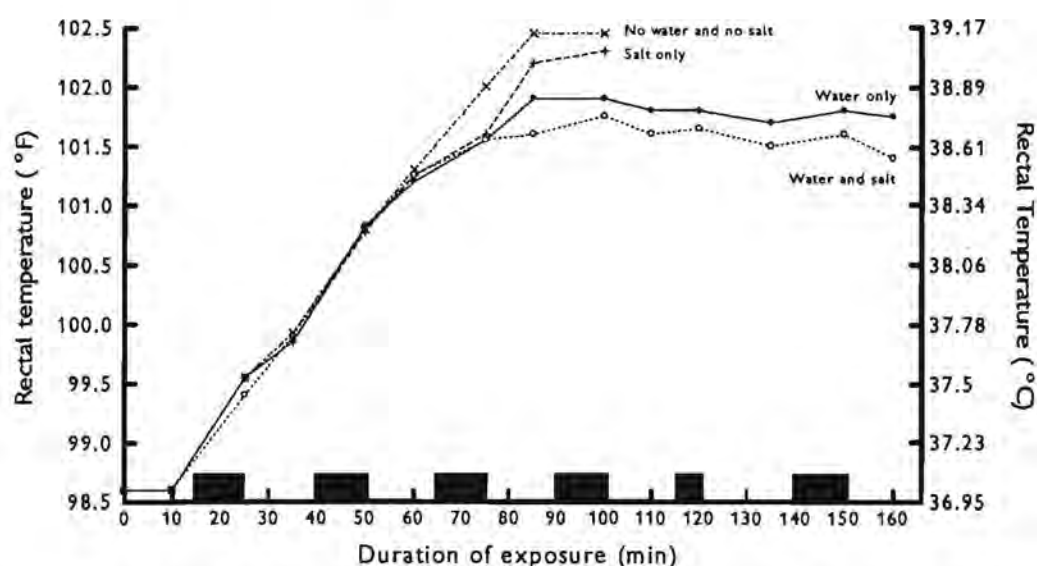


Figure 2. The rectal temperature responses in two subjects from Ladell (1955). The ambient conditions were 37.8 °C (dry bulb) and 30.5 m.min⁻¹ wind speed. During the 25 min rest/work cycles the mean work rate was approximately 0.8 L O₂.min⁻¹, and the ingested fluid volumes were equal to weight losses. Ladell was the first to demonstrate the effect of fluid ingestion on temperature regulation in hot environments. Note that even in such a high ambient temperature and when ingesting no fluid, peak T_{re} 's are well below 40 °C when the subjects fatigued.

Interestingly, although Ladell was one of the first to show that fluid ingestion has some effect on temperature regulation (Figure 2), he reached a similar conclusion to that of Nielsen (168). Ladell stated that "thermal equilibrium was established at a higher level" when subjects did not ingest fluid compared to when they did (119), which echoes Nielsen's conclusions from 1938 (168). Ladell's other contributions to the literature include an extensive review article in which he described seven kinds of "heat disorders" (120), and other observations about the

relationship between T_{re} and heart rate (121; 122) although these articles are not relevant here.

Thus the early researchers in this area contributed two important concepts. First, the earliest scientists found that T_{re} is a direct function of the (relative) metabolic rate (168; 201). Second, their successors showed that *ad libitum* fluid ingestion is sufficient to enhance performance when exercising in hot (>35 °C) environments such as the desert (4).

Fluid ingestion during exercise and its effects on thermoregulation

The 1960's represents an era in which there was an intellectual shift from a paradigm that emphasized metabolic rate as the determinant of the rectal temperature during exercise to one that emphasized the crucial role of fluid balance in thermoregulation during exercise. In that decade different investigators published studies that represented both the "old" and "new" paradigms. For example authors such as Nielsen, Saltin, and Robinson (165; 198; 201) published articles in which they discussed the concepts that 1) T_{re} is regulated at a higher level during exercise, and 2) metabolic rate is the primary predictor of T_{re} . In the same decade, however, other researchers (90; 219; 256) began to emphasize the role of fluid balance, and, in particular, the level of dehydration on temperature regulation.

Two primary articles that appeared in 1969 (256) and 1970 (41) signal the apparent start of the shift in paradigms from one in which metabolic rate is accepted as the key regulator to one in which the role of dehydration is advanced as the primary determinant of the rise in rectal temperature during endurance exercise. This paradigm later evolved to incorporate the concept that dehydration is the ultimate determinant of the peak core temperature during exercise. Although the data of Buskirk and Beetham (27) demonstrated an apparent causal

relationship between dehydration and rectal temperature nine years earlier, their data was overlooked by their peers.

The 1969 article by Wyndham and Strydom (256) was titled “The danger of an inadequate water intake during marathon running.” In this study the researchers measured the post-race rectal temperatures and weight losses of runners from two different 32 km races in temperate conditions. In runners who lost more than 3% of their pre-race mass, they found a significant relationship ($r^2 = 0.45$) between the post-race T_{re} and the amount of weight lost during the race. Their conclusion was that dehydration predicted the post-race rectal temperature so that the more dehydrated a runner was, then the higher the post-race rectal temperature. It is now known, however, that they misinterpreted their data since they could not draw this conclusion on the basis of a cross-sectional study (172).

What Wyndham and Strydom (256) should have concluded was that fast and dehydrated runners appear to have a remarkable heat tolerance (172), and that although they completed the events at speeds of $4.8 \text{ m}\cdot\text{s}^{-1}$ and slower and in relatively cool conditions (ambient temperature not exceeding $17 \text{ }^\circ\text{C}$), their post-race rectal temperatures were between $38.3\text{-}40.8 \text{ }^\circ\text{C}$. Although these temperatures represent “hyperthermia” they are nevertheless far below the critically dangerous temperature of $42 \text{ }^\circ\text{C}$ at which a differential diagnosis for clinical heat stroke is made. It should be noted that the subject who achieved rectal temperatures of 40.6 and $40.8 \text{ }^\circ\text{C}$ was the race winner, who almost certainly was exercising at the highest absolute and relative metabolic rate as is evidenced by his victory. In addition, the authors did not report any signs and symptoms of “heat illness” in any of the subjects in their study (256), although the title of the study suggests a deleterious effect of dehydration on health.

The second study that supported this new theory was published by Costill et al. in 1970 (41). In their study four highly trained ($73.9 \pm 2.8 \text{ mL}\cdot\text{kg}\cdot\text{min}^{-1}$) professional male runners ran for two hours at a high intensity (70% VO_2max) fixed workload on three occasions. Each run differed only in the type of fluid that the runners ingested, which was either water, a sports drink, or no fluid. The runs were completed at 24-25 °C and 50-55% RH. This classic study was the first to show that when the workload is fixed during high-intensity and continuous exercise—unlike earlier researchers who used intermittent work/rest cycles—the consumption of fluid does help to maintain T_{re} at a lower level compared to when an athlete ingests no fluid.

However a major limitation to this study is that the facing wind speed was a mere $5.7 \text{ km}\cdot\text{h}^{-1}$ although the workload was 70% VO_2max which approximates marathon race pace. Considering that the four subjects were highly trained runners, it is likely that a running speed corresponding to 70% VO_2max is $>15 \text{ km}\cdot\text{h}^{-1}$, which is far in excess of the facing wind speed in this study. The authors did not report any signs of "heat illness" in their subjects, and all of the runners completed each of the two-hour runs regardless of which fluid they ingested and regardless of what their final T_{re} was. The difference in T_{re} at the end of the fluid ingestion trials and no fluid trial was only $\sim 0.6 \text{ }^\circ\text{C}$. In fact, one subject in this study had won the Boston Marathon in 1968 without ingesting any fluid, and the authors note that another subject who achieved a T_{re} of $40.6 \text{ }^\circ\text{C}$ during the no fluid trial rated that run as his easiest (41).

A third article appeared also in 1970 (54), although it is less robust compared to Costill et al. (41) since the exercise duration was only one hour. Ekblom et al. (54) examined the T_{re} response to exercise in neutral ambient conditions ($21.7 \pm 0.7 \text{ }^\circ\text{C}$, 38% RH) in three male subjects ($\text{VO}_2\text{max} = 61.4 \text{ mL}\cdot\text{kg}\cdot\text{min}^{-1}$). The investigators examined the temperature response to

both intermittent and continuous work and controlled for the amount of heat production during the two trials. There was also a fluid intervention so that the subjects replaced 0% or 100% of their weight losses. After one hour the difference in T_{re} between the fluid interventions was 0.3 °C in the continuous trial and 0.4 °C in the intermittent trial, perhaps demonstrating an effect of fluid ingestion on the ability to dissipate heat, but at the same time demonstrating that this effect is nevertheless small.

These two early studies by Wyndham and Strydom in 1969 (256) and Costill et al. in 1970 (41) sparked further investigation into the effects of fluid ingestion on temperature regulation. In 1974 Gisolfi and Copping (72) investigated treadmill running in the heat. Six men completed a series of experimental trials in a hot environment (33.5 °C, ~50-60% RH) that investigated the effects of ingesting warm or cold water, and applying external cooling in the form of cold towels. The results preceded those of others (147) in that there was a graded effect of fluid ingestion on the post-run T_{re} . In the conditions in which the subjects ingested the most fluid (~2000 mL), the T_{re} was however only ~0.6 °C lower compared to when they ingested no fluid.

In summary, four early studies (41; 54; 72; 256) and one more recent one (147) appear to form the foundations of a model that hydration status, or more specifically the level of dehydration reached during exercise, affects thermoregulation negatively and therefore places an individual at a greatly increased risk for “heat illness” and heat stroke. While evidence suggests that dehydration can indeed affect thermoregulation (41; 147), its effects are most profound only when the workload is fixed (and the exercise therefore is *not* self-paced) and in environmental conditions that exceed 30 °C. Even in particularly hot environments, this effect is small after two hours (~0.6 °C). The available evidence in no way suggests that dehy-

dration is a dangerous situation, particularly since none of the authors report any signs of “heat illness” in their subjects, and also because the mean peak rectal temperatures achieved in all of these studies were far below 42 °C and generally below 40 °C, except in individual subjects in some of these trials.

Fluid ingestion before exercise—hyperhydration

Although some early researchers (87) attempted to investigate the effects of ingesting a large bolus of fluid *before* exercise (hyperhydration), this line of research proliferated mostly in the 1990's. The primary hypothesis for this concept, as presented by Kay and Marino (108), is that the increased fluid ingestion enhances the heat storage capacity of the body, therefore permitting the individual to perform more work in a given situation before reaching a limiting core temperature.

Greenleaf et al. (87) were the first to use this methodology, which required subjects to ingest a volume of fluid immediately prior to the start of exercise. The primary aim of this study was to investigate if any changes in body temperature were related to changes in sweating. The three experimental trials were completed by eight men in moderate ambient conditions (23 °C, 50% RH) at 49% VO_2max . The experimental intervention consisted of three pre-exercise fluid conditions during the hour prior to the exercise bout. In the hyperhydration trial, the subjects ingested 40 mL·kg⁻¹ of water before exercise. In the *ad libitum* trial, they were permitted *ad libitum* access to tap water, while in the hypohydration trial they did not ingest fluids. During exercise in the *ad libitum* and hypohydration trials, the subjects ingested 0.9% saline to match weight losses during exercise, while during the hyperhydration trial they ingested tap water.

The effects of the hypohydration and hyperhydration were that the peak T_{re} at the end of 70 minutes of exercise were somewhat higher in the hypohydration trial (38.51 °C) compared to the hyperhydration trial (37.65 °C). Thus Greenleaf et al. were one of the first to demonstrate the effects of hypohydration and hyperhydration during this mode and intensity of exercise, and concluded that the elevated T_{re} in the hypohydration trial were a direct effect of a decrease in sweating rate.

In 1987 Grucza et al. (92) tested subjects in a similar manner as Greenleaf in 1971 (87). The 60 min experimental trials were performed in neutral conditions (23 °C, 50% RH) and at 52% VO_{2max} . As is the case with most studies, the wind speeds were negligible ($<0.2 \text{ m}\cdot\text{s}^{-1}$). The main findings were that in the hyperhydrated trial the subjects began sweating earlier, the consequence of which was a lower T_{re} at the end of the exercise bout (37.7 vs. 38.1 °C).

However, in both trials the subjects appeared to have reached a temperature steady state after 50 minutes, with the only difference being that in the hyperhydration trial the T_{re} was ~ 0.4 °C lower. The importance of this is that in euhydrated trial the T_{re} did not continue to rise, while the limitation of this study is that the exercise bout was of relatively short duration.

The earlier studies in this area typically used water as a means to achieve the hyperhydration. However, a rapid diuresis usually follows the ingestion of large volumes of tap water, and a newer methodology using glycerol as a vehicle to promote fluid retention was developed. The findings in the studies that use this glycerol protocol are mixed with some showing an effect of hyperhydration on T_{re} and others showing no effect.

In 1990 Lyons et al. (130) tested a glycerol protocol to enhance fluid retention during 2.5 h prior to exercise. The experimental protocol consisted of three 4.5 h trials. Each 4.5 h trial included a 2.5 h fluid intervention followed by a 1.5 h exercise bout (running) at 60% $\dot{V}O_{2\max}$ in dry heat (42 °C, 25% RH). The three experimental conditions were “*ad libitum*” (5.4 mL·kg⁻¹), hyperhydration with glycerol (21.4 mL·kg⁻¹), and hyperhydration without glycerol (21.4 mL·kg⁻¹). The glycerol ingestion in this study resulted in less urine production before exercise and lower T_{re} 's and elevated sweat rates during the 1.5 h exercise bout.

The study of Latzka et al. found no effect of glycerol hyperhydration on thermoregulation (125). The subjects increased their body water by 1.5 L via glycerol or water ingestion before exercising in hot conditions (35 °C, 45% RH) for 120 min, but mean sweat rates and T_{re} 's were not different between the experimental trials.

Other investigators have examined the effects of hyperhydration on exercise performance with the hypothesis that the hyperhydration will attenuate a rise in T_{re} and therefore enhance performance. In his review in 1999 Wagner (245) summarizes a number of studies to show that the effects of glycerol hyperhydration on performance are equivocal.

Two studies that examined hyperhydration and its effects on exercise performance in the heat are those of Hitchins et al. (102) and Marino et al (133). Hitchins et al. (102) reported an effect of hyperhydration on performance so that subjects maintained a higher power output in the glycerol trial even though the T_{re} response between trials was not different. However Marino et al. (133) failed to find a difference in performance (total distance cycled in one hour) or T_{re} between their two experimental trials.

These two studies are typical of this area of the literature in that they reported contrasting findings. Wagner (245) argues that this is likely due to variation in experimental protocols and performance tests, and could also be due to differences in the subject population of each study. Finally Sawka (209) concluded there are no clear benefits of glycerol hyperhydration on performance. A common finding is that sweating starts earlier when hyperhydrated, and this is likely the physiological response to a decreased osmolality resulting from an acutely increased total body water.

Fluid restriction before exercise—hypohydration

Effects on thermoregulation during subsequent exercise

Although there remains much debate about the “dangers” and effects of dehydration during prolonged exercise, the evidence that hypohydration effects a range of parameters is robust and plentiful. Hypohydration refers to a state of dehydration incurred at a previous time, such as during a prior exercise bout or during a period of fluid restriction. The data show that when beginning an exercise bout in a hypohydrated state, the sweating response is delayed, therefore producing a steeper rise in rectal temperature. The mechanism of this is likely that of an increased osmolality, which the body will defend more vigorously than the core temperature (233). Hence the sweat rate is decreased as the body defends the osmolality by lowering fluid losses and thus attenuating any rise in osmolality, indicating that the defense of osmolality takes precedent over the regulation of body temperature during exercise.

Although there are numerous studies available that have examined hypohydration, the relevance of it to this thesis is limited. Therefore only a selection of studies that are characteristic of this area of research have been included. It should be noted that studies of hypohydration are more relevant to activities such as wrestling and boxing, in which athletes are likely to be-

gin exercise in a state of fluid deficit. This is due to the nature of these sports in that athletes compete in weight classes. On the other hand, athletes competing in endurance events are likely advised to ensure that they do not start their events in a state of fluid deficit.

Greenleaf et al. (89) were the first to investigate the effects of hypohydration in 1967 when they examined two groups of trained women, although the primary aim of this study appears to have been to assess the effects of hypohydration on various simple physical tasks such as sit ups and push ups, and on isometric strength. The main findings were that hypohydration affected the circulatory system as measured by recovery HR, and that maximal isometric strength was not affected by hypohydration.

In 1971 Greenleaf and Castle (87) were the first to study the effects of hypohydration on thermoregulation during a subsequent exercise bout. They dehydrated subjects by having them perform low-intensity and intermittent work in a hot environment the evening prior to the experimental protocol. The subjects then slept in the laboratory and remained in a fluid restricted state before completing a 70 min experimental trial in neutral environmental conditions (~23-24 °C, 50% RH) the next morning on waking. During the one hour prior to the start of each trial, the subjects drank either nothing, *ad libitum*, or 2.5-3 L of water, while during the experimental trials the subjects consumed water or saline in all conditions to match body weight losses.

The data from this study (87) showed that, when hypohydrated, the subjects peak T_{re} was 0.61 °C and 0.87 °C higher compared to the other two conditions in which they ingested fluid before the exercise bout. The authors also reported a reduction in sweat rate in the hypohy-

dration trial, probably again illustrating that the body defends osmolality first and then fluid balance, although plasma osmolality was not measured in this study.

Claremont et al. in 1976 (37) were the first to use diuretics to produce a reduction in plasma volume prior to an exercise bout to examine the effects of dehydration in the absence of thermal stress. Plasma volume was reduced by 15.3% with oral diuretic administration prior to the exercise bout. This study reported similar sweat rates between the dehydrated and euhydrated trials, yet significantly higher T_{re} 's at the end of the exercise bout. Therefore the authors concluded that the reduction in plasma volume resulted in a decreased ability to conduct heat from the skin to the core.

Armstrong et al. (7) followed with a study of diuretic-induced decreases in plasma volume (~9.8%) and its effects on performance and VO_{2max} . The first finding of this study was that the diuretic-induced plasma volume reduction had no effect on VO_{2max} . However, the authors reported a reduction in running speed of 3.1%, 6.7%, and 6.3% over distances of 1500 m, 5000 m, and 10,000 m, respectively, when comparing the dehydrated time trials to the euhydrated time trials. They concluded that the performance decrements are "most logically explained by . . . impaired thermoregulation. . ." although it is not clear how they reached this conclusion since they did not measure any thermoregulatory variables. The mechanisms by which the reduced plasma volume created the performance decrement were not elucidated since only running speeds were reported.

In 1983 Sawka et al. (210) used a similar research model to examine hypohydration, although the pre-exercise dehydration was achieved via an exercise bout. These scientists examined subjects in extremely hot environmental conditions of 49° C and 20% RH who had been pre-

viously dehydrated in a graded fashion by exercise of a moderate intensity in the heat. The subjects then performed heat stress tests, which lasted 140 minutes, and which consisted of walking at 1.34 m/sec for 25 min, followed by 10 min rest, repeated four times. Subjects reclined in the chamber during the periods of rest. The subjects started these tests in either a euhydrated condition ($\leq 1\%$ of baseline body weight) or a 5% dehydrated condition.

Sawka concluded that the thermal strain on an individual increases with increasing severity of hypohydration, since the ability to thermoregulate appeared to be compromised. However, core temperatures in the 0 and 3% hydration states were not significantly different during the first exercise test. In the following two tests, the core temperature difference was not more than 0.3 °C between the 0 and 3% hypohydration states. While sweating began at a higher core temperature with increasing levels of hypohydration, the rate of increase of sweating rate remained constant through the 0, 3 and 5% hypohydration trials, and the mean sweat rate was not different between the 0 and 3% trial states. In addition, sweat rates in the 3% and 5% hypohydration trials appeared to reach the same maximal values as the 0% trial. These results indicate that the thermoregulatory mechanisms of the body function normally at a 3-5% level of hypohydration, even when subjects are previously dehydrated. The increased core temperature at the end of the trial was a result of a higher starting temperature. Furthermore, the ability to begin sweating only at a higher temperature may in fact be an adaptation to an increased serum osmolality resulting from a greater degree of dehydration and the function of which is to prevent excessive further body water loss (which would cause a further increase in osmolality) without any detrimental effects on physical performance, rather than a negative side-effect of a dehydrated state.

Buono et al. (26) examined the interaction between hypohydration and the environmental conditions by having eight physically active male subjects complete one hour of cycle ergometry (60% VO_2max) in either hot (33 °C, 40% RH) or moderate (23 °C, 40% RH) ambient conditions. Hypohydration was achieved with a period of light exercise followed by fluid restriction on each day prior to the trial so that the subjects reported to the laboratory with a 5% reduction in weight.

The physiological responses to the two trials conducted in moderate ambient conditions did not differ from each other significantly in that T_{re} and forearm blood flow were similar. However in the hot environment, the hypohydrated subjects finished the 60 min of exercise with a higher T_{re} and a decreased forearm blood flow. Although there is likely some effect of hypohydration on these variables, it must be noted that one limitation to the study of Buono et al. is that there appeared to be no air movement in their environmental chamber as it was not reported in the methods. This lack of convection might amplify any effects of the hypohydration and therefore lead to a Type I error, especially since cycling at 60% VO_2max corresponds to speeds of at least 15-20 $\text{km}\cdot\text{h}^{-1}$ in moderately trained subjects.

Fluid ingestion and temperature regulation in field events

If the current model of thermoregulation which predicts dehydration as the primary determinant of the T_{re} is indeed correct, then the ingestion of large amounts of fluid to prevent dehydration would also keep core temperature low both in the laboratory and in the field. However, currently no evidence suggests that ingesting such large volumes of fluid during actual events in which athletes compete while employing their own self-selected pacing strategies is 1) beneficial to the athlete, 2) does anything to lower core temperature, and, perhaps most importantly in competitive athletes, 3) enhances performance. Rather, the ingestion of large

volumes of fluid has been shown instead to be a risk factor for developing exercise-associated hyponatremia (6; 98).

The literature indicates that athletes replace between 40-60% (or less) of their weight losses during competition (99; 124; 159; 162; 197; 200; 253) (a situation described as “voluntary dehydration”) and so lose between 2-5% of their pre-race weight. Yet even though fluid replacement and any effects of dehydration are emphasized in both the scientific and lay literature, there is a substantial dearth of real evidence showing that dehydration of this magnitude has serious health consequences. The literature suggests rather that time and again athletes at all levels of ability are able to complete various distances without succumbing to heat stroke or “heat injury” even though they lose up to 5-10% of their pre-race weight.

Peak rectal temperatures following different time trials and races seldom reach 40 °C, or peak at approximately 40-41 °C (2; 36; 131; 137; 138; 143; 159; 193; 198; 256) (Table 1) after which time they decrease rapidly. Most importantly, though, these peak T_{re} 's are achieved only at the end of the performance trial or race, most likely when the athlete increases the power output in a sprint for the finish, and are followed by an immediate drop in metabolic rate (and therefore T_{re}) when the athlete stops exercising. The cause of collapse after the end of a race has been shown to be caused by postural hypotension, which is a function of the peripheral vascular resistance and not a function of the T_{re} (103-105).

A number of studies in the literature have described the fluid replacement strategies and dehydration levels of both recreational (131; 158; 159; 162) and elite (2; 27; 136-138) athletes during various running performances.

The first known study to examine weight changes and T_{re} before and after distance running race was performed by Buskirk and Beetham in 1960 (27). This paper preceded others (193) by many years yet remains largely overlooked by modern scientists even though it was the first to report on body weight changes and T_{re} in marathon runners. The authors used the Boston Marathon (42.2 km), the Brighton Road Race (28.7 km), and a practice race on the Boston Marathon course (28-29km) to measure the pre and post-race T_{re} and changes in weight in a number of elite runners. The ambient conditions were mild to cold during each run (Boston: sunny, 20.4 °C, 37% RH; Brighton Road Race: sunny, 14.8 °C, 20% RH; Practice race: sunny, 4.3 °C, 53% RH).

This paper is perhaps the first to express percent weight losses as a predictor of T_{re} in runners. Although other investigators prior to Buskirk and Beetham examined this relationship during low intensity exercise (4), they did not plot the relationship between weight losses and peak T_{re} . Buskirk and Beetham reported a significant relationship ($r = 0.58$, $r^2 = .33$) between the percentage of weight lost during each race and the post-race T_{re} , much in the same way Wyndham and Strydom reported in 1969. Yet in their discussion the authors acknowledge the findings of Nielsen (168) that T_{re} is a function of metabolic rate, and they go on to state that a new level of thermoregulation is perhaps achieved. They also speculated that dehydration of 2-3% might actually benefit an athlete since it will decrease the time required to achieve this higher level of temperature regulation.

More importantly, however, is that when the data from the three runs are plotted separately the relationship between the change in mass and the post-race T_{re} is clearly spurious. The relationship between these two variables in each of the three runs can be seen in Figure 3, which reveals two important findings. First, the relationship between these two variables

does not exist in two of the three races. If this were a causal relationship so that the level of dehydration always predicts the extent of the rise in T_{re} , then this relationship should be present in all three data sets independently. Therefore this can only mean that the presence of this relationship in the Boston Marathon data is spurious. Second, even if this were a causal relationship, and even if it existed only in this race, according to the finding in the Boston Marathon data a runner would have lost 11.5% of his initial mass before reaching a critical T_{re} . This is a weight loss nearly 1.5 times greater than the runner who lost the most weight during the race. Furthermore, subjects in Adolph et al. (4) reached complete exhaustion at levels much lower than 11.5%, suggesting that in the unlikely event that an athlete achieves such a level of weight loss, fatigue will likely occur and the metabolic rate will thus be decreased (perhaps from collapse), resulting in the athlete never achieving a critical T_{re} .

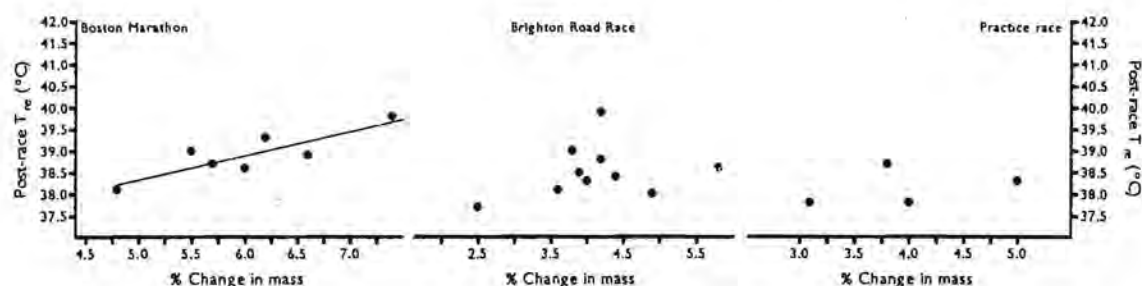


Figure 3. Data from Buskirk and Beetham that have been replotted as separate races. When plotted in this fashion the relationship between % change in mass and the post-race T_{re} becomes spurious.

It is unclear why this relationship exists only in the one data set of Buskirk and Beetham (27). One might argue that it is due to the environmental conditions since in the Boston Marathon the ambient temperature was $\sim 20^{\circ}\text{C}$, while in the other two races the ambient temperature was much lower (~ 15 and 5°C). Wyndham and Strydom (256) also reported a relationship between these two variables, yet the ambient conditions in their study were also cool ($< 20^{\circ}\text{C}$). While these findings perhaps suggest some effect of dehydration on the post-race T_{re} ,

the size of this effect must be sufficiently low or else the relationship would 1) be more profound and 2) be present in all data sets in the literature. However, this supposed relationship fulfills neither of these two criteria.

One of the earliest comprehensive field studies in runners was performed by Pugh et al. (193), who examined the pre- to post-race changes in weight and the post-race T_{re} 's in 56 finishers of a 42.2 km marathon raced moderate ambient conditions (22-23 °C, 52-58% RH). The first important finding of Pugh et al. was that three of the first four finishers had T_{re} 's above 40 °C. The race winner had the highest post-race T_{re} . He finished the race in 158 min with a T_{re} of 41.1 °C. Second, and perhaps more important, is that the authors' conclusion from their own data and others (198) that "tolerance of a high body temperature is a necessary condition of success in marathon running" (193). Although they recognized that the highest rate of failures occurred in runners with T_{re} 's between 39.5-40.0 °C, Pugh et al. reached no conclusions about dehydration affecting temperature regulation and instead associated dehydration as a cause of collapse unrelated to the T_{re} .

In 1974 Magazanik et al. (131) used a marathon race over "hilly terrain" in moderate ambient conditions (21-26 °C and 50-60% RH) to study various biochemical changes in six male runners (Age 24-33 years, mean $VO_{2max} = 61.5 \text{ mL}\cdot\text{kg}\cdot\text{min}^{-1}$). The investigators weighed the runners before and after the race, and recorded a post-race T_{re} . Fluids were available every 5 km starting at 10 km, and the runners had *ad libitum* access to these cold fluids. One novel aspect of this study was that the investigators measured the volume of ingested fluid to a reported accuracy of $\pm 5 \text{ g}$. Fluid intakes ranged from 570-2570 mL, which corresponds to a range of hourly intakes of $155\text{-}567 \text{ mL}\cdot\text{h}^{-1}$. The authors did not analyze the relationship be-

tween the weight losses and post-race T_{re} 's, which when plotted retrospectively yields no relationship between these two variables ($r^2 = 0.00$, $p = 0.92$; Figure 4a). There was also no relationship between the total volume of fluid ingested and the post-race T_{re} ($r^2 = 0.25$, $p = 0.31$; Figure 4b). Thus from an early stage it was shown that when running an actual race in moderate ambient conditions (21-26 °C, 50-60% RH), there appears to be no relationship between the level of dehydration, the extent of fluid ingestion, and the post-race T_{re} .

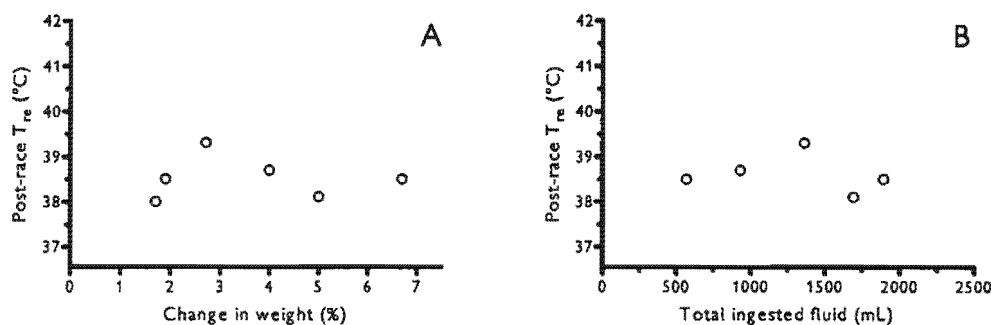


Figure 4. The data in this figure have been plotted from Magazanik et al. (1974). The authors did not plot these data, which demonstrate no relationship between the post-race rectal temperature and the percentage of weight lost during the race ($r^2 = 0.00$, $p = 0.92$) (4a), and the post-race rectal temperature and the total volume of ingested fluid ($r^2 = 0.25$, $p = 0.31$) (4b).

The mean race time in the study of Magazanik et al. (131) was 217 ± 39 minutes, with only one subject running faster than three hours (163 min) and four of the six finishing slower than 220 min. The runners completed the race with mean weight losses of $3.7 \pm 2.0\%$ and their post-race T_{re} values were remarkably similar (38.5 ± 0.5 °C) with only one runner completing the race with a T_{re} above 39 °C. There was a wide range of weight losses (1.7-6.7%) and volumes of fluid ingested (570-1890 mL). The authors made an interesting observation that even though runners had *ad libitum* access to fluids at numerous aid stations, they still completed the race with a mean weight loss of 3.7%. They noted that if dehydration is to be prevented, drinks must not only be made available but also forced on the runners.

Myhre et al. (159) examined four ($N = 2$, male, $N = 2$, female) middle-aged runners during a "warm-weather" marathon (15.5-24.5 °C WBGT). The authors noted wide individual differences for fluid ingestion and heat tolerance, citing that one female runner forcibly ingested 2050 mL ($591 \text{ mL}\cdot\text{h}^{-1}$) "in an effort to resist hypohydration," which resulted in nausea and vomiting and her subsequent withdraw from the race at 35 km, while another runner who ingested 650 mL ($180 \text{ mL}\cdot\text{h}^{-1}$) finished the race 6.6% dehydrated had no complaints. An interesting finding of this study was the continuously-recorded T_{re} in one runner. The data show that the T_{re} in this runner climbed for approximately 60 minutes and then reached a plateau at ~ 39.3 °C, where it remained for the duration of the race which he finished with weight losses of 3.9%. This finding is similar to that of Nielsen (168), who also reported the T_{re} response to four hours of continuous exercise in moderate ambient conditions (22-23 °C). Interestingly, the T_{re} response of the subject in Nielsen's study (168) is nearly identical to that reported in Myhre et al. (159) in that the T_{re} increased for approximately one hour before reaching a plateau and remaining there for the remainder of the exercise bout.

The data from this one runner alone show that, if there is any effect of dehydration on temperature regulation, it is not present when individuals select their own exercise intensity. Any effect of fluid balance on temperature regulation must therefore be small, or rather the effects of metabolic rate large, so that only minor alterations in the rate of heat production are sufficient to maintain the core temperature within an individually acceptable range.

Although the majority of subjects in these studies are men, in 1983 Christensen and Ruhling published the continuously measured T_{re} in one female runner during a cool weather (12-20 °C) marathon. They did not measure fluid ingestion and pre and post-race weights, but recorded her T_{re} at 10 min intervals during the race. Their data show that the runner main-

tained her T_{re} within a very narrow range (38.9-39.1 °C) for nearly the entire race except for a four km uphill section where it reached 40.0 °C, after which it decreased back to ~39 °C, illustrating that this athlete successfully regulated her temperature.

In keeping with their previous work, Myhre and colleagues (158) studied plasma volume (PV) changes in both men and women during a marathon race. They attempted to record the T_{re} continuously on one subject and intermittently in three others. They reported similar findings as Maron et al. (138) and Myhre et al. (159) with regards to the T_{re} responses.

Perhaps the more important contribution of this study is that the authors measured the fluid ingestion of these athletes and weighed them both before and after the race. The mean weight losses were $3.1 \pm 1.2\%$ in all of the subjects, and they ingested 1248 ± 422 mL during the entire race which equates to 326 ± 95 mL·h⁻¹. Not surprisingly, since the runners were ingesting fluid *ad libitum*, they replaced $41.3 \pm 17.7\%$ of their estimated sweat losses and finished the marathon without symptoms.

Nelson et al. (162) studied fluid and electrolyte balance in 39 men and six women in a cool weather marathon (7.8 °C, 100% RH (raining)). Post-race T_{re} 's were not measured, and one limitation to this study was that the fluid ingestion volumes were self-reported via a questionnaire administered two weeks after the race. Nevertheless, the mean reported fluid ingestion volume was 1650 ± 180 mL and the mean weight losses were 1.9 kg or 2.5%.

More recently, Millard-Stafford et al. investigated the effects of different fluids (CHO electrolyte beverage or a placebo) on five km running performance in the heat (25-32 °C, 60-80% RH) after a 35 km self-paced road run (143). In all trials the subjects consumed a bolus of 400

mL of their specified beverage prior to beginning their run. The subjects then ingested their designated fluid in 250 mL doses every five km during the 35 km run. The results of this trial were that the CHO drink enhanced the five km running performance by 2.5 min compared to the placebo. However, T_{re} , HR, and RPE were all similar during the two trials.

An interesting finding from Millard-Stafford et al. (143) is that even though the ambient conditions were relatively hot and the subjects finished the 40 km running trials with a mean weight loss of ~4.5%, the authors did not report any signs or symptoms of “heat illness” or “heat injury.” In addition to this the subjects all experienced a relatively rapid decline in T_{re} after the runs in spite of the fact that they remained in the hot environment, were ~4.5% dehydrated, and did not receive any cooling therapies. The mean T_{re} in the two groups decreased by $0.09\text{ }^{\circ}\text{C}\cdot\text{min}^{-1}$ during the 30 min recovery period during which the subjects ingested the CHO drink *ad libitum*.

Other investigators have examined similar variables over the marathon distance, but in elite runners (2; 136-138) as opposed to recreational runners in the previous studies.

In 1975 Adams et al. published what can only be described as a thorough and intricate series of experiments on one national class marathon runner over 65 days. The study consisted of a total of 13 treadmill runs of various distances in cool ($10\text{ }^{\circ}\text{C}$), moderate ($22\text{ }^{\circ}\text{C}$), and hot ($35.4\text{ }^{\circ}\text{C}$) conditions and one marathon race. It encompassed a heat acclimatization period (13 days) and included appropriate amounts of convective cooling with wind speed of $255\text{ m}\cdot\text{min}^{-1}$ for all of the treadmill runs.

Their main finding was a clear effect of heat acclimatization on thermoregulation during exercise. The subject ran for an additional 33 minutes during the post-acclimatization run in the heat and maintained a lower T_{re} . However, a more interesting finding is that during two pre-acclimatization runs, one of 60 minutes and the other a run to fatigue, the subject achieved an essentially identical T_{re} even though the run to fatigue was 42 minutes longer. After the 60 minute run, the duration of which was of known by the athlete before he began the exercise, his T_{re} was 40.1 °C. At the end of the run to fatigue, which was 102 minutes and an open loop test, his T_{re} was 40.2 °C.

The subject in Adams et al. had *ad libitum* access to fluid in all of the experimental runs, and the volume he ingested during each run was measured accurately, although they did not discuss these results in depth. The subject ingested remarkably repeatable hourly volumes in each of the environmental conditions and runs, with the acclimatization protocol appearing to have little or no effect on his thirst mechanism. For example, in the moderate pre- and post-acclimatization runs he ingested $\sim 500 \text{ mL}\cdot\text{h}^{-1}$ independent of the distance he ran. In the hot pre- and post-acclimatization runs he ingested a similar volume ($\sim 500 \text{ mL}\cdot\text{h}^{-1}$). Perhaps unremarkably, these volumes of ingested fluid led to similar percent changes in weight in each of the four runs mentioned above, which in turn suggests that the thirst mechanism is more robust than has been concluded more recently.

Maron et al. (136-138) studied highly trained and competitive marathoners in three consecutive Santa Barbara (California) Marathons (1973-75). Their work resulted in three unique papers reporting the blood biochemical, oxygen uptake, and thermoregulatory response to marathon running.

In the first study (136) Maron et al. studied six highly-trained and competitive male runners (136). The subjects in this study had well above average VO_2max values (mean $\text{VO}_2\text{max} = 67.3 \pm 1.7 \text{ mL}\cdot\text{kg}\cdot\text{min}^{-1}$), a mean race time of $164.34 \pm 4 \text{ min}$, and finished within the top 14 places of the race field. The ambient temperature on the race day was $\sim 20 \text{ }^\circ\text{C}$, although at three "hot spots" as identified by the authors at 16, 28, and 34 km the ambient temperature rose to 28.3, 28.9, and 25.2 $^\circ\text{C}$ respectively.

One main finding of Maron et al. (136) was that the post-race T_{re} values were higher than those reported previously by Magazanik (131) and Pugh (193). They concluded that this might be a result of the faster marathon times reported in their study. When these values are plotted against the pre- to post-race change in T_{re} , a significant negative relationship exists so that the fastest runners finished with the highest T_{re} and had the largest pre-race to post-race increase in T_{re} (Figure 4a), while no relationship was observed between the post-race T_{re} and the percentage of weight lost during the race (Figure 4b). The authors cite the findings of Nielsen (168) when interpreting the T_{re} data, recalling Nielsen's conclusion that during exercise T_{re} is regulated at a higher level than at rest, and that the degree of elevation is proportional to the workload (metabolic rate) (136).

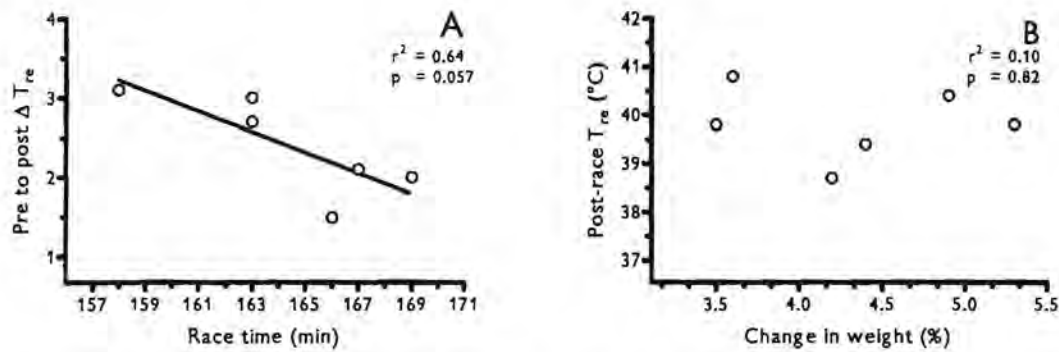


Figure 5. Data from Maron et al. (1975) showing the relationship between the pre-race to post-race change in T_{re} compared to each runner's performance time in the marathon race (5a). In this study there was no relationship between the post-race T_{re} and the pre-race to post-race percentage change in weight (5b).

The fluid ingestion volumes of the runners in this study were modest even though each runner had access at three mile intervals to personalized fluid bottles containing fluid(s) of his choice. At these points the runners were permitted to drink *ad libitum*, yet the mean total volume of ingested fluid was only 520 ± 210 mL, corresponding to a mean hourly volume of only 191 ± 80 mL.

At the following year's Santa Barbara Marathon (1974) Maron et al. followed up on their previous work by measuring the oxygen uptake responses in two of the six runners (best marathon times of 2:36:34 and 2:28:33) (137) from their previous study (136). The weather on race day was cool and calm (17.9 $^{\circ}\text{C}$ dry bulb, 15.7 $^{\circ}\text{C}$ wet bulb). Although the primary aim of this study was to measure the oxygen uptake of these two runners, fluid ingestion, pre and post-race weight, and pre and post-race T_{re} were also measured. Both runners were given *ad libitum* access to a CHO/electrolyte solution of their choice during the race.

The rates of fluid ingestion of the two runners varied widely as one runner ingested 1453 mL and the other runner only 379 mL. These fluid ingestion volumes consequently produced dif-

ferent amounts of weight loss (2.6 vs. 3.6%), indicating that their sweat rates were similar (1.2 vs. 1.0 L·h⁻¹). However even though each runner ingested different amounts of fluid and thus finished the marathon at different levels of dehydration, both runners exhibited a similar T_{re} response. The post-race T_{re} 's (39.9 and 39.9 °C) in both runners were 2.2 and 2.6 °C higher than the pre-race T_{re} 's (37.7 and 37.3 °C). This is likely because both runners completed the marathon at similar running speeds (271 ± 37 vs. 266 ± 16 m·min⁻¹) and therefore metabolic rates (3.63 ± 0.44 vs. 3.65 ± 0.21 L·min⁻¹) and hence their rates of heat production and heat storage were similar.

Finally, at the 1975 Santa Barbara Marathon Maron et al. completed their series of experiments (138) by measuring the same two runners as in the study above. The primary aim of this final study was to measure the thermoregulatory responses to marathon running. This was achieved by having the runners complete the race with a rectal probe inserted prior to the start, and then measuring the T_{re} and skin temperature from a car traveling along side the runners at various points along the route by coupling the probes with a telethermometer to obtain a reading. The main finding of this study was that one runner appeared to have a transient decrease in sweat rate after approximately two hours of running, resulting in a sudden increase in T_{re} from 40.9 °C to 41.9 °C. This increase in T_{re} could not, according to the authors, be a result of an increase in metabolic rate or increasing dehydration. More importantly, however, is that this runner maintained his running speed for the final 45 minutes of the race and apparently reached a higher level of thermal balance, as evidenced by a constant T_{re} yet an increasing running speed during the final 45 minutes of the race.

The general conclusion from all these studies that have measured fluid ingestion, changes in weight, and post-race T_{re} following marathon races must be that the faster runners have

higher rectal temperatures compared to the slower runners. Also, the mean post-race rectal temperatures in these studies are $\sim 39^{\circ}\text{C}$ (Table 1) except in a few individual runners who are able to tolerate higher rectal temperatures. Although these temperature values are representative of “hyperthermia,” they are far below any critical temperature that would necessitate immediate medical intervention and active cooling. Instead, the vast majority of runners completed these races with T_{re} 's $\sim 39^{\circ}\text{C}$ and without the need for medical treatment.

Although Wyndham and Strydom (256) reported a significant relationship between post-race rectal temperatures and the percentage of weight lost during 32 km running races, when the same variables in the study of Maron et al. (136) are plotted (Figure 5b), no relationship exists ($r^2 = 0.01$, $p = 0.84$). Yet a strong one is present between the race time and post-race T_{re} and the race time and pre to post the change in T_{re} (Figure 5a), and the same holds true for the data of Magazanik et al. (131) ($r^2 = 0.00$, $p = 0.91$) (Figure 4).

The findings of these field studies have been summarized in Table 1. The common result among them is that post-race T_{re} values are between $38\text{-}40^{\circ}\text{C}$ regardless of the ambient temperature and ability of the runners. Although many of these studies examined highly-trained competitive runners (<3 h marathon time), others studied recreational runners who completed the marathon in much slower times. Yet the findings are similar, suggesting that post-race T_{re} 's of $38\text{-}40^{\circ}\text{C}$ are the normal response to this mode and duration of exercise.

Most importantly, however, is that the studies that measured the T_{re} response continuously or intermittently during marathons (36; 138; 158; 159) have one aspect in common, which is that they all demonstrate that the T_{re} in both recreational (36; 158; 159) and competitive (138) runners rises for approximately one hour, reaches a plateau, and then remains within a

very narrow range for the vast majority of these races. Furthermore, the other studies in the literature that have measured only post-race T_{re} 's show that values of 39-40 °C are in fact the norm and that while some athletes who collapse during or after the race have similar T_{re} 's, the vast majority of the athletes finish the race with T_{re} 's that many scientists deem "hyperthermic," yet without any symptoms of "heat illness" and/or "heat injury." Therefore, the only conclusion that can be reached from these data is that in the vast majority of athletes a T_{re} of 39-40 °C is an entirely normal response to endurance exercise (even in neutral conditions (158)) and one from which these athletes recover very quickly (143).

Table 1. Selection of studies from the literature in which rectal temperature and ambient conditions were measured during a marathon race or shorter.

Authors (year)	Subjects	Env. Conditions (°C, % RH)	Distance	Time (min)	Final T_{re} (°C)
Buskirk and Beetham (1960)	10 highly trained runners	14.8 °C, 20%	28.7 km	126	38.5
Buskirk and Beetham (1960)	7 highly trained runners	20.4 °C, 37%	42.2 km	156	38.9
Robinson (1963)	2 professional male runners	30.0	10 km	31	41.1
		30.6	3 miles	15	40.0, 41.1
		5-16.1	5 km	15	39.7
Pugh et al. (1967)	63 finishers	23 °C, 58%	42.2 km	--	37-41
Pugh et al. (1967)	3 of top 4 finishers	23 °C, 58%	42.2 km	158	41.1
				164	40.5
				166	40.2
Wyndham and Strydom (1969)	1 trained runner (race winner)	16-18 °C, 80-95%	32 km	<120	40.6
			32 km	<120	40.8
Magazanik et al. (1974)	6 trained runners	21-26 °C, 50-60%	42.2 km	163-272	38-39.3
Adams et al. (1975)	1 highly trained runner	10 °C, 30%	42.2 km	165	39.6
		22 °C, 30%	42.2 km	165	39.8
		22 °C, 30%	42.2 km	165	39.4
		--	42.2 km	166	39.6
Maron et al. (1975)	6 highly trained runners	~20 °C	42.2 km	158-168	38.7-40.8
Maron et al. (1976)	2 highly trained runners	17.9 °C, 80%	42.2 km	156	39.9
				159	39.9
Maron et al. (1977)	2 highly trained runners	~20 °C, --	42.2 km	165	39.3
				163	41.7
Myhre et al. (1982)	1 trained runner	15.5-24.5 (WBGT)	42.2 km	204	39.7
Millard-Stafford et al. (1992)	8 trained runners	25-32 °C, 62-82%	40 km	--	~40

Dehydration and sub-maximal, fixed workload exercise

In studies that use fixed sub-maximal workloads as the exercise mode, various outcome variables such as core temperature (rectal or esophageal) and heart rate, stroke volume, skin temperature, forearm blood flow and cardiac output are measured. The cardiovascular variables are often measured together with other endocrine and various biochemical markers (81; 82). The common finding among these studies is that rectal temperature and heart rate increase to a larger degree during exercise when smaller fluid volumes are ingested, while skin blood flow has been shown to decrease under these conditions. This effect is enhanced in environments in which the thermal stress is more extreme (54; 72; 117; 119; 210).

A distinction must be made between experiments that investigate the effects of dehydration on subsequent bouts of exercise and those in which the effects of dehydration on the current bout of exercise are investigated. The importance of this has been discussed above, and the subsequent section will discuss only experiments which investigate the effects of dehydration on the current bout of exercise.

The first studies to investigate any effects of fluid ingestion during exercise were published in the 1940's (4; 190) and 1950's (119; 189). However the mode of exercise in these studies was either marching in the desert or in extremely hot environments, or intermittent work/rest cycles. In either protocol, the exercise intensity was relatively low. Therefore these early studies will not be discussed here, and instead only those that have used steady-state fixed workload protocols will be discussed.

In the early 1970's Costill et al. (41) were the first to use the now common methodology of two hours of exercise at a medium to high exercise intensity (60-70% VO_2max) while using

similar fluid ingestion interventions as the earlier studies by having subjects ingest 100% or 0% of their weight losses. Since then this protocol has been repeated many times.

Ekblom (54) found that thermoregulatory responses were impaired at a dehydration level of only 1% during both continuous and intermittent cycling exercise at 62% VO_2max in a temperate environment (22 °C and 38% RH; 7.2 $\text{km}\cdot\text{h}^{-1}$ wind speed). The conclusions drawn from this study are debatable, however, since only three subjects were studied. The large inter-individual variability in the measurement of sweat rate and body temperature makes the results from studies with a small subject number difficult to interpret. In all the responses measured except T_{re} , at least one out of the three subjects showed a movement in the measured variable opposite to that interpreted by the conclusion (i.e., sweat rate in a dehydrated state increased in one subject while decreasing in another), and there are no statistical analyses for the study due to the small sample number.

Gisolfi (72) also showed an elevated rectal temperature only after a 2% decrease in body mass when subjects ran for 1.5 to 2.5 hours at 75% VO_2max on a treadmill in the heat (33.5 °C dry bulb, 21.5 °C wet bulb, 34% RH; 2.16 $\text{km}\cdot\text{h}^{-1}$). However, this effect did not occur in all subjects, and the authors (72) emphasize the large inter-subject variability associated in this response. What can be seen from this trial, though, is that there was no difference in rectal temperature during the first hour of exercise. This finding occurred despite subjects ingesting no fluid vs. copious amounts of fluids equal to sweat rate during the first hour (and for the remainder of the exercise bout).

The conditions of the study can, however, lead to incorrect interpretation or a Type I error. Subjects ran at 75% VO_2max for 1.5 to 2.5 hours in the heat (33.5 °C, 34% RH) with wind

speed of only $2.1 \text{ km}\cdot\text{h}^{-1}$. This will reduce the convective heat loss to very low levels despite the low humidity. Therefore the small changes in body temperature may not be the result of thermoregulatory failure, but instead any small effect that dehydration has on thermoregulatory capacity might be amplified by the inability of the environment to absorb the heat, rather than by the primary failure of the body's thermoregulatory mechanisms.

Sawka (205) studied runners during two consecutive bouts of treadmill running at 70% VO_2max for 80 min, separated by a 90 min rest. The study took place in conditions of 22–25 °C and 45–60% RH. A wind was generated by a large fan behind the subject but the velocity is not reported in the methods. Although the runners began the second run dehydrated by 2%, rectal temperature still reached an equilibrium level, finishing 0.6 °C higher than after the first run. The rectal temperature followed exactly the same pattern as that of the first run, except that the starting level was higher. The rate of heat storage, a reflection of the function of the thermoregulatory systems, was the same. Therefore since the initial elevation in rectal temperature can be attributed to heat storage from the first run, it is questionable whether there was any impairment of the thermoregulatory function at this level of dehydration.

Between 1980-90, numerous investigators examined different aspects of fluid ingestion and temperature regulation (10; 19; 20; 29; 203).

Armstrong et al. in 1985 investigated the effects of water temperature on "voluntary dehydration" during prolonged exercise in the heat. During six hours of intermittent walking in 42 °C heat, subjects ingested water *ad libitum* at temperatures of 6, 22, or 46 °C. The water temperature had an effect on the ingested volume so that the subjects drank the most fluid in the 22 °C trial and the least fluid in the 46 °C trial. In addition, the 46 °C water ingestion had an

effect on the T_{re} . In that trial the peak T_{re} was 0.53 °C higher compared to the 6 °C trial, and the T_{skin} was 0.84 °C higher than the 6 °C trial. However sweat rates and cumulative sweat losses were not different between the three trials, and so the main contribution of this study was that the water temperature has an effect on *ad libitum* fluid ingestion in the heat.

Candas et al. studied the effects of five different fluid interventions on an array of temperature and cardiovascular variables during four hours of exercise in the heat (34 °C) (29). The exercise mode was a work/rest protocol consisting of 25 minutes of work and 5 min of rest for four hours. The interesting finding from this study was that even though there was minimal convective cooling ($0.6 \text{ m}\cdot\text{s}^{-1}$) and the subjects replaced only 80% of their fluid losses, the T_{re} reached a plateau after two hours of exercise in all conditions except the one in which the subjects did not ingest any fluid. However one limitation to this study is that the mode of exercise was work/rest cycles and not continuous and dynamic steady-state exercise.

In the study of Bassett et al. (20) the investigators used two different water spraying techniques to test the effects of wetting the skin on thermoregulation. Although there was no effect of the skin wetting, the interesting finding from this study is similar to that of Candas et al. (29), namely that even though the subjects were exercising in hot (29.5 °C and either 33% or 66% RH) conditions and ingesting only $236\text{-}279 \text{ mL}\cdot\text{h}^{-1}$ the T_{re} rose only $\sim 1\text{-}1.5$ °C above resting values. This volume of fluid ingestion represents the replacement of only $\sim 20\text{-}30\%$ of the sweat losses, yet the peak T_{re} values achieved by the subjects after running for two hours at 60% VO_2max were only ≤ 38.5 °C. One reason why no effect of such a small fluid ingestion volume was identified in this study could be that the wind speeds were close to the running speeds. For example, the wind speed is reported as $10.8 \text{ km}\cdot\text{h}^{-1}$ and the mean running

speed is reported as $12.8 \text{ km}\cdot\text{h}^{-1}$. Therefore no effect of fluid ingestion on temperature regulation was apparent in this study perhaps because the convective wind speeds were (more) appropriate.

Carter and Gisolfi (30) had subjects exercise at 60% VO_2max for three hours in the heat (31.5°C , 22% RH) while ingesting either water or a CHO-electrolyte drink *ad libitum*. The main finding that was not reported by the authors but is relevant here is that the subjects finished the three hours of exercise with weight losses of 1.92% and 2.17% in the water and CHO-electrolyte drink trials, respectively. However, the T_{re} 's in this trial followed the same pattern as described by the earliest investigators—it rose for approximately one hour and then reached a plateau where it remained for the duration of the exercise bout. Given the fact that the subjects were ingesting fluid *ad libitum*, again suggests that thirst may be an accurate indicator of one's fluid needs even when exercising in the heat.

Barr et al. used even more prolonged exercise (6 h) in the heat (30°C , 50% RH) to study the effects of either water, a 25 mmol Na^+ solution, or no fluid on PV and the regulation of Na^+ concentration (19). The first important finding from this study is that only one subject completed the no fluid trial, and the mean exercise time for all subjects in this trial was 4.48 ± 0.29 h. This demonstrates that fluid restriction has an effect on endurance performance as was first shown by Adolph (4) and Ladell (119). Second, they again showed that the effect of fluid ingestion on temperature regulation is small since in the no fluid trial the peak T_{re} was $\sim 39^\circ\text{C}$ while in the two fluid ingestion trials (in which the subjects ingested fluid to replace 100% of their weight losses), the peak T_{re} was $\sim 38.2^\circ\text{C}$, and interestingly was not achieved at the end of exercise. In fact at the end of two hours of exercise, which is the standard duration of many other trials, the differences in T_{re} were even smaller, approximately 0.2°C .

Then, in 1992 an article that subsequently became known as a “classic” study was published by Montain and Coyle (147). Its title, “Influence of Graded Dehydration on Hyperthermia and Cardiovascular Drift During Exercise,” suggests that the level of dehydration affects thermoregulation during exercise in a proportional manner. Eight endurance-trained male cyclists ($\text{VO}_{2\text{max}} = 4.72 \pm 0.33 \text{ L O}_2 \cdot \text{min}^{-1}$) completed four two-hour rides at 60% $\text{VO}_{2\text{max}}$ (mean power output 204 W) in 33 °C and 50% RH and 2.5 $\text{m} \cdot \text{s}^{-1}$ wind speed. The ingested fluid volume was altered to produce four different interventions in which the subjects replaced 0%, 20%, 48%, or 80% of their sweat losses. T_{re} , esophageal temperature, cardiac output and skin blood flow were the primary measurable outcomes. The main finding of this study was that of a linear relationship between the percentage of weight loss and the rise in T_{re} , so that as subjects lost more weight their T_{re} 's were higher at the end of each trial.

The research design and experimental interventions used in this study (147) did produce an effect of dehydration on core temperature during exercise, as had many before it (19; 41; 72). However, while the research methods are sound and the data robust, the findings are not applicable and remain limited due to the nature of the study design and the environmental conditions. Instead of findings that can be applied to self-paced exercise, the data remain applicable only to laboratory-based exercise.

Any difference in the temperature response to exercise becomes evident only once there is an apparently large enough difference in body water loss. This could therefore be interpreted as evidence that the thermoregulatory systems of the body function competently up to a certain level of dehydration, and thereafter deteriorate in a linear fashion with dehydration level. Ladell (119) first identified the idea of a threshold level of dehydration when he proposed that

there was a volume of “free circulating water” in the body that could be lost before there was any detriment to the bodily functions. Ladell (119) found that in non-sweating men subjected to water deprivation for 24 hours, saliva flow and urine production only decreased after the loss of two Liters of fluid through both normal urine production and through insensible sweat losses. In similar experiments of resting men, it has been shown that sweating may be the first water-losing physiological function to be affected by dehydration (210). Whether this hierarchy of events also applies during exercise remains to be seen, since both urine production and salivary flow are severely decreased during exercise and sweating increases in response to the increase in sympathetic stimulation. Similar to Montain and Coyle (147), Ladell (119) also found no difference in rectal temperatures of men working intermittently in the heat during the first hour, despite different volumes of fluid ingestion (Figure 2), which may also provide evidence indicating that thermoregulation is not significantly affected while men are still in this “free circulating water” zone.

It could also be interpreted that, in light of the above thermodynamic principles, small non-significant differences have been amplified by the environmental conditions in these studies and in which the potential for convective cooling was limited. To date no evidence of this effect of fluid ingestion on temperature regulation exists when the appropriate amount of convective cooling is present, although Chapter 5 of this thesis contributes new data to this area of the literature.

Furthermore, there is often no significant effect of the experimental conditions and rates of fluid ingestion in these studies with regards to T_{re} , HR, change in PV or RPE during the first 60 min of exercise. The differences in T_{re} in Montain and Coyle (147), for example, between a large fluid intake (80% of sweat losses replaced) and a moderate fluid intake (48% replaced)

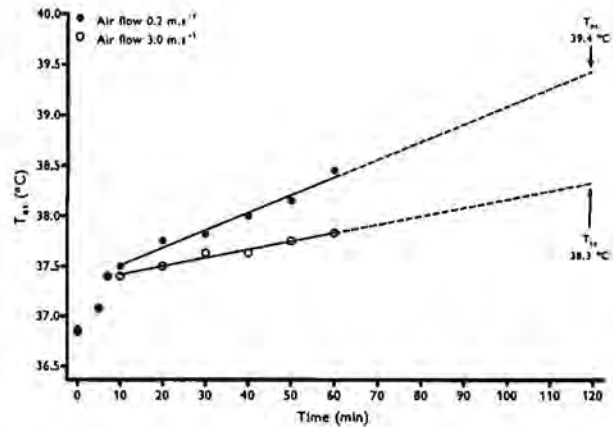
reached significance only after 120 min, at the final recording, and the difference in T_{re} at that time was only 0.2 °C (147). Although this remains a statistically significant difference, 0.2 °C is a relatively small difference and is indicative of the likely (quite small) magnitude of the effect of fluid ingestion on temperature regulation. Therefore the collective body of evidence from the above studies indicates that humans have a large capacity to store heat (for up to an hour) before then reaching a stage where increases in T_{re} are relatively small (< 0.3 °C per hour) even when no fluid is ingested. This suggests that the effect of fluid ingestion on temperature regulation during exercise is sufficiently small that an exercising individual is not placed at risk of heat stroke or “heat injury” even when ingesting no fluid during prolonged exercise.

The effect of active dehydration during exercise on temperature regulation has not been determined conclusively. The studies in this section represent controlled laboratory trials that examined the effects of fluid ingestion on temperature regulation *independent of metabolic rate*. However, early investigators clearly identified metabolic rate, both absolute and relative, as the primary determinant of T_{re} (12; 38; 163; 165; 166; 168; 198; 201; 255), and the absence of appropriate amounts of convection may amplify the apparently small effect of dehydration.

The air velocities in these experimental trials were very low and not representative of the pace at which trained subjects would be cycling or running at 60–70% of VO_{2max} . A more representative speed when cycling at that intensity is 35 km·h⁻¹ in cyclists, and at least 14 km·h⁻¹ in runners. This cycling speed was calculated with a road speed equation (48) by an estimation of a power output of 200 W (204 W mean in Montain and Coyle (147), which is 60% of PPO for a slightly above average cyclist PPO of 340 W), but by no means well trained (128). Therefore there is a large capacity for convective and evaporative heat loss that is not

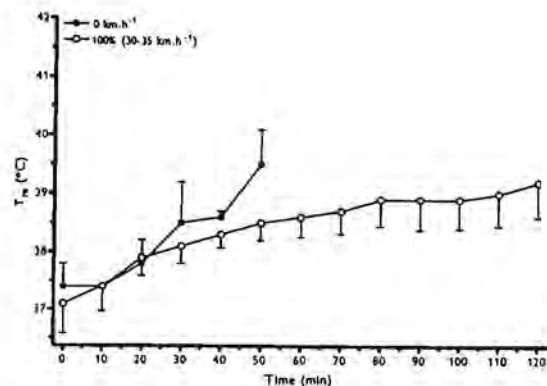
present in these trials but which would be available during out-of-doors exercise (147). Indeed increased rates of convection have been proven to have profound effects on temperature regulation (33; 45; 202; 214; 243) (Figure 6).

Figure 6. The effects of air flow on esophageal temperature during exercise in 35 °C as studied by Adams et al. (1992). The subjects exercised at 56% $\dot{V}O_{2\max}$ and did not ingest any fluids. The mean sweat rates were 1.3 $L \cdot h^{-1}$ in the low air velocity trial and 1.1 $L \cdot h^{-1}$ in the higher air velocity trial. The difference in temperature at the end of 60 min of exercise in this study was 0.54 °C. When extrapolated to 120 min of exercise, this difference becomes much larger at approximately 1.1 °C.



This lack of convective cooling might have amplified any effects of fluid ingestion on temperature regulation, leading to Type I error. This is illustrated in Figure 6 (from Adams et al. 1992 (3)) and in Figure 7 (from Saunders et al. (202)). The study of Adams et al. (3) investigated the effects of very low or high air velocity ($0.2 m \cdot s^{-1}$ vs. $3.0 m \cdot s^{-1}$) on T_{es} during 60 min of exercise at $\sim 56\% \dot{V}O_{2\max}$ in 35 °C. These data have been plotted in Figure 6, and when a linear regression is performed the difference in T_{es} after 120 min of exercise would be approximately 1.1 °C.

Figure 7. Selected data redrawn from Saunders et al. (2005). Subjects cycled at 60% PPO (183 W) in 33°C and 50% RH. There was no wind speed in the "0" trial and 50 min was the longest that all nine subjects completed. In the 100% trial the wind speed corresponded to their calculated road speed and was in the region of 30-35 $km \cdot h^{-1}$.



While the data from these studies represent well controlled laboratory investigations, the use of such data to help determine the best practices of athletes in field events is not logical. Although the studies reviewed in this section investigated the effects of fluid ingestion on temperature regulation, they did not examine any effects of fluid ingestion on exercise performance since they employed fixed-workload (and not self-paced) exercise bouts. Yet the findings from these experiments have been used to prescribe fluid replacement strategies that are purported to improve exercise performance or enhance thermoregulation during competitive events (40).

Fluid ingestion, temperature regulation, and exercise performance

This section will examine the studies that have emphasized the study of the effects of fluid ingestion on exercise performance as the primary outcome. Although most of the research investigating fluid replacement during exercise has employed sub-maximal, fixed workload exercise purely to study the physiological effects, numerous trials have sought to examine the effects of fluid balance on exercise performance. The performance tests are normally open-loop time to fatigue tests (4; 119; 141; 190; 247) or closed-loop time trials over a given distance (40 km) or amount of work (~500 kJ) or time (60 min) (15; 16; 21; 44; 109; 142; 199; 234; 248).

Open-loop performance and dehydration

Those studies that have used open-loop performance tests to measure the effects of dehydration on exercise performance typically consist of a fixed workload exercise bout followed by a time to fatigue exercise bout at a very high (>80% $\dot{V}O_2\text{max}$) intensity (4; 119; 141; 190; 247).

Adolph and Pitts (4; 190) studied the effects of open-loop performance tests (marching to fatigue) on soldiers in the heat. Pitts et al. (190) examined fixed-workload marching in the

heat while ingesting no fluid, *ad libitum* fluid intake, or 100% of weight losses. The findings show that when the soldiers consumed fluid *ad libitum*, after four hours T_{re} was 38.4 °C, and when replacing 100% of their sweat losses for four hours their T_{re} was 38.3 °C. These data suggest that there is no additive benefit to ingesting such large fluid volumes to replace 100% of sweat losses.

Adolph (4) on the other hand did not report on any outright performance tests, but instead investigated fluid ingestion during prolonged marching in the desert either with or without water. In this experiment Adolph showed that thirst was not the primary complaint given by the soldiers on why they stopped marching. Adolph also showed that even though one group of men was fluid restricted and had a 15% attrition rate (11 out of 70 men), the other group that was ingesting fluid *ad libitum* completed the march with only one failure, thus indicating that fluid restriction has some effect on performance when performance is defined as the ability to complete an endurance task.

Walsh et al. (247) examined mild levels of dehydration (1.8%) on high-intensity cycling performance. Six trained male cyclists exercised for 60 minutes at 70% VO_{2peak} before commencing a performance ride to fatigue at 90% VO_{2peak} . In one condition subjects consumed no fluid, while in the other they consumed fluid to match their weight losses. The first interesting finding from this study was that the T_{re} was similar in both experimental conditions (38.4 °C vs. 38.2 °C, fluid vs. no fluid, respectively), suggesting that thermoregulation is unaffected by fluid ingestion during one hour of moderate-intensity exercise in the heat (32 °C, 60% RH). The main finding of this study relevant to this section was that the time to fatigue was reduced in the no fluid trial (6.8 ± 3.0 vs. 9.8 ± 3.9 min).

The study of Walsh et al. also investigated the effect of rinsing the mouth with water in the 0% trial. They reported a significant effect of fluid ingestion on time to fatigue so that in the 100% trial the subjects cycled 9.3 ± 3.9 min compared to 6.8 ± 3.0 min in the 0% trial. Hence there was no effect of mouth rinsing on the performance variable so that subjects fatigued earlier during the performance test in the 0% trial.

Finally, McConell (141) examined seven trained males who completed two hours of cycle ergometry at 69% VO_2max while ingesting no fluid, enough to maintain body weight, or 50% of this volume. The findings were the same as previous studies (41; 72; 147)—there was a graded effect of the fluid ingestion on the rectal temperature so that when the subjects replaced all of their weight losses the rectal temperature was lower. Immediately following the exercise bout, the subjects cycled at 90% VO_2max until volitional fatigue. A graded effect of dehydration on the open-loop exercise performance was shown since subjects cycled longest in the full replacement condition (328 ± 93 s), shortest in the no fluid condition (171 ± 75 s), and in between in the 50% condition (248 ± 107 s).

A limitation to these previous studies, however, is that the performance tests consisted of open-loop exercise to fatigue. These tests have large variation (10-20%) and poor repeatability (15-25%) (101). Such large variations make it difficult to interpret the effects of the experimental treatment on the prescribed performance measure. More importantly, however, these experimental trials do not represent the effects of dehydration on performance, but rather the effects of *hypohydration* on performance since the subjects all began the performance aspect in much similar circumstances as subjects in other more explicit fluid restriction and hypohydration studies (7; 8; 37; 160; 210) in that they are beginning the performance trial at a certain level of dehydration. Therefore what these studies (141; 247) prove is that when

athletes begin a performance bout in a dehydrated state, performance and thermoregulation appear to be affected.

Closed-loop performance and dehydration

Closed-loop performance trials have less day to day variability and are therefore a more reproducible and more reliable protocol to assess exercise performance. Typically the studies using closed-loop performance tests use an outright time trial or used a shorter duration time trial preceded by a steady-state exercise bout (15; 16; 21; 44; 109; 142; 199; 234; 248).

Marino et al. (134) developed a reproducible and variable intensity cycling performance test which they used subsequently to test various fluid ingestion and temperature interventions on exercise performance. The test is a one-hour cycling time trial in which the subjects must complete one minute sprints at 10 minute intervals. Performance is measured as the total distance each subject completes in the one hour. The CV for this test after a familiarization trial was found to be 1.34%.

Kay et al. (109) used this protocol to investigate the effects of complete (100%) or incomplete (0%) fluid replacement on cycling performance in both moderate (19.8 ± 0.6 °C) and hot (33.2 ± 0.2 °C) conditions. Their hypothesis was the ingested fluid would create a "heat sink" so that more heat could be stored by the body, therefore enhancing an individual's capacity to perform work. However, the subjects cycled similar distances regardless of the fluid and environmental interventions. Furthermore, the post-trial T_{re} 's were similar and the pacing strategies used by the subjects did not vary between the experimental conditions.

Other investigators (199) used a similar duration trial (60 min) but without the intermediate sprints to test 100% vs. 0% fluid replacement in moderate ambient conditions (20 °C, 60% RH, 3 m·s⁻¹ wind speed). Eight endurance trained males cycled as far as possible in one hour while either attempting to replace 100% weight losses (mean ingested volume = 1.49 ± 0.14 L) or ingesting no fluid. While the fluid ingestion resulted in lower heart rates and lower serum osmolalities, there was no effect on T_{re} . Most importantly, though, was that these investigators reported that the fluid ingestion resulted in uncomfortable levels of fullness and shorter distances covered in the one hour time trial (43.1 ± 0.7 vs. 42.3 ± 0.6 km, no fluid vs. fluid, respectively). Thus the fluid ingestion resulted in subjects cycling 800 m less, suggesting that there was no benefit of full fluid replacement in moderate ambient conditions during short duration exercise. Rather, attempting to ingest such large volumes of fluid had a detrimental effect on cycling performance.

Below et al. also used a ~60 min duration protocol to test the effect of both CHO and fluid ingestion in a warm environment (31 °C, 54% RH) (21). The subjects ingested either a large (1300 mL) or a small (200 mL) volume of fluid. CHO ingestion was controlled for since the fluid ingested in the smaller volume trial was 40% maltodextrin so that the total amount of CHO ingested during all the trials was ~80 g. However, the experimental protocol in this study was not a 60 min performance test as it consisted of 50 min of cycling at 80% VO_2max followed by a performance test in which a pre-determined amount of work (150-200 kJ) had to be completed.

These investigators (21) did find a significant difference in esophageal temperature between the large and small fluid volume trials. However, as in many other studies that demonstrate an effect of fluid ingestion on temperature regulation, the effect was small (0.33 °C). In addi-

tion, the increase in esophageal temperature during the performance test was apparently identical between the large and small volume trials. More importantly they found an effect of fluid ingestion on the exercise performance so that in the large fluid volume trial the subjects cycled at a power output 18 W higher in the large fluid trial, which resulted in a performance enhancement of 42.6 s.

These authors (21) conclude that the ingestion of the large volume of fluid is the key factor that enhanced performance in this trial. However, another possibility is that the restriction of fluid below *ad libitum* values created a reduction in performance. One limitation of this study is that there was no trial in which subjects were allowed to ingest fluid *ad libitum*. The inclusion of this trial would have determined if such large volumes (~1300 mL) are necessary to enhance performance, or if performance is optimized when drinking *ad libitum*.

Using the already established protocol of a 60 min performance trial similar to Below et al. (21), the subjects in McConell et al. (142) cycled for 45 minutes at 80% VO_2max followed by a 15 min performance test in which subjects were encouraged to perform as much work as possible. The primary differences in study design from Below et al. (21) is that this study was performed in neutral ambient conditions of 21 °C and 41% RH, and that in this study there was a third fluid intervention so that the subjects ingested 0, 50, or 100% of their weight losses. These authors found no differences in performance, HR, or T_{re} between any of their experimental conditions.

Dairies et al. studied the effects of fluid ingestion on running performance (44). Although the total duration of the trial was two hours, this consisted of 90 minutes of fixed-workload exercise (65% VO_2max) followed by a 30 minute performance test in which the subjects ran as far

as possible. This study was conducted in neutral ambient conditions of 25 °C and 55% RH, but with appropriate amounts of convection (13-15 km·h⁻¹). During the 90 min steady-state part of each trial the subjects ingested fluid at rates of either 350 mL·70 kg⁻¹, 150 mL·70 kg⁻¹, or *ad libitum*.

The main finding of Dairies et al. (44) was that the additional fluid intake in the 350 mL·70 kg⁻¹ trial resulted in severe gastrointestinal discomfort so that two of the eight subjects could not complete the performance trial. Furthermore, no differences in plasma volume, urine production, or sweat rates were found between any of the trials. However, although differences were not significant, perhaps because of small subject numbers, subjects ran at the fastest speed during the performance trial that followed the *ad libitum* drinking intervention, at an intermediate speed following the full fluid replacement, and at the slowest speed when no fluid was ingested.

Although most studies used trained recreational athletes, Tatterson et al. (234) studied the T_{re} responses of elite Australian road cyclists to 30 min cycling time trials in both hot (32 °C, 60% RH) and neutral (23 °C, 60% RH) ambient conditions. Although there was no fluid intervention, the importance of this study is that the T_{re} response was similar between the hot and cold conditions even though the mean power output was 6.5% lower in the heat. This suggests an anticipatory reduction in power output in the heat specifically to ensure that a critical elevation of T_{re} did not occur during exercise in the heat.

While other studies discussed in this section manipulate the ingested volume of fluid, Bachle et al. (15) investigated the effects of the type of fluid ingested by having both male and female subjects cycle as far as possible for 60 min while ingesting 1200 mL of either water or a 6%

CHO sports drink. In a third trial the subjects ingested no fluid. The ambient conditions were neutral (19-21 °C, 72% RH). The major limitation to this study was that T_{re} was not measured and that the subject population consisted of recreational athletes and not trained cyclists. There was no effect of the type of fluid on performance. In addition, the ingestion of the 1200 mL of fluid did not enhance performance compared to when the subjects ingested no fluid.

Finally, in 2003 Backx et al. (16) examined the effects of fluid ingestion on one-hour self-paced time trial performance of trained cyclists in neutral conditions (20 °C, 70% RH). The authors controlled for CHO ingestion by having subjects ingest a bolus of a CHO drink 10 min prior to the start of each time trial. The volume of fluid was then altered during each of three experimental trials. Similar to Bachle et al. (15), this study did not measure T_{re} , and also found no differences between the trials when the subjects ingested small (120 mL), medium (450 mL), or large (1200 mL) volumes of fluid.

To summarize, the studies reviewed in this section that have examined fluid ingestion and exercise performance have two common characteristics. First, the experimental interventions consist of replacing 100% or 0% of fluid losses, with no studies investigating if there is a graded effect of the fluid ingestion on performance. Second, and more importantly, the performance trials in these studies are only one hour in duration.

The importance of these studies is that they demonstrate that during exercise lasting approximately one hour, replacing 100% of weight losses does not appear to enhance performance. Alternatively, replacing 0% of weight losses does not appear to reduce performance, suggesting that in events lasting approximately one hour or less an athlete will receive no

benefit from fluid ingestion. The reason for this perhaps lies in the findings of the early researchers (165; 168), who showed that during prolonged exercise in both cool and hot conditions, the T_{re} rises for approximately one hour before reaching a steady state level. The fact that T_{re} rises continuously during this time period can mean only that the body is in a state of net heat gain. Furthermore, a rise in temperature during this time period appears to be a desired outcome, for the subjects in these studies are sweating at sub-maximal rates. If a lower temperature were desired the body would only have to increase the sweat rate, which would result in a higher volume of evaporated sweat and therefore a higher rate of heat loss. Therefore the available evidence shows that for approximately one hour the body 1) sweats at sub-maximal rates and 2) stores heat, the result of which is a continuous rise in the T_{re} during this time, after which it is regulated at a new higher level for the remainder of the exercise bout.

Summary

The evidence from the literature tells a specific story about how the body regulates its temperature during exercise. The earliest researchers of this topic identified metabolic rate as the best predictor of T_{re} . Furthermore, this finding holds true independent of the environmental conditions so that in either cool (<10 °C) or hot (up to 30 °C) conditions, the T_{re} response is similar when the exercise intensity is controlled. Successive investigators further found that the T_{re} response is a function not of absolute metabolic rate, but rather a function of the relative metabolic rate so that even if the energy expenditure between individuals varies, their T_{re} responses are similar.

The importance of these early findings is that they remain largely overlooked by modern scientists. The 1960's appear to signal a shift in thinking, after which time the concept that the

fluid balance plays a large role in determining the T_{re} response came to the fore and has remained there to the present. Therefore the current model used to explain and understand thermoregulation proposes that the level of dehydration is the most important predictor of the T_{re} . In this model, the role of the metabolic rate is essentially forgotten.

Research performed during the past 30 years has illustrated clearly that fluid status does have some effect on temperature regulation. However, this effect is small, and in comparison to the effect(s) of the metabolic rate is nominal.

Research questions

Having reviewed the literature and identified gaps in the body of evidence in this area of temperature regulation and fluid ingestion, the bases for this thesis revolve around the following questions, which will be answered throughout the text:

Question 1

What are the fluid replacement strategies and post-race plasma $[Na^+]$ concentrations in endurance cycling?

Fluid replacement and post-race serum $[Na^+]$ in runners has been widely described, but the same cannot be said for cycling. We identified the need to study this population and its fluid replacement strategies and post-race serum $[Na^+]$, and accordingly measured these variables during an annual one-day 109 km cycle race.

Question 2

What are the rectal temperature responses to endurance exercise during actual running and cycling races when athletes replace fluid ad libitum, and how critical are these temperatures with regard to the development of "heat illness?"

The post-race T_{re} in marathon runners (27; 136-138; 193) and other runners (198) has been reported previously, and both Maron et al. (138) and Myhre et al. (159) reported semi-continuous measurements of marathon runners during their respective races. However, the most recent technology allows for much more frequent and therefore continuous measurement of the T_{re} during endurance exercise. The important contribution of this thesis is to examine the T_{re} response to ultra-marathon running (>42.2 km) and to endurance cycling, neither of which has been reported previously.

Question 3

How does fluid ingestion affect temperature regulation and performance in hot and humid environments?

Studies that have investigated the effects of fluid ingestion on temperature regulation when controlling for work rate are numerous. Some of these studies have attempted to measure the effects of fluid ingestion on performance by introducing either a closed-loop or open-loop

performance test at the end of a fixed-workload protocol. Other studies have used experimental interventions that consist of either 100% or 0% fluid replacement and performance tests of only one-hour in duration. Therefore the effects of varying amounts of fluid ingestion on both the T_{re} response and performance are not well known, and we aimed to test this relationship in this thesis.

Question 4

Is there a graded effect of dehydration on exercise performance in the heat so that ingesting more fluid will enhance time trial performance?

The effect of fluid ingestion on temperature regulation has been shown to occur in a graded manner. Furthermore, high rates of fluid ingestion are prescribed on the basis that ingesting more fluid, and therefore preventing any weight losses during exercise, will enhance performance. Yet all of the studies that have previously examined performance and dehydration have used experimental interventions only of either 0% or 100% fluid replacement and have not tested the effects of varying amounts of fluid on performance. Finally, the performance trials in these studies are 60 minutes or less in duration, and so no studies to date have examined the effects of different volumes of fluid ingestion on higher-intensity performance of a more prolonged nature.

Chapter 2

Lower sweat rates but same rates of fluid ingestion in women and men during a one-day 109 km cycle race

Introduction

There is an established consensus that exercise-associated hyponatremia (EAH) occurs as a result of over-consumption of fluid (6; 68; 69; 97; 186; 225; 228) and that it occurs more frequently in women than in men (223). One possible reason why women are more likely to develop fluid overload during exercise than are men could be that the fluid requirements of women are less than are those of men. To our knowledge there are no large scale studies of fluid balance in competitive, out-of-doors activities in men and women matched for performance time. Yet such data clearly are important if drinking guidelines appropriate to both men and women are to be produced. A consideration of first principles suggests that since sweat rate is a function of the absolute metabolic rate (177), then the smaller mass of women should cause them to sweat at a lower rate than do men when cycling at the same velocity, therefore requiring smaller absolute fluid volumes during exercise. In fact this appears to have been studied previously (24; 63; 156; 250), yet all the fluid replacement guidelines fail to address this sex difference (32; 40; 73; 140; 153).

Accordingly the aim of this study was to determine the rates of weight loss and fluid intake in a representative cross section of men and women cyclists matched for finishing time in a one-day, 109 km cycle race.. Such data would allow the comparison of both estimated sweat rates and drinking behaviors in a large sample of men and women cyclists exercising at similar absolute velocities. A secondary aim was to determine the prevalence of EAH in this population after they had been advised about the dangers of overdrinking during prolonged exercise, as was the case in the 224 km 2001 South African Ironman triathlon (215).

Methods

Subjects

Male and female cyclists were recruited for this study via email during the weeks preceding the race. The study adhered with the guidelines of the Declaration of Helsinki during all phases of this research. All procedures were approved by the Research Ethics Committee of the Faculty of Health Sciences at the University of Cape Town and were explained clearly in the recruitment email message. Potential subjects were encouraged to ask questions about the experimental protocols over a two to four week period preceding the race. Each subject's affirmative electronic reply was accepted as his or her informed consent. The recruitment message was sent to 2000 race entrants. From this pool 243 replied and consented to participate in the research protocols. All subjects who provided their consent electronically were included in the study population and were informed via email in more detail of the pre-race and post-race weighing procedures and of the post-race blood sampling procedures to confirm risks, clarity of the information they received electronically, and their expectations. Subjects were regularly informed in each round of correspondence that they were permitted to withdraw from the study at any point without prejudice or penalty.

On the race day 196 cyclists (N = 93, women; N = 103, men) completed all of the research protocols. From this final pool of 196 cyclists, 60 men and 60 women were matched for finishing time to produce two groups with matched performances in the cycle race (Men, N = 60; and Women, N = 60).

Experimental Procedures

Subjects were weighed on three scales. All scales were accurate to 0.1 kg. All three scales were checked the day prior to the race to ensure inter-scale consistency. All scales read

equal values and were therefore used interchangeably for the measurements performed on the race day.

Prior to entering their starting areas for the race, cyclists reported to the designated research area and their weights were recorded. Cyclists were weighed with their shoes on, the cycling helmet removed, and their pockets empty. Subjects were instructed to complete the race as they normally would without any intervention(s) from the research team. Upon completing the race, subjects reported immediately to another designated research area at the finish, located approximately 20 m from the bicycle storage facilities, and a post-race weight was recorded in the same manner as the pre-race weight. After weighing, each cyclist reported to the trained medical staff who obtained a 2-3 mL blood sample by veni-puncture via a Vacutainer system drawn into a tube containing lithium heparin. Whole blood samples were then analyzed for sodium concentration ($[Na^+]$) on a Chiron 348 blood/gas analyzer.

Each subject was then interviewed to determine the volume of fluid ingested during the race. The volume of each drinking bottle that had been used by each athlete was measured and recorded as was the volume of fluid ingested from any partially-emptied bottle(s). These volumes were summed to calculate the total fluid intake. Together with the pre-race to post-race change in mass, the total ingested fluid was used to estimate the sweating rate. Sweating rates were not corrected for respiratory or urinary losses, or metabolic water, all of which were assumed to be sufficiently small not to influence materially the estimated sweat rates in this large sample.

Weights were not corrected to obtain an estimation of nude body mass. It was assumed that since all cyclists wore standard cycling clothing, each had similar volumes of sweat in their clothing at the end of the race.

Statistical Analysis

An analysis of variance (ANOVA) was used to determine pre-race to post-race differences in body mass. All other variables were analyzed with independent T-Tests between groups. A general linear regression model was used to analyze the relationships between specific variables.

Results

Environmental Data

Ambient temperature and relative humidity data were obtained from the South African Weather Bureau from a weather station located approximately three kilometers from the start and finish areas. The ambient temperature at 6:00 AM was 18.3 °C and the relative humidity was 99%. At 9:00 AM the ambient temperature was 19.6 °C and the relative humidity was 99%. At 12:00 PM the ambient temperature was 21.7 °C and the relative humidity was 77%. Finally, at 4:00 PM the ambient temperature was 23.4 °C and the relative humidity was 75%. The wind conditions were calm throughout the day.

Race Details

A total of 27,470 cyclists finished the race. The total number of female finishers was 5291 and the total number of male finishers was 22,179. The distribution by finishing time for all race finishers and for the males and females in this study are presented in Figure 1. Subjects' and all finishers' race times are presented in Table 1. The women in the study were significantly faster by approximately 40 minutes than the total of all female race finishers. As a result, the

average race finishing time for the women and men in this study was significantly faster than was the average for the total group of all cyclists in the race.

Table 1. Values are mean \pm SD. Race times of subjects (N = 60, men; N= 60, women) and all race finishers (N = 22,179, men; N = 5291, women). Pre- and post-race weights, post-race plasma Na⁺ concentrations, total and hourly volumes of fluid ingestion, estimated sweat rates, and percent replacement of weight losses in both men and women subjects.

	Women	Men	Women and Men
Subjects' Race Times (min)	259 \pm 47 [*]	256 \pm 46	258 \pm 47 [†]
All Finishers' Race Times (min)	298 \pm 54	262 \pm 55	269 \pm 57
Pre-race Weight (kg)	63.9 \pm 8.5 [‡]	85.0 \pm 15.6	74.4 \pm 16.4
Post-race Weight (kg)	63.0 \pm 8.3 ^a	83.0 \pm 15.3 ^a	73.0 \pm 15.8 ^a
Post-race [Na ⁺] (mmol·L ⁻¹)	140.7 \pm 3.0 ^b	142.9 \pm 2.3	141.8 \pm 2.9
Fluid Ingestion (mL·h ⁻¹)	389 \pm 149	419 \pm 170	406 \pm 160
Fluid Ingestion (mL·kg ⁻¹)	26 \pm 12 ^b	21 \pm 7	24 \pm 10
Sweating Rate (mL·h ⁻¹)	602 \pm 168 ^c	901 \pm 279	752 \pm 273
Replacement of Weight Losses (%)	66.9 \pm 33.7 ^b	47.3 \pm 16.0	57.2 \pm 28.0

*	Significantly different from all women race finishers	p < 0.01
†	Significantly different from both men and women race finishers	p < 0.05
‡	Significantly different from men pre-race weight	p < 0.01
^a	Significantly different from pre-race weight	p < 0.01
^b	Significantly different from men	p < 0.01
^c	Significantly different from men	p < 0.02

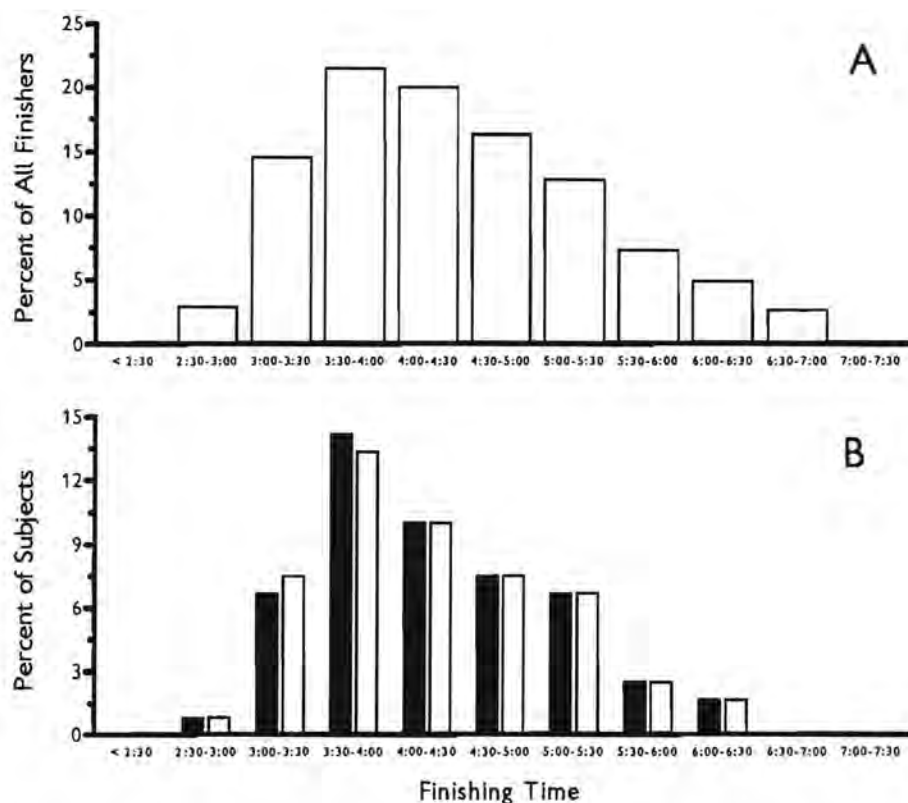


Figure 1. Distribution by finishing time for all cyclists in the race (A), and distribution by finishing time for male (shaded bars) and female (clear bars) subjects (B).

Weight Changes and Post-race plasma Na^+ concentrations

Pre- and post-race weights are presented in Table 1 and absolute (kg) and percent changes in weight are presented in Figure 2. Women lost significantly less weight than the men (-0.9 ± 0.7 kg vs. -2.0 ± 0.9 kg, women vs. men, respectively; $p < 0.001$) (Figure 2a) and consequently had smaller relative percent changes ($-1.4 \pm 1.1\%$) than the men ($-2.4 \pm 0.9\%$; $p < 0.001$) (Figure 2b).

Post-race plasma $[\text{Na}^+]$ are shown in Table 1. The mean post-race plasma $[\text{Na}^+]$ was significantly lower in the women compared to the men by $2.2 \text{ mmol}\cdot\text{L}^{-1}$ ($p < 0.001$) (Table 1). The distributions of post-race plasma $[\text{Na}^+]$ are presented in Figure 3 and represents a normal Gaussian distribution.

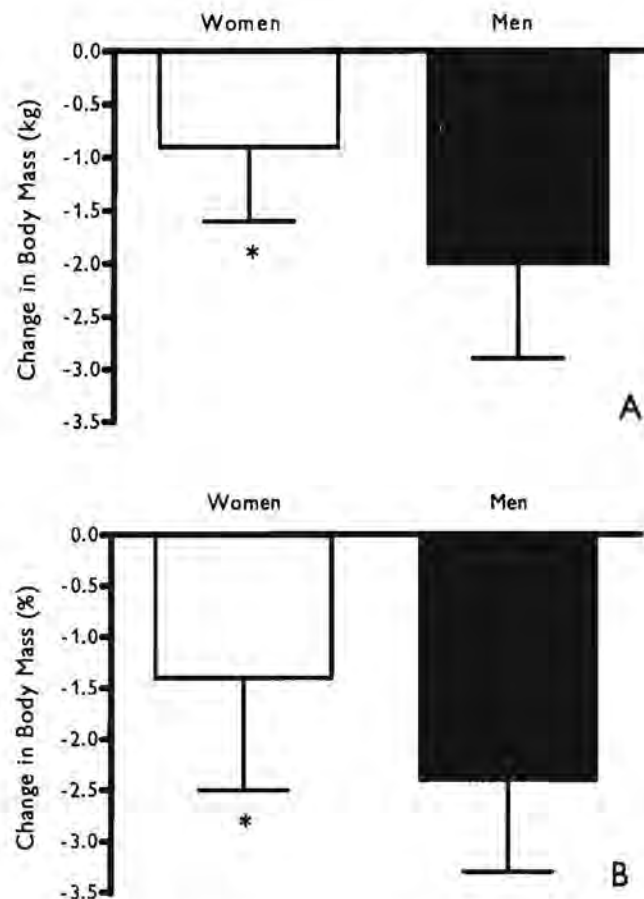


Figure 2. Absolute (A) and percentage (B) changes in mass in women (N = 60) and men (N = 60) cyclists in the study.

* Significantly different from men (p < 0.001)

Fluid Ingestion and Sweating Rates

Hourly and per kilogram rates of fluid ingestion are listed in Table 1. Men and women in this study ingested similar hourly volumes of fluid during the race (Table 1), although women ingested significantly more fluid per kg body weight ($26 \pm 12 \text{ mL}\cdot\text{kg}^{-1}$) compared to the men ($21 \pm 8 \text{ mL}\cdot\text{kg}^{-1}$) (p < 0.001).

Estimated hourly sweating rates are listed in Table 1. The mean rate of sweating in the women was 33% lower than that of the men (603 ± 168 vs. $901 \pm 279 \text{ mL}\cdot\text{h}^{-1}$) (p < 0.001), while women replaced a significantly higher percentage of their weight losses ($66.9 \pm 33.7\%$) compared to the men ($47.6 \pm 16.0\%$) (p < 0.001) (Table 1). In addition, women had signifi-

cantly lower estimated rates of sweating relative to body weight compared to the men (40 ± 11 vs. 45 ± 10 mL.kg⁻¹; $p < 0.02$). The mean per kilogram sweating rate in all subjects was 42 ± 11 mL.kg⁻¹.

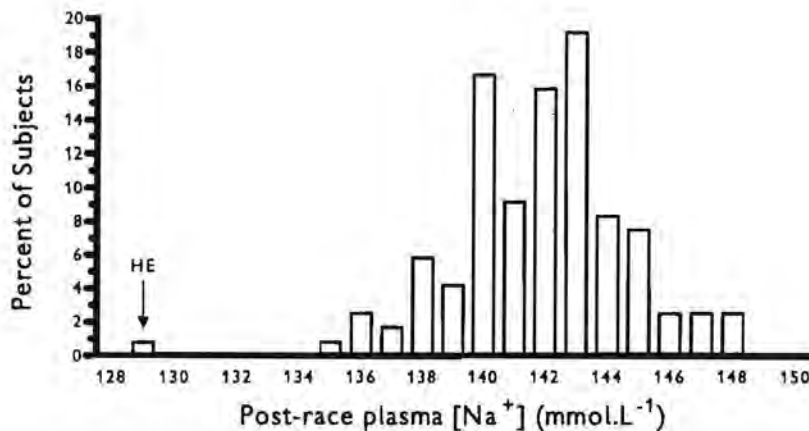


Figure 3. Distribution of post-race plasma [Na⁺] in all subjects (N = 120). "HE" denotes one female subject with exercise-associated hyponatremic encephalopathy and is the subject of further discussion in Chapter 6.

Post-race plasma [Na⁺] in all subjects were linearly related to percentage weight loss ($r^2 = 0.25$; $p < 0.001$) (Figure 4a). In the women this relationship was stronger ($r^2 = 0.39$; $p < 0.001$) (Figure 4b), while in the men there was no relationship (Figure 4c). One athlete developed hyponatremic encephalopathy with a plasma sodium concentration of 129 mmol.L⁻¹. She was the only athlete in this study to gain significant weight (approximately 3.5%) during the race (arrowed "HE" in Figures 4, 5a, and 5b).

In all subjects the percentage of weight losses replaced by the subjects was linearly related to the post-race plasma Na⁺ ($r^2 = 0.29$; $p < 0.001$) (Figure 5a). In the women the relationship was stronger ($r^2 = 0.35$; $p < 0.001$) (Figure 5b) while in the men there was no relationship (Figure 5c). However in all groups the scatter of plasma [Na⁺] at any body weight change was large (10-15 mmol.L⁻¹) indicating that factors other than body weight changes alone determine the post-race plasma [Na⁺] (181).

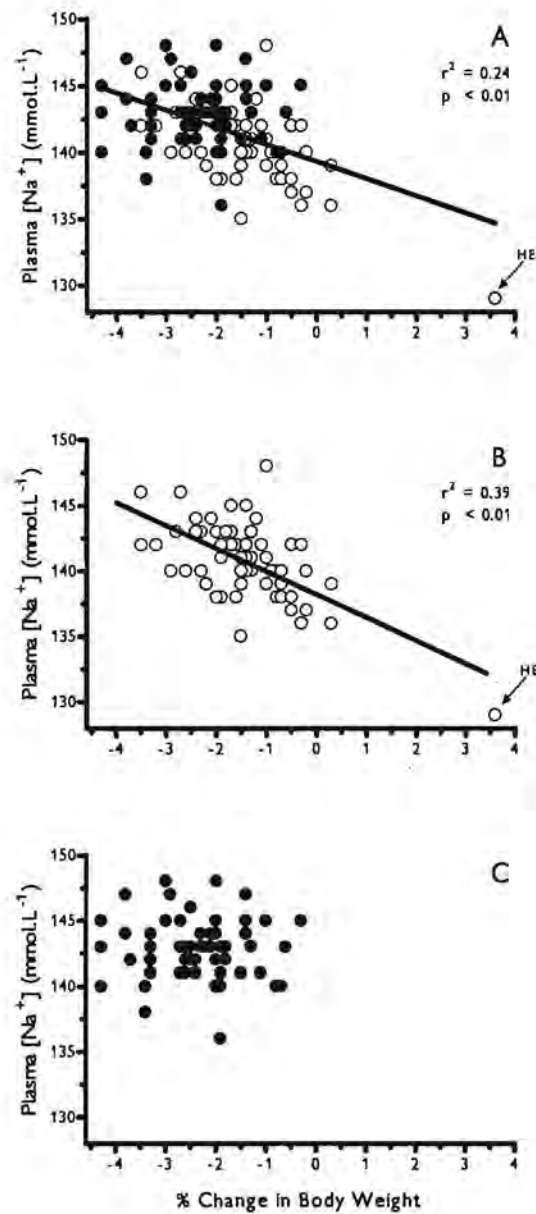


Figure 4. Relationships between the percentage change in mass and post-race [Na⁺] in all subjects (A; N = 120), women (B; N = 60) and men (C; N = 60). "HE" denotes the one hyponatremic subject who is discussed in Chapter 6.

Significant relationships were found between the pre-race mass and the total volume of estimated sweat losses in women, men and all subjects (Figure 6).

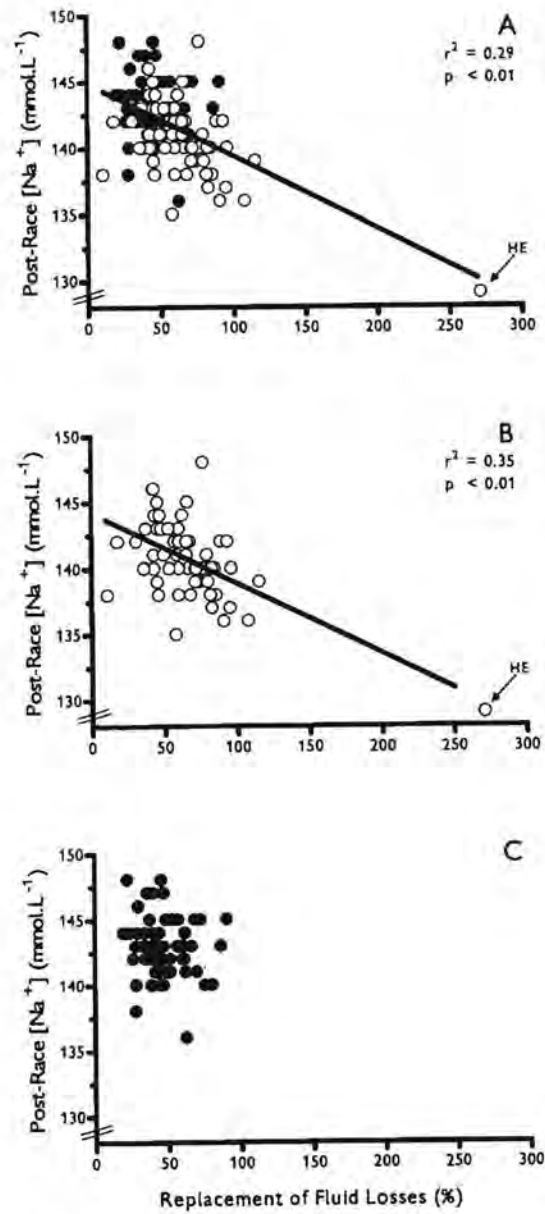


Figure 5. Relationships between the percentage of fluid losses replaced during the race and the post-race plasma [Na⁺] in all subjects (A; N = 120), women (B; N = 60) and men (C; N = 60).

This relationship was most marked in all subjects ($r^2 = 0.53$; $p < 0.001$) (Figure 6a), but was also significant in both the women ($r^2 = 0.31$; $p < 0.001$) (Figure 6b) and the men ($r^2 = 0.38$; $p < 0.001$) (Figure 6c).

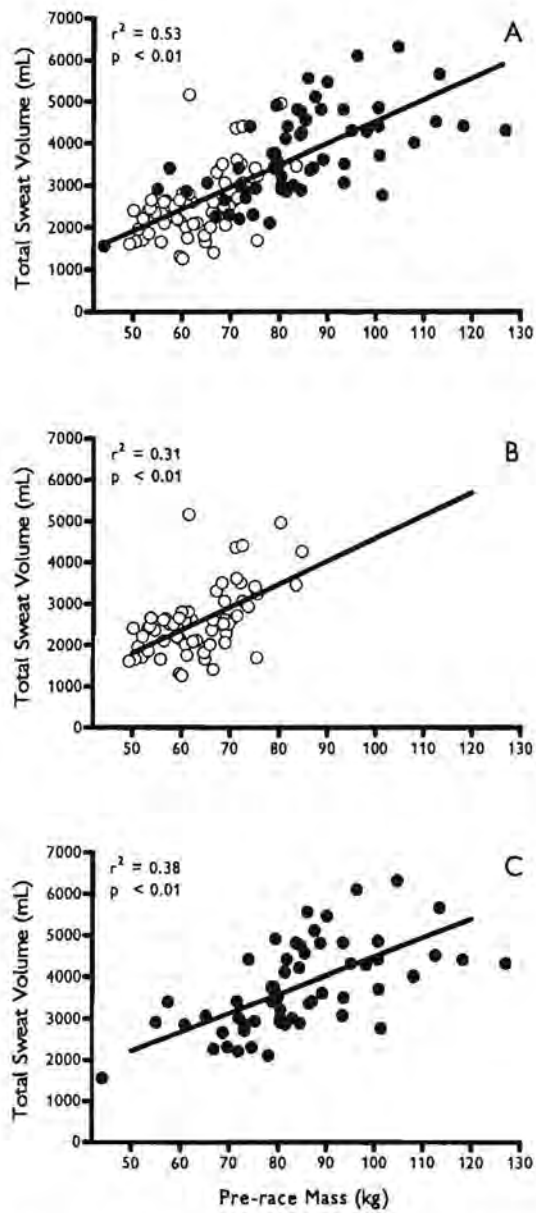


Figure 6. Relationships between pre-race mass and total volume of estimated sweat losses in all subjects (A; N = 120), women (B; N = 60) and men (C; N = 60).

Discussion

To our knowledge this study is the largest yet reporting fluid balance and fluid ingestion practices during a competitive sporting event including the largest cohort of female athletes.

The first important finding of this study was that the post-race plasma $[\text{Na}^+]$ were distributed normally over a range of $135 \text{ mmol}\cdot\text{L}^{-1}$ to $149 \text{ mmol}\cdot\text{L}^{-1}$ (Figure 2), with one female subject finishing the race with a plasma $[\text{Na}^+]$ of $129 \text{ mmol}\cdot\text{L}^{-1}$. This athlete developed exercise-associated hyponatremic encephalopathy (EAHE) as a result of a body weight gain of 2.4 kg during the race and is the subject of a separate case study (50). This range of plasma $[\text{Na}^+]$ appears to represent a healthy response to endurance exercise as previously shown in a group of 226 km Ironman triathletes who were also encouraged to ingest fluid "ad libitum" during prolonged exercise and to desist from drinking too much (215).

The second important finding was that there were marked sex-related differences in estimated sweat rates (both absolute and relative), relative rates of fluid ingestion, and the extent of fluid replacement in men and women matched for race finishing times.

Women sweated significantly less than did men (Table 1) even though they cycled at the same speed (Table 1). Since women were approximately 20 kg lighter than men, their lower sweat rates were likely the result of a lower metabolic rate during cycling since the energy cost of cycling is a function of the cyclist's body size, mass, and aerodynamic profile, all of which will be lower in smaller athletes (150; 188). The approximately 33% higher sweat rate in the men matches their approximately 33% higher body mass and hence their higher absolute rates of energy expenditure during the 109 km race. This finding is illustrated in Figure 6, in which

there is a strong relationship ($r^2 = 0.53$, $p < 0.001$) between the pre-race mass and the total volume of estimated sweat losses in all subjects

Most importantly, however, although they had lower sweat rates the women drank at the same rate as the men (Table 1). As a result women replaced a higher percentage of their weight losses. Thus whereas the men replaced only $47.3 \pm 16.0\%$ of their weight losses, the women replaced $66.9 \pm 33.7\%$ of their weight losses, or approximately a 20% greater replacement value than the men. Interestingly, the men's replacement value of 47% corresponds to other *ad libitum* replacement values reported elsewhere in the literature (99; 124; 197; 200), while the women's replacement value of 67% corresponds to female runners' *ad libitum* replacement values of 60-70% reported by Cheuvront and Haymes (34).

The hourly fluid intake rates of $393 \pm 156 \text{ mL}\cdot\text{h}^{-1}$ for the women and $412 \pm 164 \text{ mL}\cdot\text{h}^{-1}$ for the men are similar to the drinking rates in a number of other studies (34; 197; 223). Cheuvront and Haymes (34) reported *ad libitum* hourly rates of fluid intake that ranged from 130-470 $\text{mL}\cdot\text{h}^{-1}$ in cool conditions, 260-540 $\text{mL}\cdot\text{h}^{-1}$ in moderate conditions, and 310-700 $\text{mL}\cdot\text{h}^{-1}$ in hot conditions in female runners running a 30 km indoor treadmill run at their 42.2 km marathon pace.

Those data (34) suggest an environmental effect on rates of fluid ingestion during exercise. For example, when the women runners in that study completed the 30 km run in hot conditions, they consumed 180-230 $\text{mL}\cdot\text{h}^{-1}$ more than when they ran in the cool conditions. The environmental conditions in the 2003 Cape Argus/Pick 'n' Pay Cycle Tour matched those in the mild condition in the study of Cheuvront and Haymes (34), as did the female subjects' rates of fluid ingestion.

The third finding was the inverse relationship between the post-race plasma sodium concentration and weight loss during the race as frequently reported (97; 181; 181; 225; 227).

In this context, it is interesting to estimate what would have happened if these cyclists had adopted the current guidelines that all athletes should drink $1.2 \text{ L}\cdot\text{h}^{-1}$ or “as much as tolerable” during prolonged exercise (40). Since sweat rates in the men and women in this study were respectively 901 ± 279 and $602 \pm 168 \text{ mL}\cdot\text{h}^{-1}$, had they followed these guidelines, the men would have gained $1.4 \pm 1.3 \text{ kg}$ (1.5%) and the women $2.6 \pm 0.9 \text{ kg}$ (4.1%). According to Figure 4a and to calculations made by Weschler (251), such acute increases in body weight (and hence total body water) would have caused the mean post-race plasma $[\text{Na}^+]$ to fall to $137 \text{ mmol}\cdot\text{L}^{-1}$ compared to the measured mean value of $142 \text{ mmol}\cdot\text{L}^{-1}$. This decrease in plasma $[\text{Na}^+]$ would occur independently of the type of fluid the athletes ingested, since CHO and electrolyte-containing sports drinks are hypotonic and therefore have only a small effect on the decrease in plasma $[\text{Na}^+]$ in those who overdrink (17; 19; 251).

Rather, it appears that the advice to drink “*ad libitum*” was more appropriate. For the sole athlete to ignore this advice was the only one in the study of 196 athletes to develop EAHE (arrowed “HE” in Figures 4 and 5) (50). We (180) and others (227) have shown that EAHE can essentially be eliminated from endurance events if strategies to reduce fluid availability during these events are introduced. This study suggests that the same applies for a 109 km cycling race.

Some researchers have hypothesized that a loss of sodium in the sweat produces hyponatremia (42; 100; 155; 157; 187). If this model is correct, and if sweat Na^+ losses cause hypona-

tremia, then those athletes who sweat the most (and therefore lose the most weight and Na^+) during any activity must be at the greatest risk for developing hyponatremia. This would result in an inverse relationship between the total volume of sweat and the post-race plasma $[\text{Na}^+]$.

However, data from this study do not support that theory (Figure 6). There was a very weak positive relationship ($r^2 = .07$) between the total weight losses and the post-race plasma $[\text{Na}^+]$, indicating that those cyclists with the highest estimated total sweat losses generally had the higher post-race plasma $[\text{Na}^+]$, a finding consistent with and explained by Noakes et al. (181).

Furthermore, if EAH were related to Na^+ lost in sweat, then the fastest athletes (i.e. those with the highest metabolic and therefore the highest sweat rates) should be the most prone to developing hyponatremia during endurance exercise. For the same reason the heaviest athletes with the largest mass would also be at the greatest risk for developing hyponatremia since mass is strongly related ($r^2 = 0.53$; Figure 6) to sweat losses.

However all studies show the opposite; that is, those who complete athletic contests in the slowest times and who must therefore have the lowest metabolic and sweat rates are at increased risk for developing hyponatremia, because they have the lowest rates of water excretion and are the most likely to drink to excess during exercise (6; 169; 174).

Finally we found a mild ($r^2 = 0.14$) relationship between the race time and total volume of fluid ingested, which suggests that as the cyclists spent more time on the race course, so their total volume of ingested fluid increased. This has been reported in runners (6; 97) and sug-

gests that it is easier to drink when exercise is performed at lower intensities. This could be as a result of behavioral responses—physiological responses would include a lesser inhibition of gastric emptying and intestinal absorption at lower exercise intensities; while behavioral responses would include more time to spend drinking since the performance imperative is less.

In conclusion the data from this study show a clear sex-effect on fluid balance during prolonged cycling. In particular, women had lower sweat rates when cycling at the same speed as the men. But because they consumed fluids at the same rate as the men, they lost significantly less weight during the race and consequently had lower post-race plasma $[\text{Na}^+]$ (Table 1). Furthermore the relatively modest rate of fluid consumption for the men appears to have been appropriate since they finished the race without specific symptoms and with modest post-race hypernatremia (mean plasma $[\text{Na}^+] = 142.9 \pm 2.3 \text{ mmol}\cdot\text{L}^{-1}$).

Therefore we conclude that when cycling at the same speed, recreational male and female cyclists of different body weights have different sweat rates but which are lower in the women. As a result, the fluid replacement requirements of men and women are different during endurance exercise.

This conflicts with the current guidelines (32; 40; 42; 73; 208) which make no distinction between the fluid replacement needs of women and men, and suggest that all athletes should consume enough fluid to replace sweat losses (32; 42; 73; 208) or “as much as can be tolerated” (40). Rather, these data suggest that *ad libitum* fluid ingestion is an effective approach since it will likely lessen the risk of overdrinking without any evidence that it increases the risk that medical complications will develop.

Finally, these data may help to explain why women are at a higher risk of developing fluid overload leading to EAH during endurance exercise since they are likely to replace a larger proportion of their sweat losses than are men, and in some, the extent of this replacement may be inappropriately large (50).

Chapter 3

Variations in continuously measured rectal temperatures during a 56 km foot race in cool conditions

Introduction

In 1977, Maron et al. (138) appear to have been the first to measure continuously the rectal temperature of competitors who completed a 42.2 km marathon run in cool conditions (average wet bulb = 13.2 °C, average dry bulb = 18.6 °C). The subjects completed the race in less than 165 minutes, and both athletes lost less than two kilograms (approximately 3% of body weight (BW)) during the race although they ingested fluid at rates of 268 and 588 mL·h⁻¹. Despite cool environmental conditions, relatively low levels of dehydration, and running eight and 16 minutes (5% and 10%, respectively) slower than their personal best times, the athletes finished with rectal temperatures >39 °C. Indeed, the final rectal temperature in one runner was 41.7 °C, amongst the highest values ever reported in runners (193; 256). But, neither athlete either reported symptoms nor exhibited signs of "heat illness."

An unexpected finding was that rates of sweat losses were only 0.7-0.9 L·h⁻¹ which are substantially lower than values measured in other marathon runners racing in hotter conditions (143). It seems unlikely that that these were the maximal sweat rates for these athletes. One possibility is that the need to conserve water by sweating sub-maximally may have been a dominant physiological drive during the exercise, thus allowing a higher rectal temperature than would have been the case at a higher sweat rate.

Humans should be able to prevent any rise in rectal temperature during exercise if they were to sweat more. That they do not indicates that the human body has developed a strategy to reduce fluid losses during exercise by allowing the rectal temperature to rise. This same phenomenon may exist in wild animals: the African hunting dog chooses to maintain a higher rectal temperature during exercise, and therefore a lower evaporative loss (by panting), than does the domestic dog running at the same speed (235). The benefit of this adaptation is that

fluid is conserved so that hunting can be continued for longer before there is a need to drink. Presumably this is a more effective hunting strategy.

A weakness of the study of Maron et al (138) was that the measurement of rectal temperature required coupling each runner's rectal probe to a telethermometer in a car travelling along side the runner. These measurements were performed only every nine minutes so that continuous measurement of the rectal temperature was not possible. However, the introduction of modern data loggers permits essentially continuous monitoring of rectal temperature during exercise with data capture at frequencies of up to 75 times per minute (43; 65). Although such continuous monitoring of core temperature has been reported in long distance swimmers (126), we are not aware of any such published data in ultra-marathon runners. Such continuous monitoring may provide the development of more detailed hypotheses of the likely factors determining the rectal temperature during exercise (35; 177), and promote a better understanding of human thermoregulation during prolonged exercise.

Accordingly, we chose to study rectal temperature responses measured continuously in a larger group of nine athletes during a 56 km ultra-marathon foot race. While this study is primarily descriptive since there is no specific experimental intervention that was investigated, we nevertheless evaluated two hypotheses. According to the current theory that progressive dehydration will cause a progressive rise in the rectal temperature (40; 147), we theorized that progressive dehydration would cause a steady rise in rectal temperature throughout the race, so that the subjects would have markedly higher post-race rectal temperatures compared to pre-race. Alternatively, rectal temperature might be homeostatically regulated so that changes in pace would offset any possible effects of progressive dehydration on the rectal temperature.

Methods

Experimental Design

All procedures were approved by the Research Ethics Committee of the Faculty of Health Sciences at the University of Cape Town. Nine runners entered in the Two Oceans 56 km marathon held on April 10, 2004, in Cape Town, South Africa, were recruited from the local running population for this study. Each runner visited the laboratory once during the days preceding the race to read and complete an informed consent form, and to become familiarized with the data-logging equipment. During the race each runner then carried a data logger on their person and ran with a flexible rectal temperature probe placed in the rectum. Each probe was inserted by the subject to a distance 10-12 cm beyond the anal sphincter. One runner removed the temperature probe during the race. One logger experienced a malfunction that produced a loss of data from approximately 44-53 km of the race. Therefore, full data were available for seven subjects and almost complete data, including pre- and post-race temperatures, for one other.

Rectal Probes

Standard single-use, sterile rectal probes (VHA Plus, Irving, TX, USA) were modified by replacing the output end with a special interface so that they were compatible with the locally-designed and built data loggers.

Data Loggers

Each logger weighs 79 g and has dimensions of 105 x 58 x 20 mm (length x width x height, respectively). The loggers use a 16-bit micro-processor operating at 13 Mhz, and draw 20 milliamps during operation. Each is powered by a single AAA/1.5v battery that can supply enough current for up to 12 hours of logging. Data are stored on a FLASH device similar to those used in digital cameras. This allows for the retention of data even if power is removed during use.

The minimum heart rate (HR) that can be logged is $30 \text{ beats}\cdot\text{min}^{-1}$. The loggers convert beat to beat intervals in milliseconds to heart rate. For these experiments, the transmitter was hard-wired to the data logger to ensure maximal accuracy.

Temperature is measured by supplying the probe with a very accurate reference voltage/current and then measuring the changes with a 20 bit analog to digital converter. This is then converted to a temperature by means of a calibration table, as supplied by the temperature probe manufacturer.

The logger also includes a Motorola MPX4115 air pressure sensor which is sensitive to pressure changes within one meter of vertical height. However, in order for the air pressure sensor to measure altitude accurately in meters above sea level, the sensor requires a more sophisticated calibration. Therefore, the loggers were designed not to return altitude readings, but rather to indicate relative changes in pressure. These relative changes create a profile of the race course, which was used later to place runners at specific points on the course according to the obvious geographical features of the race course.

The software for the micro-processor was compiled initially on a PC and then loaded via a special wire interface to the processor. Data were retrieved by interfacing the logger with a PC via a download cable inserted into the HR transmitter input. Data were stored in a raw binary format that was later delimited in a Microsoft Excel workbook.

Race Day Procedures

On the morning of the race, subjects reported to the laboratory at a predetermined time before the start of the race. At that time their pre-race mass was recorded and they were equipped with a data logger and rectal probe. Each subject inserted the rectal probe himself. After fitting the probe and data logger, the data logger was initialized and the subject reported to the starting line of the race.

Subjects completed the race without intervention(s) from the researchers. Participation in the study did not require that they alter any of their normal racing and running behaviors. If at any time a subject felt that the data logger and probe were interfering with their running, they were entitled to relieve themselves of the apparatus and leave it with persons on the course for later retrieval by the researchers.

Upon completion of the race, the rectal probe was removed and the data logger was retrieved by the researchers. A post-race body mass was recorded to calculate weight losses incurred during the race, and an interview was conducted to estimate the total volume of ingested fluid during the race so that the fluid replacement strategies for each runner could be documented.

Results

Environmental Conditions

Race day was overcast and rainy. The ambient temperature was 13.3 °C at the start (6:00 AM), the relative humidity was 98%, and the wind conditions were calm. At 9:00 AM, the temperature was 13.4 °C, the relative humidity was 99%, and the wind conditions were calm. Finally, at 12:00 PM the temperature was 15.1 °C, the relative humidity was 70%, and the wind conditions were calm. The maximum temperature on that day was 16.1 °C.

Subject characteristics, pre- to post-race changes in weight, estimated fluid consumption and estimated sweat losses, and race times are presented in Table 1. The mean weight loss for all subjects was 1.6 ± 0.4 kg ($2.1 \pm 0.6\%$). Estimated rates of fluid ingestion were 579 ± 247 mL·h⁻¹, and estimated sweat losses were 848 ± 277 mL·h⁻¹. The mean race time for all subjects was 356 ± 39 minutes.

Table 1. Selected subject characteristics in eight athletes studied during the 2004 Two Oceans 56 km Ultra-marathon.

Variable	Mean \pm SD	Range
Age (years)	47.1 ± 6.2	39 - 56
Race Time (min)	356 ± 39	296 - 403
Pre-race Weight (kg)	74.5 ± 7.3	60.8 - 83.9
Post-race Weight (kg)	$72.9 \pm 7.4^*$	59.3 - 82.6
Δ Weight (kg)	1.6 ± 0.4	0.8 - 2.1
% Change in weight	2.1 ± 0.6	1.0 - 2.7
Fluid Ingestion (mL·h ⁻¹)	579 ± 247	243 - 1011
Sweat rate (mL·h ⁻¹)	848 ± 277	576 - 1335
Pre-race T _{re} (°C)	37.3 ± 0.5	36.4 - 37.7
Post-race T _{re} (°C)	$38.7 \pm 0.5^*$	37.9 - 39.3

* Significantly different than Pre-race (p < 0.05)

Temperature and other data

Figure 1 shows mean \pm SD data for running speed (top line) and rectal temperatures (middle line) for the complete group at different distances during the race, as well as the profile of the course (bottom line). Rectal temperatures remained just below 39 °C from 13 km to 56 km, despite a significant reduction in running speed likely produced in part by "fatigue" and the

more demanding nature of the course from 28-56 km including two long hills from approximately 24-33 km and 39-46 km (bottom line, Figure 1).

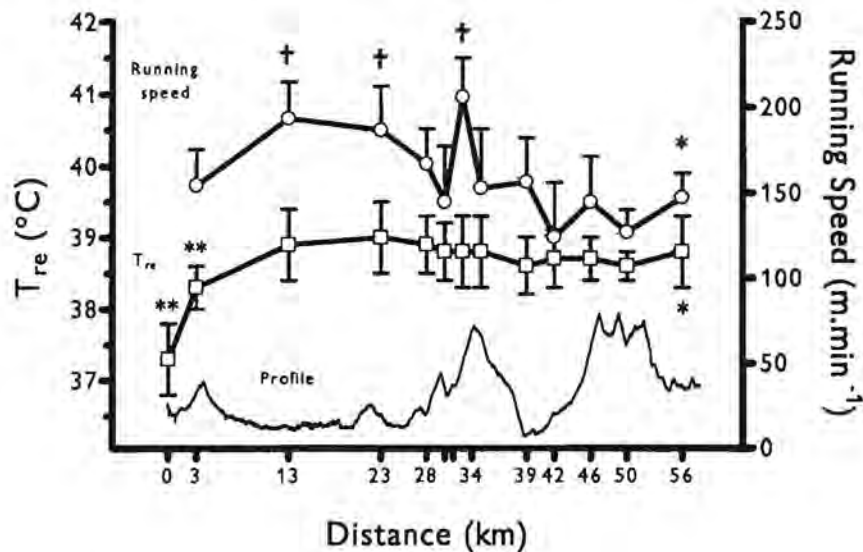


Figure 1. Mean \pm SD running speeds (top line) and T_{re} (middle line) from selected points on the race course. The bottom line represents the course profile. $N = 8$ at all points except 46 and 50 km, where $N = 7$. Note that 30-32 km represents a steep downhill section between two hills on the course.

- * Significant time effect ($p < 0.01$)
- ** Significantly different from rectal temperatures at all other distances ($p < 0.05$)
- † Significantly different from running speeds at all other distances ($p < 0.05$)

Using a Dunnet's test for post-hoc comparisons, the rectal temperatures from 13 km to 56 km were not different from each other, suggesting that the rectal temperature rose steadily for approximately 13 km before reaching approximately 39 °C, and then did not change for the remainder of the race (Figure 1, middle line).

Subjects achieved the fastest sustained running speeds ($194 \pm 21 \text{ m}\cdot\text{min}^{-1}$) between 3-13 km on the course, which is a slightly downhill and flat section (Figure 1, bottom line). These running speeds were significantly different ($p < 0.05$, Dunnet's post-hoc test) from all other sections of the course except 13-23 km, which is another flat section, and 30-32 km. Running

speeds were significantly higher only between 30-32 km, which is a steep downhill section between two hills.

Figure 2 shows individual data for rectal temperature and heart rate in eight athletes for whom complete data for rectal temperature are available. Subject 5 wore his own HR monitor and therefore did not log HR data on the data logger. A number of different patterns for the rectal temperature response during prolonged exercise were observed.

With regard to the rectal temperature data, Subject 9 maintained his rectal temperature at an essentially constant level after the first 50 minutes. This was a unique pattern. Subjects 2 and 7 also maintained their rectal temperatures within a narrow range during the race, but some variation occurred, most obviously in subject 7. Subjects 3, 4 and 8 reached their highest temperatures early in the race, approximately within the first 75 minutes of exercise, but these decreased in the middle portion of the race with a terminal rise in subjects 2-5, 7, and 8. In contrast, subject 5 showed a progressive rise in rectal temperature for the first 300 minutes, followed by a steep drop for 50 minutes during which he walked/jogged for approximately 4-5 km, before resuming running the final 5 km to the finish.

Heart rates oscillated throughout exercise and tended to mirror changes in running speed (Figure 2). Several subjects reported stopping momentarily during the race for less than one minute at a time. However, in general minute to minute oscillations in HR were far larger than changes in rectal temperature over the same time. It seems unlikely that these minute-to-minute changes in HR and rectal temperature are due to equivalent changes in running speeds and metabolic rates which were likely relatively constant, at least for the first 23-28 km of the race during which the course is relatively flat (Figure 1).

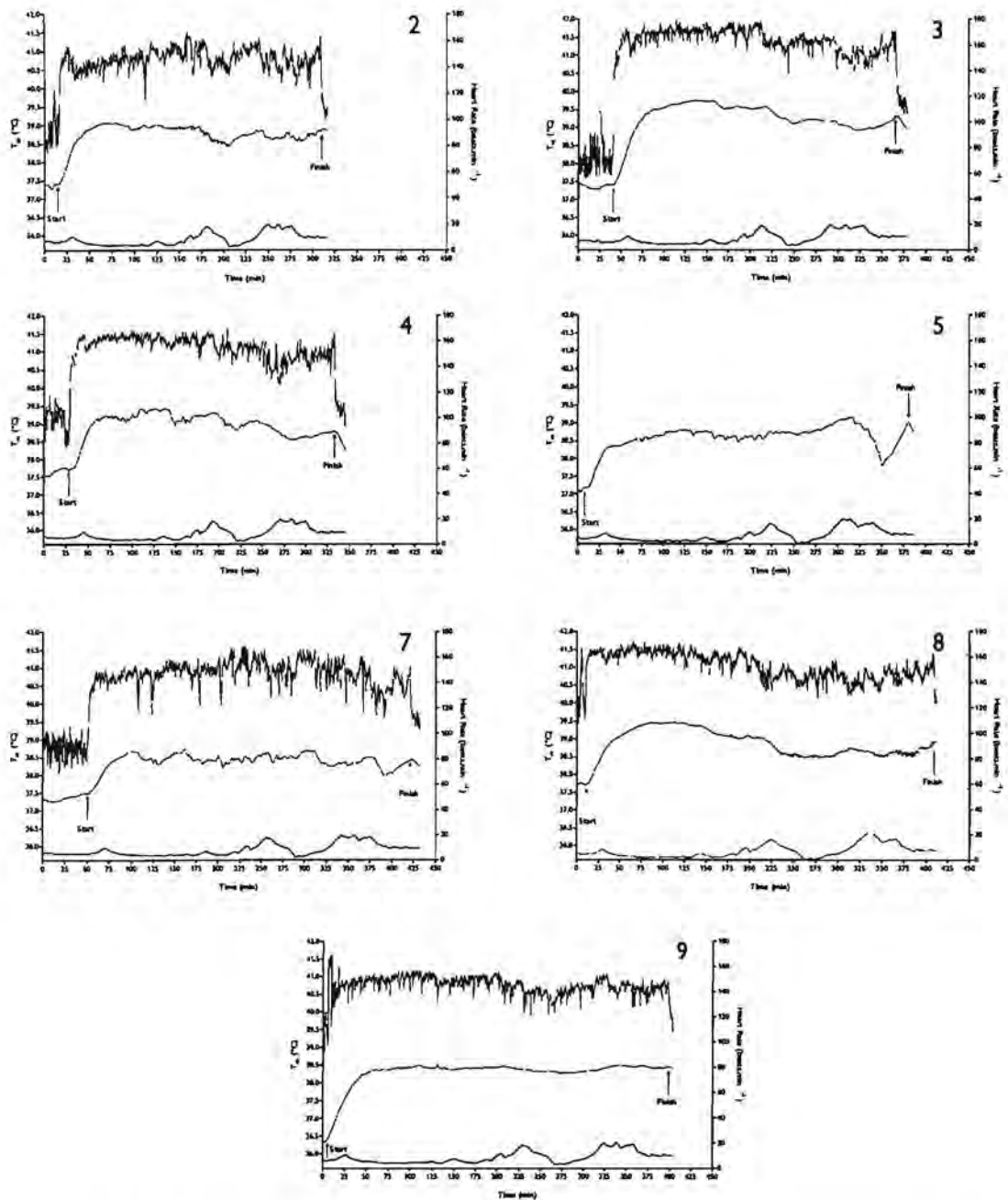


Figure 2. Continuously-measured heart rates (top line) and T_{re} (middle line) in seven subjects running in the 2004 Two Oceans 56 km Ultra-Marathon (course profile shown as bottom line). Note that the course profile was generated individually by each logger, and therefore indicates at which point on the course the athlete was running when the (continuously-recorded) physiological measurement was logged.

Figure 3 shows a plot of running speeds and rectal temperatures for the two halves of the race. Data are plotted for running speed and T_{re} at 3, 13, 23, and 28 km (first half of the race, Figure 3a) and for 30, 32, 34, 39, 42, 46, 50, and 56 km (second half of the race, Figure 3b).

Wide scatter indicates that factors other than running speed clearly influenced the T_{re} in these runners. There was a strong relationship between running speed and rectal temperature during the first half of the race ($r^2 = 0.38$, $p < 0.05$), although this relationship was less profound during the second half of the race ($r^2 = 0.07$, $p < 0.05$) (Figure 3a and 3b).

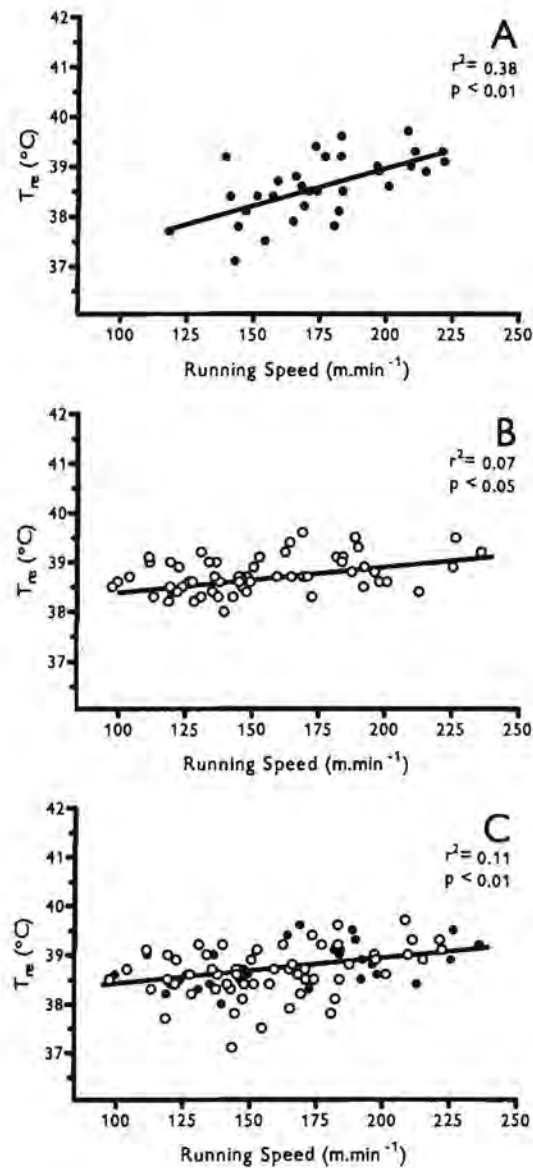


Figure 3. Relationship between T_{re} and running speed during the first half of the race (A), the second half of the race (B), and throughout the entire race (C). Running speeds were calculated from split times between geographical points on the course which could be identified by each data logger's course profile. These points were at 0, 3, 13, 23, 28, 30, 32, 34, 39, 42.2, 46, 50, and 56 km.

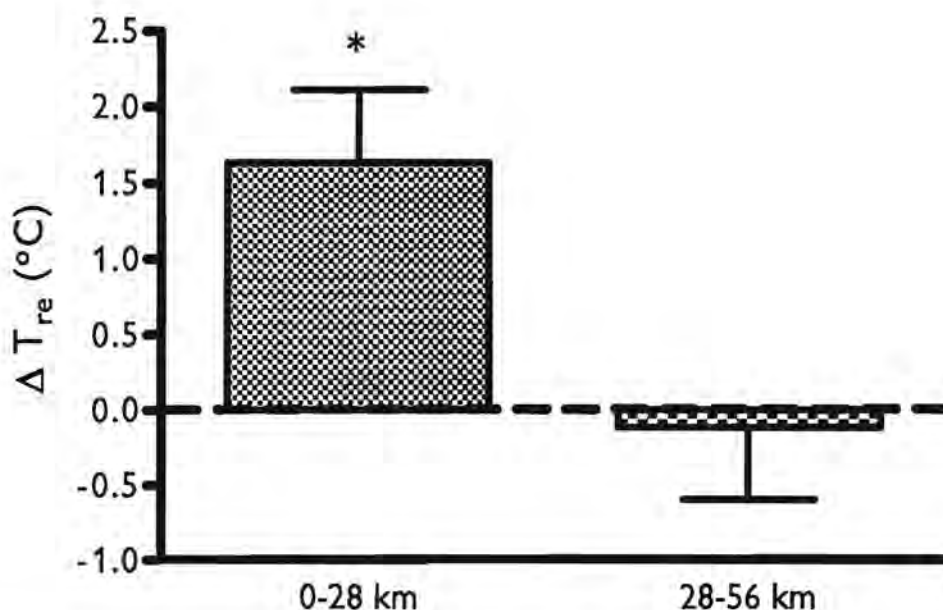


Figure 4. Mean changes in rectal temperature from 0-28 km and from 28-56 km. These data suggest that the largest changes in rectal temperature occur in the early stages of the race, and not in the latter stages.

* Significantly different from 28-56 km $p < 0.01$

The majority of the changes in T_{re} occurred during the first half (28 km) of the race (Figure 2 and Figure 4). The mean change in T_{re} from 0-28 km was 1.63 ± 0.48 °C compared to a mean change in T_{re} of -0.21 ± 0.49 °C from 23-56 km (Figure 4) ($p < 0.01$).

Discussion

Laboratory studies of the effects of fluid ingestion on the rectal temperature response to two hours of exercise (11; 72; 147; 211) have shown that the rectal temperature rises rapidly for the first hour of exercise before rising more slowly during the following hour. Fluid ingestion has been shown to reduce the rate of rise in rectal temperature during the second hour during constant workload exercise (19; 72; 147), although this effect is abolished when appropriate amounts of convective cooling are provided (33; 202). However, even with adequate cooling in those experiments the rectal temperatures seldom rose above 39.5 °C, suggesting that 39.5 °C is the approximate peak temperature that is reached during two hours of labora-

tory exercise and in those specific environmental conditions in which convective cooling is inadequate. Indeed the presence of adequate convective cooling in the study of Saunders et al. (202) produced terminal rectal temperatures of approximately 38.6 °C after two hours of laboratory exercise in the heat.

Another form of exercise in which the rectal temperature has been monitored continuously has been during exercise at a fixed workload to volitional fatigue in uncompensable heat load (84; 154; 185; 207). In these experiments, core temperature rises until exercise is terminated voluntarily by the subject. Termination usually occurs when core temperatures reach and exceed 40 °C. Exercise is terminated purportedly as a result of central fatigue and a decrease in the amplitude of efferent neural signals to the muscle (84; 185; 241). Thus the published evidence has given rise to the idea that the rectal temperature rises progressively during prolonged, fixed-workload exercise especially when there is also an uncompensated weight loss, part of which may be due to dehydration (110).

Maron et al. (138) showed elements of these patterns in the two marathon runners that were studied. The rate of rise in the rectal temperature was fastest in the first 45 minutes of exercise, a finding which has been repeatedly reported (147; 159), but continued to increase thereafter, marginally in one athlete (Subject 2) but more steeply in the other, who showed a sudden and sharp secondary increase in rectal temperature after 110 minutes of running and 20-25 minutes before he completed the race.

In contrast, the contribution of the current study, in which rectal temperatures were measured continuously and for a much longer duration than previously, is to show that when humans use a self-selected pacing strategy, their rectal temperatures appear to be regulated suc-

cessfully within a quite narrow range throughout the exercise bout. In addition, rectal temperature appears not to increase progressively and inexorably as might otherwise have been believed on the basis of early laboratory studies (37; 72; 147), but instead rises during the first approximately 60 minutes of exercise before varying regularly during the exercise, perhaps related to metabolic rate and running speed (Figure 3). Laboratory studies in which the exercise workload is fixed remove a crucial method by which the body can regulate its thermal homeostasis, namely the capacity to modify the number of motor units that are active in the exercising limbs and which therefore determine the athlete's pace. Thus our study suggests that when allowed to select their own pace during a competitive ultra-marathon race, athletes appear to choose metabolic rates that maintain their rectal temperature within a two degree (37.5-39.5) range (Figures 1 and 2).

During the first half of this race the relationship between rectal temperature and running speed (metabolic rate) was relatively strong ($r^2 = 0.38$, $p < 0.05$), but this relationship was less in the second half of the race. This strong relationship in the first half was likely due to the flat nature of the course. The metabolic rate while running on a flat course will be influenced more by running speed because higher metabolic rates are required to produce faster running speeds. However, on a hilly course this relationship between running speed and metabolic rate will weaken since while running uphill the metabolic rate can remain high while running speed is likely to decrease.

In the current study, all the runners except one showed a gradual increase in rectal temperature for approximately 100 minutes (Figure 2). The one exception was the runner who maintained his rectal temperature at approximately 38.5 °C for the entire race regardless of any changes in the terrain. Peak rectal temperatures were reached within the initial 100 minutes,

with only one runner achieving his peak temperature after running 46 km. These data suggest that metabolic rate is the strongest predictor of rectal temperature in these subjects, and that when running in conditions such as these (~ 14 °C), thermoregulation is not impaired by dehydration since these subjects appear to have lower rectal temperatures during the second half of the race compared to the first half of the race, although they were likely to be in a progressively more negative fluid balance as the race progressed.

The mean change in running speed from the first quarter of the race to the last quarter of the race was approximately $52 \text{ m}\cdot\text{min}^{-1}$ in the five runners who reached their peak temperature in the first 100 minutes. The runner who maintained his rectal temperature at 38.5 °C decreased his running speed by $32 \text{ m}\cdot\text{min}^{-1}$, a difference of approximately 38%, and clearly ran at a more even pace compared to the other runners. This might explain why this runner's rectal temperature was maintained at a more constant level compared to the other runners in this study.

This study fails to show the secondary progressive rise in rectal temperature that others have shown previously (19; 72; 110; 147). In addition, the data from the current study indicate a high degree of individual variability in the rectal temperature response to self-paced endurance exercise (Figure 2). This variability lies both in the magnitude of the change in rectal temperature from pre-race to the peak, and also in the extent to which the rectal temperature varies during the race. In addition, the peak and final rectal temperatures reached by these runners were not excessively elevated although they reported modest fluid replacement and despite weight losses approximately 2%.

Even though the ambient conditions were mild during the race (overcast, calm and wet, ~ 14 °C), nearly all the runners in this study achieved peak rectal temperatures above 39 °C. While this was an unexpected finding by itself, these data taken together with those of other studies that have measured rectal temperatures during running races (93; 138; 191; 193) suggest that endurance trained individuals have a large capacity to regulate their body temperature during this type of activity despite the environmental conditions.

For example, in Maron et al. (138) one runner also reached a peak rectal temperature of approximately 39 °C, while the other's peak rectal temperature was approximately 41 °C. Furthermore, upon reaching his peak of approximately 41 °C, that runner maintained that temperature for some 40 minutes without any further increases or without the development of symptoms of "heat illness." In the study of Myhre et al. (93) the two subjects whose rectal temperatures were measured during the marathon exhibited similar patterns to those observed in the current study—namely, rectal temperatures rose to approximately 39 °C and remained within a narrow range for the duration of the race. All these data therefore suggest that humans can regulate their temperature effectively during endurance exercise over a wide range of ambient conditions and changing hydration status.

The two subjects in the study of Maron et al (138) both ran faster than $15 \text{ km}\cdot\text{h}^{-1}$ for the entire race, while in the current study the fastest runner averaged $11.2 \text{ km}\cdot\text{h}^{-1}$ over 56 km. Despite this difference in running speed and the difference in ambient conditions, the subjects from both of these studies reached similar rectal temperatures, suggesting that there may be some biological benefit to maintaining the core temperature within this range during endurance exercise, regardless of the environmental conditions. Pugh et al. (193) reported that the post-race rectal temperatures of 47 runners completing a warm weather (22 °C, 55% RH)

marathon ranged between 39-41 °C. This finding has been repeated in other studies (2; 137; 138; 143; 151; 159; 193; 256) (Chapter 1, Table 1). Furthermore, although the post-race rectal temperatures reported in Chapter 1 (Table 1) represent a 2-3 °C (5.5-8.1%) increase in rectal temperature above resting values, these levels are still well below the commonly used value of 42 °C that is used to diagnose exertional heat stroke. These data suggest that runners completing a standard marathon footrace even in warm environmental conditions carry a relatively small risk of elevating their rectal temperatures to 41 °C or higher, even when they are dehydrated.

The above studies, taken together with data from the current study, provide evidence that during endurance exercise the human body successfully regulates core temperature within a given range, regardless of the environmental conditions and the hydration status. There is evidence that this regulation is also anticipatory (132; 241) so that the pace is regulated specifically to ensure that a dangerous rise in rectal temperature does not occur (241), and that an individual achieves the highest temperature only at the end of the task. Therefore, throughout the race both conscious and (predominantly) subconscious decisions are made to modify the pacing strategy and hence the metabolic rate so that core temperature is kept within the desired range, although sometimes reaching relatively high peaks above 40 °C in individuals who benefit from especially high heat tolerance (138), although such high temperatures seem to be reached only in the final stages of the exercise bout, during the "end spurt" (123; 176)

Even though the runners in this study were non-competitive and completed the race at a mean running speed of 158 m.min⁻¹ in cool conditions, they still reached peak rectal tempera-

tures of above 39 °C. This was in spite of the ambient conditions providing a large gradient for heat loss. Although the rectal temperatures of faster runners were not measured, there was only a weak relationship between running speed and rectal temperatures in the runners in this study over the entire course of the race. While the relationship was enhanced during the first half of the race (Figure 3a), the relationship weakened during the second half of the race. This suggests that regardless of running speed and ambient conditions, humans have an excellent ability to regulate core temperature even during more prolonged exercise when they lose up to 2% of their body weight. Furthermore, these findings suggest that during exercise a rectal temperature of approximately 38.5-39.5 °C is in some way beneficial or favorable for the body.

Conclusion

In self-paced exercise of a very prolonged nature (>4 h) undertaken in cool and humid conditions, rectal temperature appears to be regulated between set points with some individual variability, and does not increase inexorably. Environmental conditions are likely to play an important role in temperature regulation but may have less influence on rectal temperature than expected, especially when the mode of exercise is self-paced. The runners in this study completed the race at relatively low running speeds, yet they maintained their rectal temperatures within a two degree range above resting values. This suggests that there is some biological advantage gained by maintaining rectal temperature in this range during prolonged exercise.

There appears to be a highly variable core temperature response across individuals. However, this variable response might be a result of varying metabolic rates between athletes. More research is needed in which core temperatures are measured when work rates, both

absolute and relative, are controlled during prolonged exercise and can be compared to self-paced exercise of a similar duration and intensity.

Chapter 4

**Rectal temperature responses to a one-day
109 km cycle race in moderate ambient
conditions**

Introduction

Fluid replacement during exercise has been studied previously in runners (44; 99; 124; 159; 197; 200) and the evidence shows that these athletes replace approximately 50% of their weight losses, and finish recreational races and laboratory trials without any ill effects of their fluid losses. Normally, fluid replacement strategies follow *ad libitum* practices, although different sets of guidelines advocate replacing 100% of weight losses (32; 40).

The method of fluid delivery in cycling differs from that in running since cyclists carry water bottles and fluid bladders, in which up to five Liters of fluid can be stored. In contrast, runners receive fluid in small cups or sachets at various points along the route. This provides the cyclists with relatively constant access to fluids. However, despite the greater availability of fluids during cycling, athletes in both sports appear to replace similar percentages of their weight losses during the course of their respective events (51). The reasons for this are unknown, but provide some support for the hypothesis that the thirst mechanism is perhaps more accurate than is currently believed (40; 85; 208).

The rectal temperature responses to cycling in actual competition are not widely reported, although some investigators have examined rectal temperatures during self-paced cycling in the laboratory (21; 109; 111; 133; 234; 241). On the other hand, there have been numerous studies that have reported the rectal temperature responses to fixed-duration and fixed-workload exercise in both hot (37; 41; 46; 47; 93; 146-148; 152; 202) and moderate conditions (25; 141; 234) in the laboratory. Although the ambient conditions and wind speeds in these studies are different from those encountered in actual exercise, some of these studies (41; 147) appear to form a part of the basis of the model that is currently used to explain the effects of dehydration during endurance exercise. This model predicts that the rectal tempera-

ture during exercise rises in direct proportion to the level of dehydration, although much earlier researchers have shown instead that the rectal temperature during exercise rises in direct proportion to the metabolic rate (165; 168; 198; 201). According to current researchers, the rise in rectal temperature is said in part to be a result of a reduced skin blood flow, which reduces the heat conductance from the core to the skin, causing an imminent rise in rectal temperature that is proportional to the amount of dehydration (80; 147; 206). Yet early investigators before 1970 demonstrated that the metabolic rate is the best predictor of the rectal temperature during exercise (165; 168; 198; 201), and that the rectal temperature during exercise is independent of the ambient temperature up to approximately 30 °C.

The primary aim of this study, therefore, was to describe the rectal temperature responses to prolonged endurance cycling (>3 h) when athletes ingest fluid *ad libitum* and complete self-paced exercise during an actual race in moderate ambient conditions. In addition, we wished to shed light on whether the rectal temperature is regulated within safe limits during exercise even when fluid replacement would likely be much less than 100% of weight losses, or if instead the rectal temperatures would rise progressively as dehydration increased as predicted by the current model (41; 147; 256).

Methods

Seven cyclists (N = 6, male, N = 1, female) were recruited from the local cycling population for this study. All were entered in a timed local cycle race (2005 Cape Argus/Pick 'n' Pay Cycle Tour) over 109 km. Subject characteristics are reported in Table 1. All subjects except one male aimed to achieve the best possible time they could in the race. In addition, four of the subjects started in the top three groups (~5%) of the field.

Data logging equipment

Each logger weighs 79 g and has dimensions of 105 × 58 × 20 mm (length × width × height, respectively). The loggers use a 16-bit micro-processor operating at 13 Mhz, and draw 20 milliamps during operation. Each is powered by a single AAA/1.5v battery that can supply enough current for up to 12 hours of logging. Data are stored on a FLASH device similar to those used in digital cameras. This allows for the retention of data even if power is removed during use.

The minimum heart rate (HR) that can be logged is 30 beats·min⁻¹. The loggers convert beat to beat intervals in milliseconds to heart rate. For these experiments, the transmitter was hard-wired to the data logger to ensure maximal accuracy, although a sufficient clear signal was not always obtained in all subjects.

Temperature is measured by supplying the probe with a very accurate reference voltage/current and then measuring the changes with a 20 bit analogue to digital converter. This is then converted to a temperature by means of a calibration table, as supplied by the temperature probe manufacturer (VHA Plus, Irving, Texas, USA).

The logger also includes a Motorola MPX4115 air pressure sensor which is sensitive to pressure changes within one meter of vertical height. However, in order for the air pressure sensor to measure altitude accurately in meters above sea level, the sensor requires a more sophisticated calibration. Therefore, the loggers were designed not to return altitude readings, but rather to indicate relative changes in pressure. These relative changes create a profile of the race course, which was used later to place cyclists at specific points on the course according to the obvious geographical features of the race course.

The software for the micro-processor was compiled initially on a PC and then loaded via a special wire interface to the processor. Data were retrieved by interfacing the logger with a PC via a download cable inserted into the HR transmitter input. Data were stored in a raw binary format that was later delimited in a Microsoft Excel workbook.

Pre-race procedures

In the days prior to the race, subjects were familiarized with the data logging equipment and informed of all the required experimental procedures. All subjects read and completed a written informed consent form, and all research protocols were approved by the University of Cape Town Research Ethics Committee and conformed to the Declaration of Helsinki. Subjects were encouraged to remove the probe and data logger if at any time they felt it was creating a performance decrement.

On the morning of the race, subjects inserted a standard rectal probe to a distance 10-12 cm beyond the anal sphincter, and fitted themselves with the HR transmitter. They then reported to a designated research area at the start of the race, where they were weighed on a scale accurate to 100 g with their shoes and helmet off and their pockets emptied, and after voiding their bladders. After the body weight measurement, each data logger was initialized by the researchers, and the rectal probe and HR transmitter were plugged in to the logger. Subjects were then instructed to complete the race as they normally would without any intervention(s) from the researchers.

Upon finishing the race, subjects reported directly to the research area at the finish. This required cycling slowly for approximately 200 m before passing through a finish area to collect a

finishing medal, and then walking/cycling a further 200-300 m to the research area. Upon arriving in the research area subjects were asked to void their bladders and were then weighed in the same manner as before the race and on the same scale. An interview was then conducted to determine how many bottles of fluid and the amount of food each subject had consumed during the race. The volume of fluid remaining in each subject's fluid bottles was measured to calculate the amount of fluid ingested during the race. Subjects were then permitted to remove the rectal probe.

Data were retrieved from the data loggers with custom software written specifically for this task. Data were downloaded in text format and then delimited in Microsoft Excel before being reduced and analyzed.

Weight losses during the race were calculated by subtracting the post-race weight from the pre-race weight. This value was used to calculate the sweat losses, which were corrected for food and fluid consumption and for urine production by asking the subjects to void their bladders after the race before the post-race weight measurement.

Statistical Analysis

A repeated measures ANOVA was used to analyze the rectal temperature responses during the race, and the pre to post changes in T_{re} and weight. A Wilcoxon Matched Pairs test for non-parametric data was used to analyze changes in T_{re} from 0-16 km and 16-109 km.

Results

All values are Mean \pm SD. Subject characteristics and other data are reported in Table 1. Post-race weight was significantly lower than pre-race weight (74.9 ± 10 vs. 73.2 ± 9.8 kg, pre vs. post, respectively) ($p < 0.01$) (Table 1). Subjects lost 1.6 ± 0.4 kg ($2.2 \pm 0.5\%$ of their pre-

race weight), and replaced $47.3 \pm 19.1\%$ of their total estimated sweat losses by ingesting $483 \pm 260 \text{ mL}\cdot\text{h}^{-1}$.

Table 1. Subject characteristics (N = 7) and pre- and post-race variables

	Mean \pm SD	Range
Age (years)	29.1 \pm 7.5	20 – 43
Height (m)	1.79 \pm 0.09	1.68 – 1.91
BSA (m ²)	1.91 \pm 0.19	1.66 – 2.24
Race Time (min)	202 \pm 32	175 – 271
Pre-race Weight (kg)	72.9 \pm 11.9	57.9 – 94.4
Post-race Weight (kg)	71.3 \pm 11.8*	56.1 – 92.2
Δ Weight (kg)	1.6 \pm 0.4	1.2 – 2.2
% Change in weight	2.2 \pm 0.5	1.6 – 3.1
Sweat Rate (mL·h ⁻¹)	848 \pm 277	576 – 1335
Sweat Rate (mL·m ⁻² ·h ⁻¹)	495 \pm 97	365 – 671
Pre-race T _{re} (°C)	37.40 \pm 0.47	36.65 – 37.98
Post-race T _{re} (°C)	38.80 \pm 0.34*	38.4 – 39.3
Pre to post Δ T _{re} (°C)	1.4 \pm 0.28	1.09 – 1.81

*Significantly different from pre-race value ($p < 0.01$).

The environmental conditions on the day of the event were moderate and windy. At 0600 the ambient temperature was 23.9 °C and relative humidity (RH) was 47%. At 0900 the ambient temperature was 23.8 °C and the RH 50%. Finally, at 1200 the ambient temperature was 27.6 °C and the RH was 42%. The prevailing wind was east-southeast at approximately 40-60 km·h⁻¹, depending on the exact location, and was a head or cross wind for approximately the first 45 km of the race.

The mean T_{re} responses at different points during the race in all subjects are shown in Figure 1. There was a significant time effect during the race ($p < 0.01$), and the mean T_{re} was significantly higher ($p < 0.01$) than the pre-race value from four km onwards (Figure 1). Mean T_{re} rose continuously for approximately 16 km before levelling off and remaining within a narrow range of < 0.2 °C for the remainder of the race (Figures 1-3). Post-race T_{re} was significantly higher compared to pre-race T_{re} by 1.40 ± 0.28 °C (37.40 ± 0.47 vs. 38.80 ± 0.34 °C, pre vs. post- T_{re} , respectively; $p < 0.01$) (Table 1).

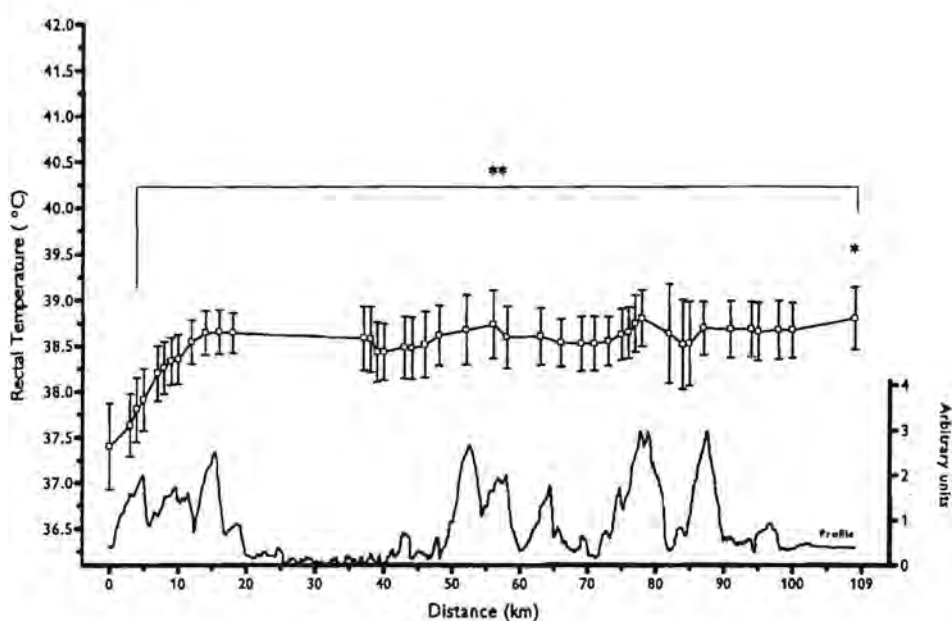


Figure 1. Mean rectal temperature response (top line) in all subjects (N = 7). Bottom line represents the course profile as produced by the air pressure sensor in the data loggers.

* Time effect ($p < 0.01$)

** Significantly different from pre-race T_{re} ($p < 0.01$)

A number of different individual responses were observed amongst the subjects (Figure 2). Subjects 3 and 8 maintained their rectal temperatures during the race within the narrowest range. Subject 7 reached his peak T_{re} after less than 2.5 hours, after which his T_{re} declined steadily for nearly an hour before remaining within a narrow range for the remainder of the

race. This subject (7) experienced a large decrease (~ 1 °C) in T_{re} after five hours during a five km downhill section of the race course, during which the race was stopped twice for medical evacuations of other cyclists. Upon reaching the bottom of the hill, his T_{re} rose steadily until he reached the finish. Subject 5 experienced a sharp decrease in T_{re} when he crashed in the lead group after approximately 40 km. After remounting his bike he experienced a gradual rise in T_{re} up to approximately 39.5 °C, after which time his temperature oscillated within a 0.75 °C range until the finish. Subjects 1, 2, and 4 exhibited various oscillations in rectal temperature throughout the race that appear to be the result of the undulating characteristic of the race course (Figure 2).

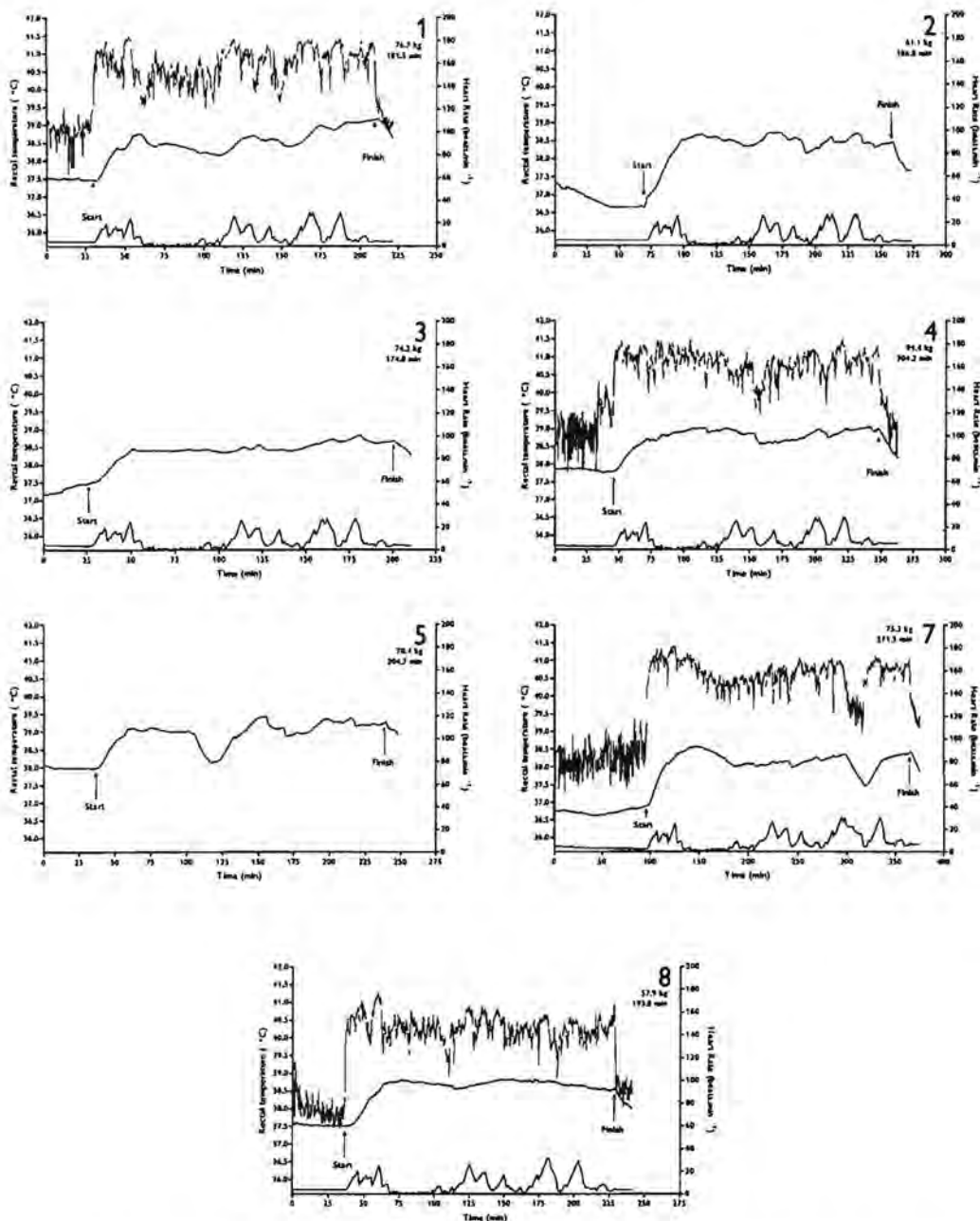


Figure 2. Individual rectal temperature responses in all subjects (N = 7). Values in the upper right corner denote subject number, pre-race weight and race time. T_{re} is plotted on the left y-axis and HR is plotted on the right y-axis. The data represent continuous temperature recordings before, during and after the event. Subject 7 was the only subject who was cycling only for completion of the race course. All other subjects were cycling for the best possible result as evidenced by a race times of <3.25 h.

A significant difference was found between the change in T_{re} during the first 16 km of the race and the change in T_{re} during the remaining 93 km of the race. Rectal temperature changed by

1.26 ± 0.34 °C during the first 16 km, compared to a change of 0.15 ± 0.20 °C from 16 – 109 km ($p < 0.02$) (Figure 3).

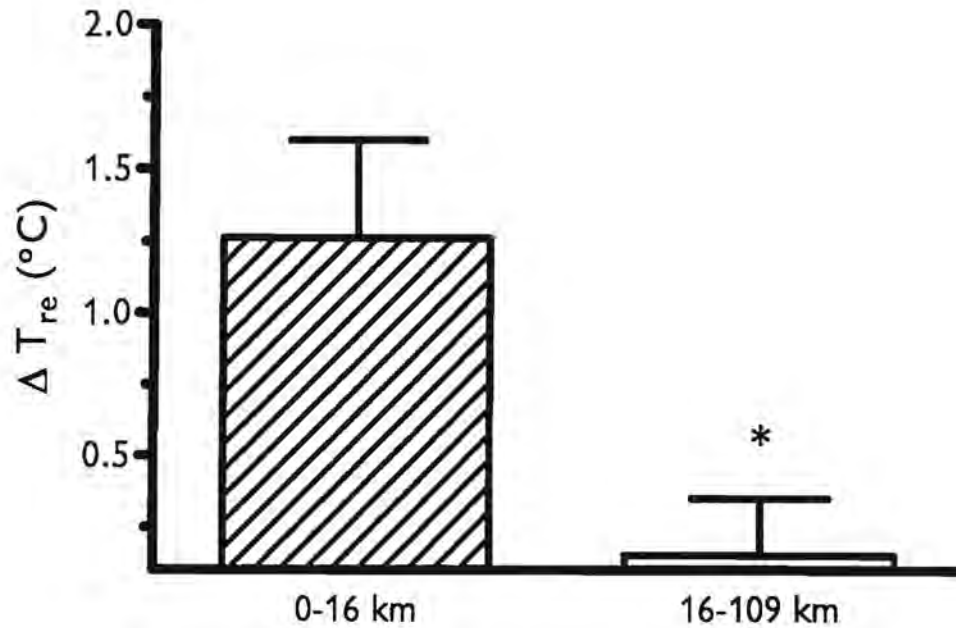


Figure 3. Changes in rectal temperature from 0-16 km and from 16-109 km. These data suggest that, as in Chapter 3, the majority of the changes in rectal temperature occur in the first 45-90 minutes during endurance races, as opposed to the latter stages of the race

* Significantly different from 0-16 km ($p < 0.01$)

A significant relationship was found between the pre-race mass and the estimated sweat rate ($r^2 = 0.92$, $p < 0.01$) (Figure 4a). No relationships were found between the post-race T_{re} and race time ($r^2 = 0.10$, $p = 0.49$) (Figure 4b) or between the post-race T_{re} and the change in weight ($r^2 = 0.10$, $p = 0.50$) (Figure 4c).

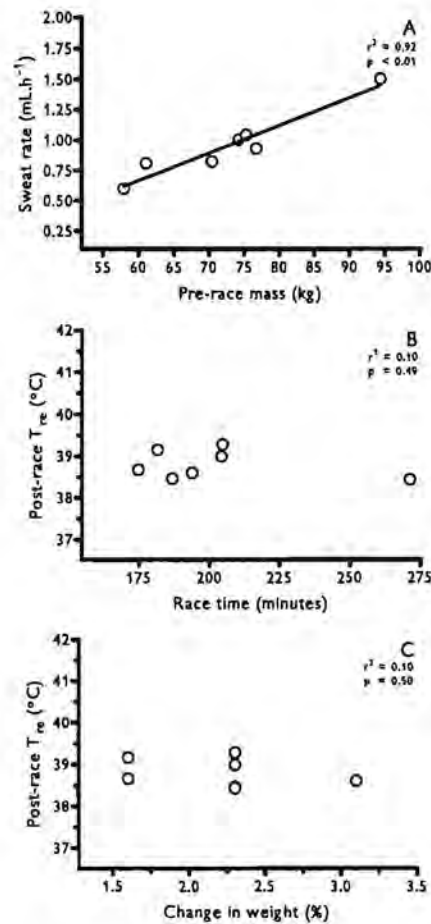


Figure 4. The relationship between pre-race mass and the estimated sweating rate during the race (4a), and post-race T_{re} vs. race time (4b) and the percentage change in weight (4b).

During the 10 minute period immediately following the race, mean HR decreased from 170 ± 3 beats·min⁻¹ to 104 ± 12 beats·min⁻¹, ($p < 0.01$) (Figure 5a) and mean T_{re} decreased from 38.78 ± 0.34 immediate post-race to 38.33 ± 0.44 °C (mean difference of 0.52 ± 0.14 °C; $p < 0.01$, immediately post-race vs. 10 min post-race) (Figure 5b). The rectal temperature was significantly lower from three minutes of recovery onwards compared to the immediate post-race value ($p < 0.01$). The mean rate of decrease during this 10 minute period was -0.05 ± 0.01 °C per minute.

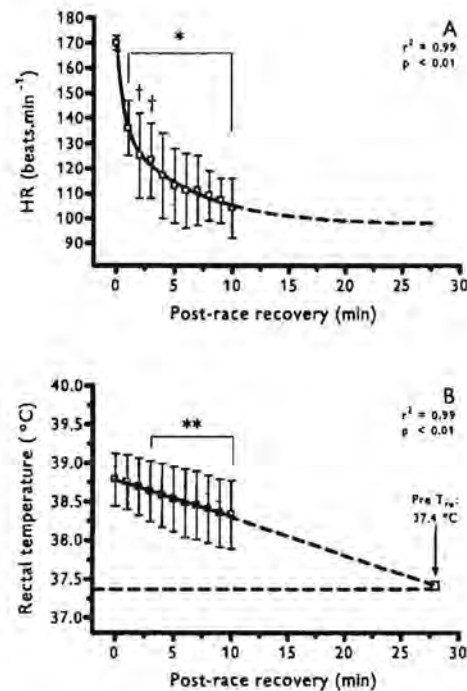


Figure 5. Rates of decline in T_{re} (A) ($N = 7$) and heart rate (B) ($N = 4$) during the first 10 minutes of post-race recovery. No specific interventions were performed during this recovery period, except that the subjects progressed to the research area and were weighed and interviewed.

- * Significantly different from zero minutes recovery
- ** Significantly different from 0-2 minutes of recovery
- † Significantly different from 4-10 minutes recovery

Discussion

The aim of this study was to investigate the rectal temperature responses to prolonged cycling during a one-day 109 km cycle tour which was completed in moderate conditions. The rectal temperatures and heart rates were recorded throughout the race with portable data loggers carried by the cyclists. Together with the course profile generated by the altitude sensor in the logging units, this allowed us to examine and compare the rectal temperatures of the subjects at specific points on the race course. To our knowledge this is the first study of its kind to report the temperature responses to prolonged endurance cycling during an actual event in any conditions. The presence of the rectal probe from start to finish also al-

lowed us to examine more closely the post-race rectal temperature response during the first 10 minutes of recovery.

The main finding of this study is that the rectal temperature rose progressively for approximately the first 45 minutes of exercise before being maintained within a very narrow range, in this case 0.15 °C, for the duration of the race. This response is similar to earlier investigators who also showed that during exercise the rectal temperature climbs steadily for approximately 60 minutes before reaching a higher level at which it is then regulated for the duration of the exercise bout (12; 168; 198; 201). This effect was present in both cool (5 °C) and warm (30 °C) environments (165; 168; 201). Although the metabolic rates of the subjects in the current study most likely varied with the undulating nature of the race course and the nature of cycling in a *peleton*, the rectal temperatures still remained within a very narrow range (<0.2 °C) from 16 km onwards.

We have also examined the rectal temperature responses to ultra-marathon running (Chapter 3). The current data are different from those observed during ultra-marathon running only in that the peak rectal temperatures were higher during ultra-marathon running (39.27 ± 0.36 vs. 38.80 ± 0.34 °C, running vs. cycling, respectively), and oscillated within a wider range after the initial rise over ~60-90 min (0.40 vs. 0.15 °C, running vs. cycling, respectively).

The volume of fluid ingested and the resulting weight losses in these athletes were similar to those measured in a large cohort of recreational cyclists completing the same event two years earlier (51). The cyclists ($N = 120$) in that study ingested 406 ± 160 mL·h⁻¹ and lost 1.9% of their pre-race weight, compared to 483 ± 260 mL·h⁻¹ and $2.2 \pm 0.5\%$ in the current study.

Therefore the ingested fluid volume and the consequent weight losses in this local population of cyclists is a repeatable finding. Interestingly, the ultra-marathon runners in Dugas and Noakes (52) also lost similar amounts of weight ($2.1 \pm 0.6\%$), as did runners completing 32 km and 42.2 km races from various other studies (3.2 – 6.1%) (131; 136; 138; 159; 193; 256) although the environmental conditions and type of subjects varied widely amongst all these studies.

The weight changes and ingested fluid volumes from other studies and this one (51; 52) all occur when the athletes have frequent access to fluids during the event. For example, the ultra-marathon runners reported in Dugas and Noakes (52) had access to aid stations on average every 1.6 km, and at every one km from 40–56 km. Many of the cyclists in Dugas and Noakes (51) carried fluid bladder systems on their backs in addition to fluid bottles, providing access to as much as five Liters of fluid during the race. Yet even with such frequent access to fluids, and even in very different ambient conditions (14 °C vs. 24 °C, running vs. cycling, respectively), these subjects ingested fluid at similar rates and therefore finished their respective races with similar and apparently safe amounts of weight losses, as evidenced by their symptom-free presentation at the end of the race. Together these data suggest that even when provided with nearly unlimited access to fluids, athletes subjectively choose to replace approximately 50% of their fluid losses, a finding that has been described previously (10; 34; 90; 171; 231; 232). This is likely the result of the homeostatically-regulated system in the human body in which other components of the system—such as the serum sodium concentrations—are defended at the cost of small changes in body weight and small fluid losses.

Of particular interest in the current study is the one female subject (Subject 8 in Figure 2), who ingested only 150 mL of water during the first 25 km, and nothing thereafter. Her pre to

post-race change in weight was -1.8 kg (3.1%). Although she consumed a negligible volume of fluid during the race, her absolute change in T_{re} was the lowest (1.09 °C) and her post-race decline in T_{re} was similar to the other subjects (-0.05 °C per minute). These findings occurred despite her completing the race in a competitive time of 193.8 minutes, placing her third in her age group and 32nd among all the women finishers. Her positions among her sex and age groups indicate that she completed the race at a relatively high intensity, which corresponds to her pre-race goals to finish the race in as fast a time as possible. Yet immediately upon finishing the race her T_{re} was only 38.6 °C, and 10 minutes later it had declined rapidly by 0.48 °C to 38.12 °C.

This contrasts with current models of dehydration and temperature regulation that state that T_{re} increases with progressive dehydration (9; 40; 42; 147). This did not happen in this athlete or apparently in any of the other athletes in this study. Instead, her T_{re} rose continuously for approximately 60 minutes and then remained within a very narrow range of 0.3 °C for the duration of the race, rising not above any of the other subjects even though her weight losses, and therefore level of dehydration, were higher. This suggests that there may be some biological advantage to maintaining an elevated core temperature during endurance exercise, or that the core temperature is regulated at a higher level during exercise as has been described previously (165; 168; 198) and that even with progressive dehydration the body regulates core temperature successfully in ambient temperatures <30 °C.

A similar pattern was observed amongst the other five athletes who were aiming to complete the race in as fast a time as possible. During the first 16 km the rectal temperature response was similar among subjects 1-5, rising during this section of the course which covered several

short (< 2 km) hills before oscillating within a narrow range for the remaining 93 km, in spite of many more climbs and descents of varying distances (1-5 km) (Figures 1 and 2). This point of the race course was chosen for this comparison because it allowed us to place the athletes at a specific point based on the geographical profile of the course. The next point at which this would have been possible would have been between 40-50 km, at which point the T_{re} 's are similar to the values at 16 km.

The next important finding was the significant relationship between the pre-race mass and the total volume of estimated sweat losses. This was a strong relationship ($r^2 = 0.92$, $p < 0.01$) even though there were only seven data points. This finding is similar to that in Dugas and Noakes (51), in which the pre-race mass accounted for approximately 53% ($r^2 = 0.53$, $p < 0.01$) of the variance in the total sweat volume. These findings show why universal fluid replacement guidelines (32; 40) are flawed and show that those athletes who weigh less require smaller absolute volumes of fluid during endurance exercise. Therefore, any guidelines for fluid replacement that prescribe hourly volumes are problematic and should rather be sex- and/or mass-specific so that athletes ingest volumes of fluid that are appropriate for their metabolic rates and rates of fluid loss during exercise, although drinking to thirst appears to be the best strategy since it will always prevent the consumption of too much or too little fluid in an individual regardless of his or her body mass.

The last finding of this study was that immediately following the race finish, rectal temperatures decreased rapidly by -0.05 °C·min⁻¹ for the first 10 min even though the subjects remained in the outside ambient conditions (23.8 °C and 50% RH), and no specific interventions were utilized to promote this rapid decline in T_{re} . When analyzed with a linear regression ($r^2 = 0.99$; $p < 0.01$) (Figure 5b), the post-race rectal temperatures would have likely reached

resting values in <30 minutes even if this rate of decline is not linear. This finding mirrors that of Millard-Stafford et al. (143) and others (165; 168). Millard-Stafford et al. (143) reported decreases in T_{re} of approximately $-2.6\text{ }^{\circ}\text{C}$ and $-2.3\text{ }^{\circ}\text{C}$ after 30 minutes of passive recovery immediately following two 40 km runs in warm ($32\text{ }^{\circ}\text{C}$) and humid ($\sim 60\%$) conditions (143). The change over 30 minutes in Millard-Stafford et al. was $-0.08\text{ }^{\circ}\text{C}$ per minute, but what is most remarkable is that this rate of decline in rectal temperature occurred when the subjects were 4.5% ($\Delta T_{re} = -2.3\text{ }^{\circ}\text{C}$) and 4.4% ($\Delta T_{re} = -2.6\text{ }^{\circ}\text{C}$) dehydrated immediately following the runs, and in ambient temperatures of $>30\text{ }^{\circ}\text{C}$ and $>50\%$ relative humidity (143). During the recovery period subjects were permitted to ingest fluid *ad libitum*, and based on their changes in weight from post-run to the end of the recovery period it appears that they ingested only 280-350 mL of fluid (143). Based on known rates of gastric emptying and absorption, and also on their relatively high levels of weight loss, this small volume of fluid likely had no direct effect on any circulatory changes, and therefore heat loss capabilities, during this recovery period.

In the current study subjects ingested no fluid during the initial 10 minutes post-race, and they completed the race $2.2 \pm 0.5\%$ dehydrated. Yet the rate of change in rectal temperature is similar to Millard-Stafford et al. (143), falling at a rate of $-0.05\text{ }^{\circ}\text{C}$ per minute compared to $-0.08\text{ }^{\circ}\text{C}$ per minute. The linear regression (Figure 5b; $r^2 = 0.99$) shows that after <30 minutes their rectal temperatures would have likely returned to the pre-race values of $37.40 \pm 0.47\text{ }^{\circ}\text{C}$.

The post-race data from the current study and from Millard-Stafford (143) therefore suggest that in apparently healthy and trained athletes the post-race decline in rectal temperature ap-

appears to occur independently of hydration status. Even with weight losses of up to 4.5% (143), rectal temperatures decrease rapidly following exercise cessation (-0.05 - 0.08 °C.min⁻¹) and return to pre-race values in approximately 30 minutes. This rapid reduction in rectal temperature occurs even when the athletes are not removed from the exercising environment, as shown in this study in Figure 5 and elsewhere (143), and is likely the result purely of exercise termination and the sudden decrease in heat production that accompanies this. These data therefore question the logic of using intravenous fluid to treat "heat injury" in hyperthermic and/or collapsed athletes after competitive exercise. Rather, the data from this study and another (143) suggest that athletes who complete endurance exercise dehydrated by up to 4.5% nevertheless return their rectal temperatures rapidly to normal without the need for any medical intervention. The treatment of "heat injury" and dehydration with saline infusion should be more closely examined to determine if it is a necessary and efficacious intervention. These data and others (143) suggest that the simple act of stopping exercise, and hence heat production, is all that is required in otherwise healthy yet dehydrated athletes.

In conclusion, these data show that *ad libitum* fluid ingestion is sufficient to maintain the rectal temperature below critical levels during self-paced endurance cycling in moderate ambient conditions. Furthermore, even in healthy yet dehydrated (2-3%) athletes following prolonged (>3 h) exercise, no fluid or medical intervention is necessary to restore rectal temperature to pre-race values. These data therefore support the premise, first promoted by others (67; 168; 201) but subsequently overlooked, that humans can successfully regulate their temperature during exercise by apparently resetting the desired core temperature and altering the metabolic rate to maintain this new set point (67; 168; 201).

The regulation of rectal temperature before and after exercise appears to be part of a complex regulation (241), the goal of which is to ensure thermal homeostasis regardless of the environmental conditions or the changing nature of the terrain over which the activity is completed. Under these conditions, athletes are able to regulate their body temperatures well below the heatstroke range ($> 41.5\text{ }^{\circ}\text{C}$) even when drinking fluid only *ad libitum* and losing up to 4% of their body weight (143). It seems improbable that higher rates of fluid ingestion would not have altered this response as they did not in our other laboratory-based studies (202). Finally, it appears that in otherwise healthy athletes *ad libitum* fluid ingestion represents the best practice for fluid replacement during self-paced endurance exercise, for it will always prevent an individual from drinking too little or too much fluid.

Chapter 5

Rates of fluid ingestion affect cycling performance in hot and humid conditions with appropriate convective cooling

Introduction

Current guidelines for fluid ingestion during exercise state that athletes should drink “as much as tolerable” in order to ensure that “all the weight lost during exercise” is replaced (40; 42). It is argued that only by replacing all the weight lost during exercise, thereby completing exercise in a mildly over-hydrated state, will athletes optimize their performance and minimize the risk of heat injury (11; 17; 32; 42; 43; 53; 56; 73).

These recommendations appear to be based mainly on the results of two studies from the 1970's (41; 72) and a more recent study published in 1992 (147). All three found that the rectal temperatures of subjects who ingested fluids at the highest rate, usually approximately $600\text{-}1200\text{ mL}\cdot\text{h}^{-1}$ during exercise, were significantly lower than when they ingested no fluid during exercise. The mechanism for this effect is unclear since the rate of sweating, which is the predominant avenue for heat loss in the hot and humid conditions in which these experiments were conducted, was not different when the subjects either did or did not ingest fluid during exercise.

One explanation is that cardiac output may be reduced with increasing levels of dehydration, although this effect is apparent only when exercising in the upright condition (80; 83; 84; 147), and that this causes a reduced skin blood flow which impairs heat conduction from the core to the skin. However, no record exists of any athlete developing heat stroke, “heat illness” or “heat injury” in any of these trials, even when they did not ingest any fluid, and differences in final rectal temperatures were small ($<0.75\text{ }^{\circ}\text{C}$) when subjects drank either nothing or $1.2\text{ L}\cdot\text{h}^{-1}$ during exercise. Furthermore, post-exercise rectal temperatures in these studies tend to be in the range of $38.5\text{ - }39.0\text{ }^{\circ}\text{C}$ regardless of whether or not the subjects ingested fluid during exercise (41; 147). These values are much below the rectal temperatures measured in pa-

tients with heatstroke (usually > 41.5 °C). Therefore these studies do not provide evidence to support the belief that fluid ingestion is necessary to prevent heat illness during constant workload exercise even in hot and humid environmental conditions. Rather they suggest that exercise of such intensity and duration can be performed in relative safety while ingesting either nothing or relatively small volumes of fluid.

There are three other important limitations inherent in the design of these foundational studies (41; 72; 147; 184). First, all were performed at a fixed power output for a fixed duration that was set before exercise on the basis of each subject's individual maximal aerobic capacity. Therefore none of these studies evaluated the effects of different rates of fluid ingestion on exercise performance. Yet the nature of competitive sport is that the athlete paces him or herself, continually altering the power output in order to achieve a specific goal, which might be different for each athlete. Marino et al. (133) and Kay et al. (109) have recently shown that the performances of athletes were not different whether they ingested either nothing or replaced 100% of their weight losses during 60 minutes of self-paced exercise in the heat. However the durations studied by these authors (60 min) were less than those (120 min) evaluated in the three original studies.

Second, the three original studies (41; 72; 147) were conducted in relatively harsh environmental conditions with little or no convective cooling (≤ 2.5 m·s⁻¹), which can have a marked effect on temperature regulation (3; 33). The facing wind speeds and the manner in which they were generated with single fans did not produce the equivalent volumes and rates of air flow that athletes encounter during out-of-doors competitions at high absolute and relative metabolic rates. During competition out-of-doors, athletes usually encounter facing wind speeds in the range of 12-20 km·h⁻¹ when running or 25-40 km·h⁻¹ when cycling. Even higher

facing wind speeds and therefore rates of convection would be produced by head or cross winds.

Others have demonstrated that rates of whole body heat storage are increased during exercise in laboratory conditions in which avenues for convective heat loss are low because of low or absent facing wind speeds (3; 202). None of the subjects in the study of Saunders et al. (202) was able to complete 120 minutes of cycling exercise at a fixed work rate (60% of peak power) in hot, humid, wind still conditions. But all completed the protocol when the facing wind speed approximated their predicted velocity for cycling out-of-doors (202). Under conditions of convective cooling that approximated those present in out-of-doors exercise, the rectal temperature response was the same whether subjects replaced 60% or 80% of the total weight they lost during the 120 minute exercise bout.

Third, these original studies failed to control for different rates of carbohydrate (CHO) ingestion during exercise. Thus any measured differences might have resulted from the different rates of CHO ingestion consequent to different rates of fluid, and hence CHO, ingestion.

Accordingly, in this study we aimed to determine if different rates of fluid ingestion during exercise produced dose-dependant effects on performance and thermoregulatory responses, in particular rectal temperature (T_{re}), sweat rates, and rates of heat storage (Q_s) when trained cyclists performed i) self-paced exercise in hot and humid environmental conditions in which ii) the potential for convective heat loss was optimized and equivalent to conditions out-of-doors, and in which iii) the amount of CHO ingested was the same in all experimental conditions.

Our hypothesis was that exercise performance would be optimized at a particular rate of fluid ingestion above which little or no further improvement would occur despite increasing rates of fluid ingestion. We also hypothesized that higher rates of fluid ingestion would be associated with proportional reductions in Q_s during exercise and that this would, in part, explain the measured changes in exercise performance.

Methods

All research protocols were approved by the Research Ethics Committee of the Faculty of Health Science of the University of Cape Town. The research and its protocols complied with requirements of the most recent publication of the Declaration of Helsinki.

Subjects

Six highly-trained males were recruited from the local cycling population. All research protocols and requirements were explained in full to each subject before he read and signed an informed consent form. Each subject was made aware that he could withdraw from the study at any time without prejudice or penalty.

Experimental Design

This study was completed in a randomized and counter-balanced fashion. Initially, each subject completed a ramp protocol on an air-braked cycle ergometer (Kingcycle, Buckinghamshire, UK) for the determination of his peak power output (PPO). The protocol started at approximately $3.3 \text{ W}\cdot\text{kg}^{-1}$ and increased $20 \text{ W}\cdot\text{min}^{-1}$ until volitional fatigue or until the subject could not maintain the required power output. Anthropometrical measurements (weight; height; sum of seven skinfolds) were taken and recorded during this visit.

Two to five days after the PPO test, each subject completed a familiarization trial (Ad Lib) in which he was permitted to consume water ad libitum but in which CHO ingestion was held constant (see below). The purpose of this trial was to introduce the testing protocols to the subjects and to calculate the estimated sweat losses so that the replacement volumes for the other five trials could be determined. Each subject then completed one experimental trial every 4-7 days thereafter to maximize recovery and to minimize any effect of heat acclimatization.

All trials were completed during the winter months. Environmental conditions remained constant at 33 °C and 50% RH for each trial. The wind speed was set to correspond to each subject's calculated road speed on the cycle ergometer (35-40 km·h⁻¹) during the first five minutes of the time trial, but was not adjusted subsequently. CHO ingestion remained constant across all trials as subjects ingested a 45 g energy bar (32.96 g CHO, 1.59 g protein, 0.21 g Fat) at the start of each trial and after completing 40 km so that the total CHO ingestion for each trial was 65.92 g.

The volume of ingested water was manipulated to produce five different experimental conditions: 0, 33, 66, 100, and WET, in which subjects ingested 0%, 33%, 66%, or 100%, respectively, of their estimated sweat losses as calculated from the initial trial (Ad Lib). In the WET trial, subjects rinsed their mouths with the same volume of water that was consumed in the 66 experimental condition, but were not permitted to ingest any fluid and instead expelled the ingested volume of water into a graduated cylinder after they had rinsed their mouths.

Experimental Procedures

All experimental trials were performed in the University of Cape Town Environmental Chamber on each subject's own cycle mounted on the King Cycle rig. Subjects received no feedback about power output, heart rate, time, and current distance during the trial. They received verbal notice only after every 10 km of cycling that they had completed.

All trials started in the early morning (05h30-07h30) after an overnight fast to control for feeding status and diurnal variations in body temperature. Subjects were asked to adhere to their normal mixed-diets and consume a similar meal the night before each trial. All trials were completed in the same standard cycling kit. Upon arrival, each subject's cycle was calibrated on the King Cycle rig. Subjects then voided their bladder and recorded three consecutive nude body weights (BW) on an electronic scale accurate to 100 g. Each subject then inserted a rectal probe (YSI 409AC, Yellow Springs, OH, USA) to a position approximately 10-12 cm beyond the anal sphincter.

Skin temperature (T_{skin}) was measured with four skin thermistors (YSI 427, Yellow Springs, OH, USA) placed on the mid-calf, mid-thigh, chest (midway between the axillary fold and the nipple) and sub-scapular region, immediately below the inferior angle of the scapula (145). Once the thermistors and rectal probe were connected to the digital telethermometer, subjects remained seated and at rest until all skin and rectal temperatures had equilibrated and baseline values had been recorded. Equilibration at each of the measured sites was defined as three identical consecutive one-minute readings at that site.

Subjects then entered the chamber, mounted their cycle and began pedaling slowly to overcome the initial resistance on the fly wheel. During the next 60 s, all wires and equipment were assembled for the time trial, and the subject was provided with a count down at the end

of which the time trial began. During the trials subjects were asked to report their rating of perceived exertion (RPE), thermal comfort (66) and thirst at 10 km intervals. The thirst rating scale was a modified Borg category-ratio scale of 0 to 10. Heart rate (HR) was recorded every two minutes with a Polar HR monitor (Polar Electro, Finland).

The fluid replacement volume for each condition except the Ad Lib, 0, WET and 33 conditions was divided into a 400 mL bolus which was consumed at the start of the trial. The remaining volume was divided into seven equal amounts which were ingested at 10 km intervals after the recording of perceptual scores. The fluid ingested during the 33 trial was divided into eight equal volumes and administered at the same intervals as during the other trials. During the Ad Lib trial subjects had continuous access to 500 mL bottles filled with water. The water to be ingested was kept inside the environmental chamber before and during each trial since, when cycling in out-of-doors conditions, the ingested fluid is normally kept at ambient conditions.

Power output, T_{re} and T_{skin} were recorded throughout the trail at one-minute intervals. Environmental conditions were monitored constantly and recorded every five minutes. Split times were recorded at 10 km intervals. On completion of each 80 km time trial, subjects remained in the chamber for 1-4 minutes for the measurement of the peak rectal temperature. Upon exiting the Environmental Chamber, skin thermistors were quickly removed. Subjects then voided their bladders, towed dry, and again recorded three consecutive nude body weights.

Changes in Body Weight

Changes in body weight (ΔBW) were used to estimate sweat losses during the trials. Sweat losses were corrected for urine production and fluid ingestion with the equation

$$\text{Pre-BW (kg)} + \text{Ingested Fluid Vol (L)} - \text{Urine Volume (L)} - \text{Post-BW (kg)}$$

Sweat rate in Liters per hour was then calculated by dividing the value from the above equation by performance time in hours.

Percent dehydration was calculated as

$$[(\text{Pre BW kg} - \text{Post BW}) / \text{Pre BW}] * 100$$

Temperature and Heat Content

The values obtained from the four skin thermistors were weighted and used to calculate mean skin temperature with the equation (196)

$$T_{\text{skin}} = 0.3 * (T_{\text{chest}} + T_{\text{back}}) + 0.2 * (T_{\text{thigh}} + T_{\text{calf}})$$

Mean T_{skin} was then used to calculate mean body temperature (T_b) with the equation

$$T_b = 0.79 * (T_{\text{re}}) + 0.21(T_{\text{skin}})$$

Heat Content (Q_c) was calculated according to the equation of Nielsen (164)

$$Q = T_b * BW * 3.47$$

Where T_b is in degrees Celsius, body weight is the pre-BW in kg, and 3.47 is the specific heat value of body tissue in $\text{kJ}\cdot\text{C}^{-1}\cdot\text{kg}^{-1}$.

Heat Storage (Q_s) was calculated as the change in Q_c between two time points with the equation

$$Q_s = T_2 Q_c - T_1 Q_c$$

where T_1 and T_2 are given time points during a trial.

Statistical Analysis

Data were analyzed using Statistica 6.0 (Statsoft Inc.). Two separate analyses were performed. Initially, a repeated measures ANOVA was used to analyze the means of the six different trial conditions. Since a small sample size increased the risk of Type II error, we combined the WET, 0, and 33 conditions into one group (LO) and the Ad Lib, 66, and 100 conditions into one group (HI). In the HI and LO group analysis, a one-way ANOVA was used to detect differences between the groups in non-continuous variables, while a repeated measures ANOVA was used where appropriate. Statistical significance was accepted when $p < 0.05$. When the overall F value of the model was significant, a Tukey's post-hoc test was performed to detect specific differences.

Results

Data for the six different experimental interventions

All values are mean \pm SD. Descriptive data are presented in Table 1. Table 2 lists the mean performance times and performance-related variables during the six different time trials.

Table 1. Subject characteristics (N = 6).

	Mean \pm SD	Range
Age (years)	23.0 \pm 3.7	20 – 29
Height (m)	1.78 \pm 0.07	1.66 – 1.85
Mass (kg)	68.6 \pm 8.1	61.2 – 82.3
BSA (m ²)	1.85 \pm 0.13	1.79 – 2.06
PPO (Watts)	426 \pm 39	383 – 501
Watts·kg ⁻¹	6.3 \pm 1.0	5.1 – 7.8

Table 2. Temperature, power output, and performance time in all subjects (N = 6) and all experimental conditions. No statistically significant interactions were found between conditions.

	Pre T _{re} (°C)	Post T _{re} (°C)	Mean Power (W)	% PPO	Performance time (min)
0	36.8 \pm 0.2	39.2 \pm 0.4*	196 \pm 25	46 \pm 3	128.3 \pm 6.3
WET	36.8 \pm 0.2	39.3 \pm 0.9*	194 \pm 26	45 \pm 5	129.4 \pm 8.1
33	36.8 \pm 0.1	39.2 \pm 0.5*	190 \pm 20	45 \pm 4	129.9 \pm 6.1
Ad Lib	36.9 \pm 0.3	39.1 \pm 0.4*	214 \pm 26	50 \pm 3	124.2 \pm 5.8
66	36.7 \pm 0.2	39.2 \pm 0.5*	205 \pm 18	48 \pm 5	126.1 \pm 4.8
100	36.9 \pm 0.2	38.9 \pm 0.4*	207 \pm 25	49 \pm 7	125.4 \pm 5.8

* Significantly different from pre T_{re} (Time effect, p < 0.01)

Fluid and weights

The volumes of ingested fluid for each trial were significantly different from each other ($p < 0.01$, Figure 1c). The percentage replacement of sweat losses (Figure 1a) was significantly different between trials ($p < 0.01$). During the 100 trial, subjects replaced $94.6 \pm 10.8\%$ of their weight losses. This value was significantly greater than in all other trials ($p < 0.01$). Percentage fluid replacement in 66 was $59.9 \pm 6.4\%$ and was significantly different from 0 (0%), 33 ($32.4 \pm 2.9\%$), 100, and WET (0%) ($p < 0.01$), but was not different from Ad Lib ($50.7 \pm 13.1\%$) (Figure 1a).

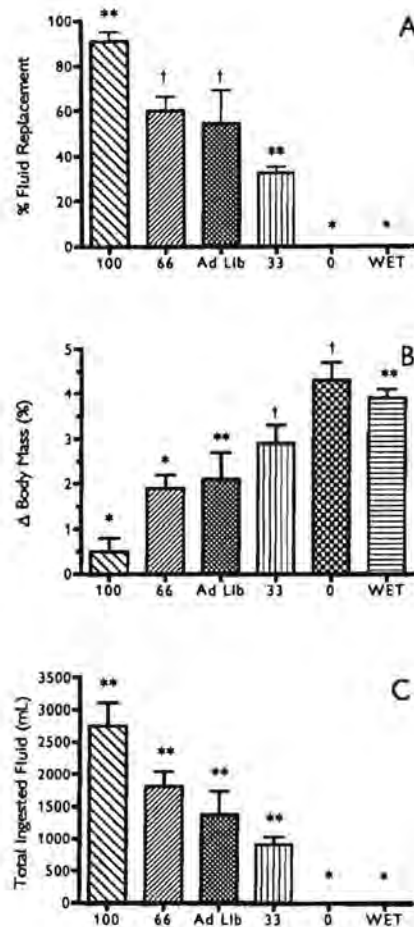


Figure 1. Percent fluid replacement (1a), pre to post percent changes in weight (1b) and total volume of ingested fluid (1c).

* Significantly different from 33, Ad Lib, 66, and 100 ($p < 0.01$)

** Significantly different from all other conditions ($p < 0.01$)

† Significantly different from 0, WET, 33, 100 ($p < 0.01$)

Mean changes in body weight (%) are also presented in Figure 1b. The fluid interventions produced significantly different levels of weight loss (Figure 1b) between all trials except Ad Lib vs. 66 ($2.1 \pm 0.6\%$ vs. $1.9 \pm 0.3\%$) and 0 vs. WET ($4.3 \pm 0.4\%$ vs. $3.9 \pm 0.2\%$). Post-trial urine volumes ranged from 34 ± 0.32 mL in WET to 170 ± 134 mL in 100, but were not graded in the same manner as percent weight loss and were not different between the experimental conditions (Ad Lib: 37 ± 43 mL, 0: 99 ± 110 mL, 33: 69 ± 43 mL, 66: 70 ± 79 mL, 100: 170 ± 135 mL, WET: 34 ± 33 mL).

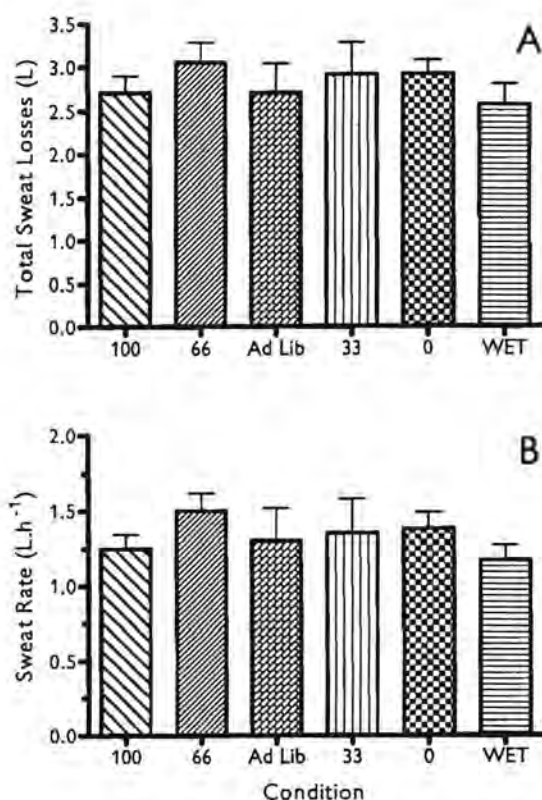


Figure 2. Total sweat losses (2a) and sweat rates (2b) in all subjects. Total sweat losses were calculated from changes in body mass during exercise corrected for fluid intake and urine production.

Although the subjects ingested significantly different volumes of fluid during each experimental condition, there were no differences in sweat rates or total sweat losses (Figures 2a and 2b).

Mean sweat rates for Ad Lib, 0, 33, 66, 100, and WET were 1.33 ± 0.22 , 1.34 ± 0.11 , 1.31 ± 0.17 , 1.44 ± 0.14 , 1.41 ± 0.20 , and $1.24 \pm 0.16 \text{ L}\cdot\text{h}^{-1}$, (NS), respectively (Figure 2b).

Temperatures and Q

Pre-exercise T_{re} and post-exercise T_{re} are listed in Table 2. No significant differences were observed in these variables between any of the experimental conditions. Peak T_{re} 's were always reached at the end of each time trial, and were similar both between conditions (Table 2) and within subjects (Table 3).

Pre- and post-exercise heat content (Q_c), heat storage (Q_s) and rate of Q_s are presented in Figure 3. No differences were found in any of these variables between any of the conditions. However, there was a significant time effect ($p < 0.01$) in Q_c in all conditions so that the subjects' post Q_c values were significantly higher compared to their pre Q_c values (Figure 3a).

Table 3. Intra-subject variation in post- T_{re} measurements.

	Subject					
	1	2	3	4	5	6
0	39.7	39.4	38.9	38.8	39.4	38.8
WET	40.6	39.3	39.0	38.8	40.0	38.1
33	40.0	38.8	38.8	38.9	39.6	38.9
Ad Lib	39.8	38.8	38.9	39.1	39.3	38.7
66	40.0	39.2	39.0	38.6	39.5	38.9
100	39.1	38.9	38.4	38.7	39.6	38.7
Mean (°C)	39.9	39.1	38.8	38.8	39.6	38.7
SD (°C)	0.5	0.3	0.2	0.2	0.2	0.3
CV (%)	1.2	0.7	0.6	0.4	0.6	0.8

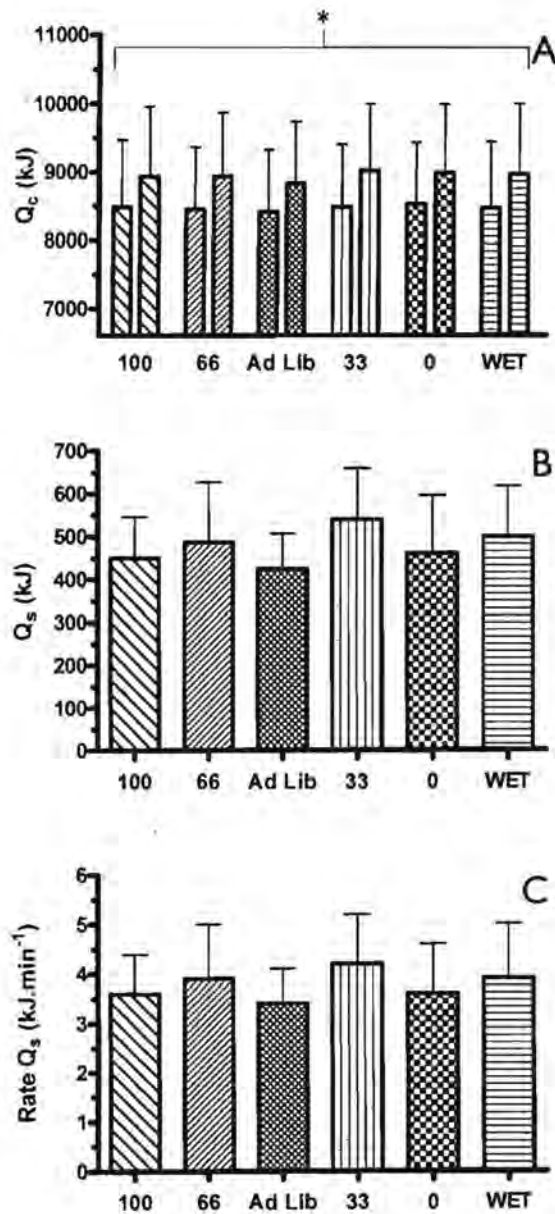


Figure 3. Pre- and post-exercise heat content (Q_c) (3a), total heat storage (Q_s) (3b), and the rate of Q_s (3c) in all subjects and conditions.

* Significant time effect ($p < 0.01$)

Perceptual Ratings

No group by time effect was found for the ratings of perceived exertion (RPE) ($p = 0.99$) (Table 4) or the thermal comfort scores ($p = 0.91$) (Table 5), although there was a significant time effect in all conditions in both of these variables so that the ratings increased significantly over the distance of each trial.

There was a significant time effect ($p < 0.01$) and a significant group by time interaction ($p < 0.01$) in the thirst ratings (Figure 4) suggesting that the subjects responded differently to each fluid replacement intervention. Although no differences were found between the conditions at 80 km or at any other specific interval, several significant time effects were found within each condition. The mouth rinsing in the WET trial delayed the elevation of thirst ratings by 10 km so that in the 0 trial the thirst ratings were significantly different from pre-values at 40 km in the WET trial compared to 30 km in the 0 trial (Figure 4). Similarly, the ingested fluid volume in the 33 trial delayed a significant elevation of thirst until 60 km (Figure 4). The ingested fluid in the Ad Lib, 66 and 100 trials maintained low levels of thirst throughout the entire 80 km distance and were not different from each other (Figure 4).

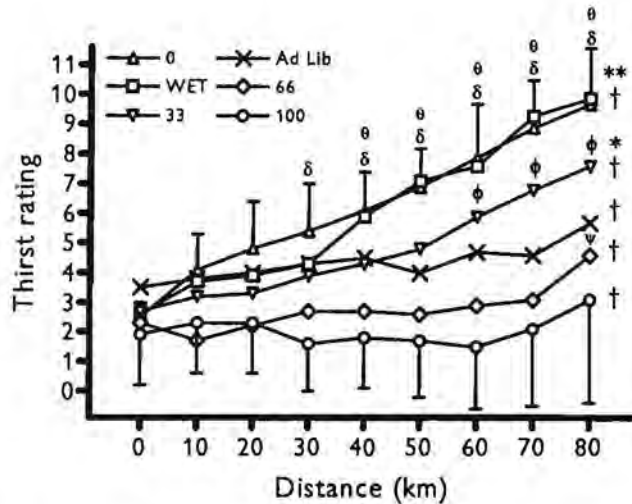


Figure 4. Ratings of thirst in all conditions. A significant time effect and a significant group by time effect were found, although no significant differences were detected between conditions at any specific points.

- * Significantly different from 100 (Group by time effect, $p < 0.01$)
- ** Significantly different from 66 and 100 (Group by time effect, $p < 0.01$)
- † Significant time effect (Time effect, $p < 0.01$)
- δ Significantly different from 0 km in the 0 trial (Time effect, $p < 0.03$)
- θ Significantly different from 0 km in the WET trial (Time effect, $p < 0.01$)
- φ Significantly different from 0 km in the 33 trial (Time effect, $p < 0.01$)
- ψ Significantly different from 10 km in the 66 trial (Time effect, $p < 0.03$)

Exercise Performance

No significant trial effect was observed for 80 km cycling time, mean velocity, % PPO, or mean power in Watts (Table 2) so that performances in the different experimental trials were not different from each other. There also was no effect of fluid ingestion on 40 km split times (0-40 km or 40-80 km) for all experimental conditions. In the 33, 66, and 100 trials, subjects completed the final 40 km (40-80 km) in a faster time compared to the initial 40 km (0-40 km), as indicated by subtracting the 0-40 km time from the 40-80 km time (Ad Lib: 1.5 ± 4.01 ; 0: 0.99 ± 2.53 ; 33: -0.46 ± 2.93 ; 66: -0.76 ± 2.80 ; 100: -0.09 ± 3.07 ; WET: 2.74 ± 3.32 min) ($p = 0.39$). However these differences were not significant.

Only a time effect ($p < 0.01$) and not a significant group by time interaction was found in the 10 km split times, indicating that there was no effect of fluid ingestion on the pacing strategies employed by the cyclists (Table 6).

Correlations

Some significant relationships were observed. First, there was a significant relationship between the percentage of PPO at which each time trial was completed and the post-exercise (peak) T_{re} achieved ($r^2 = 0.30$, $p < 0.01$) (Figure 5a). Second, the peak T_{re} also correlated ($r^2 = 0.21$, $p < 0.01$) with the performance time in minutes for the 80 km time trial (Figure 5b). In contrast, no relationship ($r^2 = 0.02$, $p = 0.36$) was found between the change in T_{re} from 10 km to 80 km and the percentage weight loss incurred during each experimental condition (Figure 5c). In addition, no relationship was found between the percentage weight loss at the end of each time trial and the total amount of Q_s ($r^2 = 0.01$, $p = 0.62$) (Figure 5d).

Table 4. Ratings of perceived exertion in all conditions. No statistically significant interactions were found between the conditions at any point.

	0 km	10 km	20 km	30 km	40 km	50 km	60 km	70 km	80 km
0	1.1 ± 0.9	4.1 ± 1.2	4.7 ± 1.2	5.2 ± 1.4	5.8 ± 1.1	6.5 ± 1.0	7.2 ± 0.9	8.0 ± 0.9	9.3 ± 0.9*
WET	1.2 ± 1.1	3.8 ± 0.5	4.1 ± 0.9	4.7 ± 0.9	5.7 ± 0.4	6.3 ± 0.4	7.1 ± 0.7	8.6 ± 1.3	9.9 ± 1.3*
33	1.1 ± 0.9	4.0 ± 1.5	4.3 ± 1.4	4.7 ± 1.5	5.2 ± 1.1	5.9 ± 1.2	6.8 ± 1.2	8.3 ± 1.2	10.0 ± 0.6*
Ad Lib	1.9 ± 1.2	4.5 ± 1.1	5.2 ± 2.3	5.8 ± 2.3	5.4 ± 1.3	6.2 ± 1.0	7.5 ± 1.2	7.8 ± 1.2	10.0 ± 0.9*
66	0.9 ± 0.7	3.8 ± 1.1	4.5 ± 0.8	4.7 ± 0.7	5.0 ± 0.7	5.8 ± 0.8	6.7 ± 1.5	7.8 ± 1.2	9.8 ± 1.0*
100	0.8 ± 1.0	3.8 ± 0.9	4.1 ± 0.8	4.6 ± 0.8	5.2 ± 0.9	5.6 ± 0.9	6.1 ± 0.9	7.6 ± 1.8	9.3 ± 1.5*

* Significant time effect ($p < 0.01$)

Table 5. Ratings of thermal comfort in all conditions. No statistically significant differences were found between the conditions at any point.

	0 km	10 km	20 km	30 km	40 km	50 km	60 km	70 km	80 km
0	4.0 ± 1.1	4.8 ± 0.7	5.2 ± 0.8	5.3 ± 0.8	5.6 ± 0.6	5.8 ± 0.8	6.0 ± 0.9	6.4 ± 0.7	6.6 ± 0.5*
WET	4.5 ± 1.2	4.8 ± 0.8	4.9 ± 0.8	5.4 ± 0.9	6.0 ± 0.6	6.1 ± 0.8	6.0 ± 0.6	6.6 ± 0.5	6.7 ± 0.5*
33	4.3 ± 1.2	5.0 ± 0.8	4.9 ± 0.9	5.1 ± 0.5	5.3 ± 0.4	5.5 ± 0.8	5.8 ± 0.7	6.3 ± 0.4	6.6 ± 0.5*
Ad Lib	4.3 ± 0.9	5.2 ± 0.8	5.4 ± 0.9	5.6 ± 0.7	5.7 ± 0.6	5.7 ± 0.6	5.7 ± 0.6	5.5 ± 1.0	6.3 ± 1.0*
66	3.8 ± 1.5	4.8 ± 0.8	5.3 ± 0.9	5.2 ± 0.7	5.2 ± 0.5	5.4 ± 0.9	5.5 ± 0.9	5.8 ± 0.8	6.3 ± 0.8*
100	4.0 ± 1.3	4.6 ± 1.0	4.8 ± 0.8	4.8 ± 0.8	5.3 ± 0.5	5.3 ± 0.8	5.4 ± 0.8	5.3 ± 0.9	5.8 ± 1.0*

* Significant time effect ($p < 0.01$)

Table 6. 10 km split times for all conditions. No statistically significant differences were found between the conditions at any point.

	10 km	20 km	30 km	40 km	50 km	60 km	70 km	80 km
0	15.74 ± 0.68	15.87 ± 0.67	16.12 ± 0.58	16.09 ± 0.64	16.60 ± 0.74	16.13 ± 1.22	16.38 ± 1.14	15.69 ± 1.28
WET	15.31 ± 0.90	15.66 ± 1.01	16.00 ± 1.15	16.35 ± 0.92	16.58 ± 0.98	16.73 ± 1.23	16.68 ± 1.33	16.07 ± 1.30
33	16.00 ± 0.75	16.09 ± 0.70	16.50 ± 0.86	16.57 ± 0.63	16.26 ± 0.90	16.58 ± 1.57	16.41 ± 1.07	15.46 ± 0.99
Ad Lib	14.86 ± 0.94	15.20 ± 0.97	15.60 ± 0.71	15.69 ± 1.24	16.50 ± 1.34	15.63 ± 1.75	15.81 ± 1.00	14.91 ± 0.65
66	15.57 ± 0.84	15.84 ± 1.00	16.01 ± 0.91	16.00 ± 0.71	16.00 ± 0.70	15.94 ± 0.56	15.66 ± 0.56	15.06 ± 0.53
100	15.53 ± 0.62	15.61 ± 0.81	15.82 ± 0.69	15.80 ± 0.63	16.08 ± 1.03	15.95 ± 0.98	15.51 ± 0.83	15.14 ± 1.21

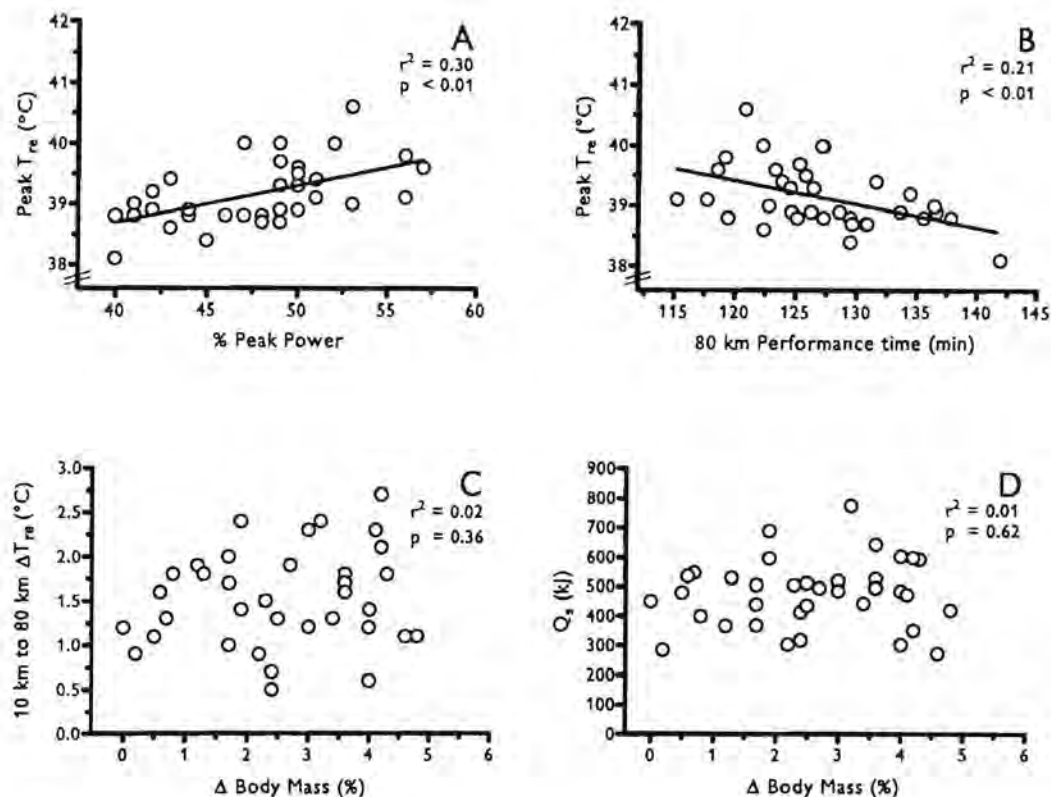


Figure 5. Post (peak) T_{re} vs. the percentage of PPO at which each time trial was completed (5a), final T_{re} vs. the 80 km time trial performance times (5b), the change in T_{re} from 10 km to 80 km vs. the pre- to post-exercise percent change in mass (5c), and total heat storage (Q_s) vs. the percentage change in weight (5d).

HI and LO Groups

Table 2 shows that although performance times were not significantly different between the six experimental conditions, performances in the three conditions ingesting the most fluid (Ad Lib, 66, and 100) were all substantially faster than were performances in the three groups that ingested the least (WET, 33, and 0). In order to exclude a false-negative finding caused by low statistical power, we therefore decided to analyze further the data according to two groups based on the extent of the fluid replacement during the time trials: HI, which contained the three trials with the highest rate of fluid ingestion (Ad Lib, 66, 100), and LO, which included the three trials with the lowest rates of fluid ingestion (WET, 33, 0).

Fluid and weights

The two groups differed significantly in the percent weight they lost during exercise and the volume of fluid they ingested (Figure 6). HI replaced $68.4 \pm 21.8\%$ of their weight losses compared to $10.8 \pm 15.8\%$ in LO ($p < 0.01$) (Figure 6a), and lost less weight ($1.5 \pm 0.8\%$) than did LO ($3.7 \pm 0.7\%$) ($p < 0.01$; Figure 6b). The total volume of fluid ingested was also significantly different between the groups ($p < 0.01$) (Figure 6c).

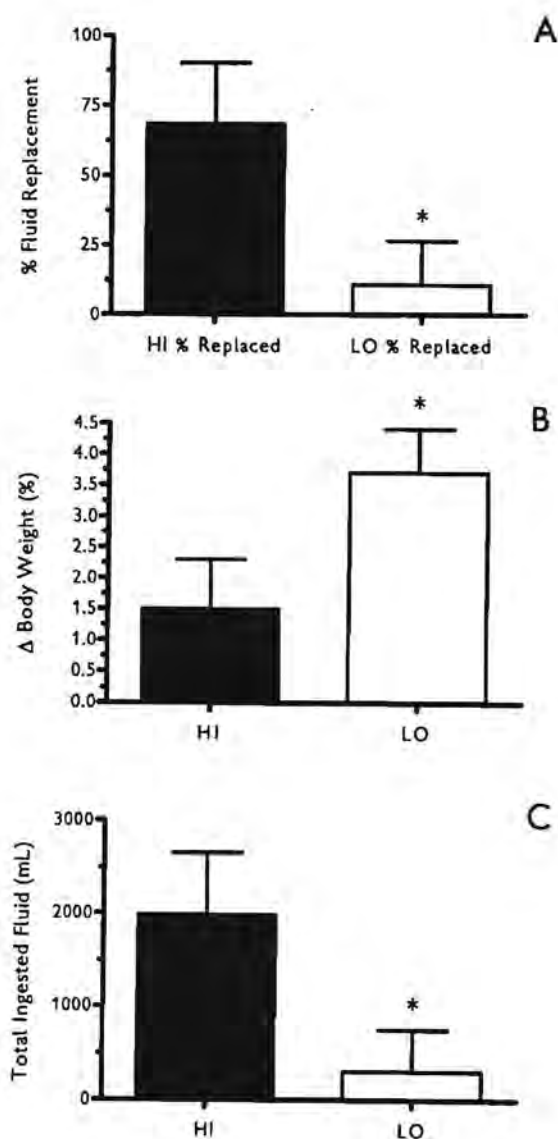


Figure 6. Percentage fluid replacement (6a), percentage change in body weight (6b), and total fluid ingestion (6c) in the HI and LO groups.

* Significantly different from HI ($p < 0.01$)

The mean absolute change in weight in HI was 1.0 ± 0.6 kg and was significantly less than in LO (2.5 ± 0.5 kg) ($p < 0.01$). Mean hourly volumes of ingested fluid were significantly higher in HI ($p < 0.01$) and are shown in Table 7. No significant differences were found between HI and LO for the total sweat losses ($p = 0.22$) or the hourly sweat rates (1.39 ± 0.18 vs. 1.30 ± 0.15 L·h⁻¹, HI vs. LO respectively) ($p = 0.88$) (Table 7).

Table 7. HI and LO groups.

	Hourly fluid volume (mL)	Total sweat losses (mL)	Performance time (min)	Post T_{re} (°C)	Mean Power (Watts)	% PPO
HI	953 ± 321	2900 ± 306	125.23 ± 5.23	39.1 ± 0.4	209 ± 22	49 ± 5
LO	$142 \pm 210^*$	2783 ± 257	$129.16 \pm 6.51^\dagger$	39.2 ± 0.6	$193 \pm 22^*$	$45 \pm 4^*$

* Significantly different from HI group ($p < 0.05$)

† $p = 0.054$

HI and LO Exercise performance

Time trial performance times, mean power in Watts, and mean % PPO are presented in Table 7. The effect of fluid ingestion on time trial performance time approached significance ($p = 0.054$) with the mean 80 km performance time in HI faster by 3.93 min compared to LO (Table 7). The HI group completed their time trials at a significantly higher power output ($p < 0.05$) and higher percentage of peak power ($p < 0.05$) compared to LO (Table 7).

There was a significant time effect ($p < 0.01$) and group effect ($p < 0.05$) on the mean power output for each 10 km split, although there was no group by time interaction (Figure 7a). The HI group maintained a higher power output from 0-40 km (209 ± 27 vs. 197 ± 22 , HI vs. LO) and from 40-80 km (209 ± 26 vs. 191 ± 26 , HI vs. LO) compared to the LO group, but only a group effect was found so that these power outputs were not significantly different from each other (Figure 7b).

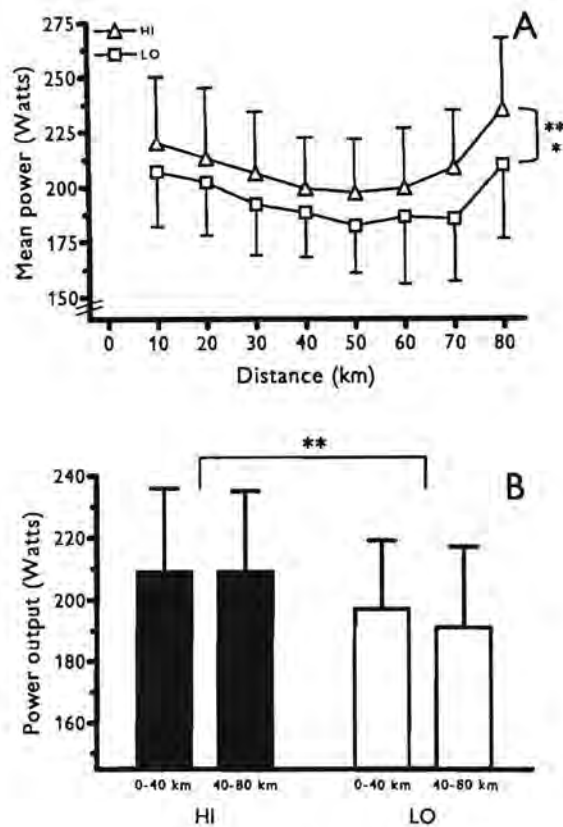


Figure 7. Mean power during each 10 km interval (7a) and 40 km interval (7b) during the time trials in both the HI and LO groups.

- * Significant time effect (p < 0.01)
- ** Significant group effect (p < 0.05)

The differences in power output over 0-40 km and 40-80 km resulted in the HI group cycling 1.59 min faster from 0-40 km (62.5 ± 3.15 vs. 64.09 ± 3.05 min, HI vs. LO, respectively) and 2.46 min faster from 40-80 km (62.72 ± 3.02 vs. 65.18 ± 4.03 , HI vs. LO, respectively) compared to the LO group. Only a group effect (p < 0.05) was found in the 0-40 km and 40-80 km split times so that these times were not significantly different from each other.

HI and LO Perceptual data

No significant group effect (p = 0.54) or group by time effect (p = 0.54) was found in the RPE data although a significant time effect (p < 0.01) was found so that the ratings increased similarly in both groups from an RPE of 1.2 ± 1.1 and 1.1 ± 0.9 (HI and LO, respectively) to $9.7 \pm$

1.1 and 9.7 ± 1.0 (HI and LO, respectively) (Figure 8a). No group effect was found in the thermal comfort data ($p = 0.10$) (Figure 8b), although there was a significant time effect ($p < 0.01$), with the thermal ratings increasing from 4.0 ± 1.2 and 4.3 ± 1.1 (HI and LO, respectively) to 6.1 ± 0.9 and 6.6 ± 0.5 (HI and LO, respectively) (Figure 8b). A group by time effect ($p < 0.05$) was found in the thermal comfort data so that the two groups responded differently, but there were no differences found between the groups at any specific time points (Figure 8b).

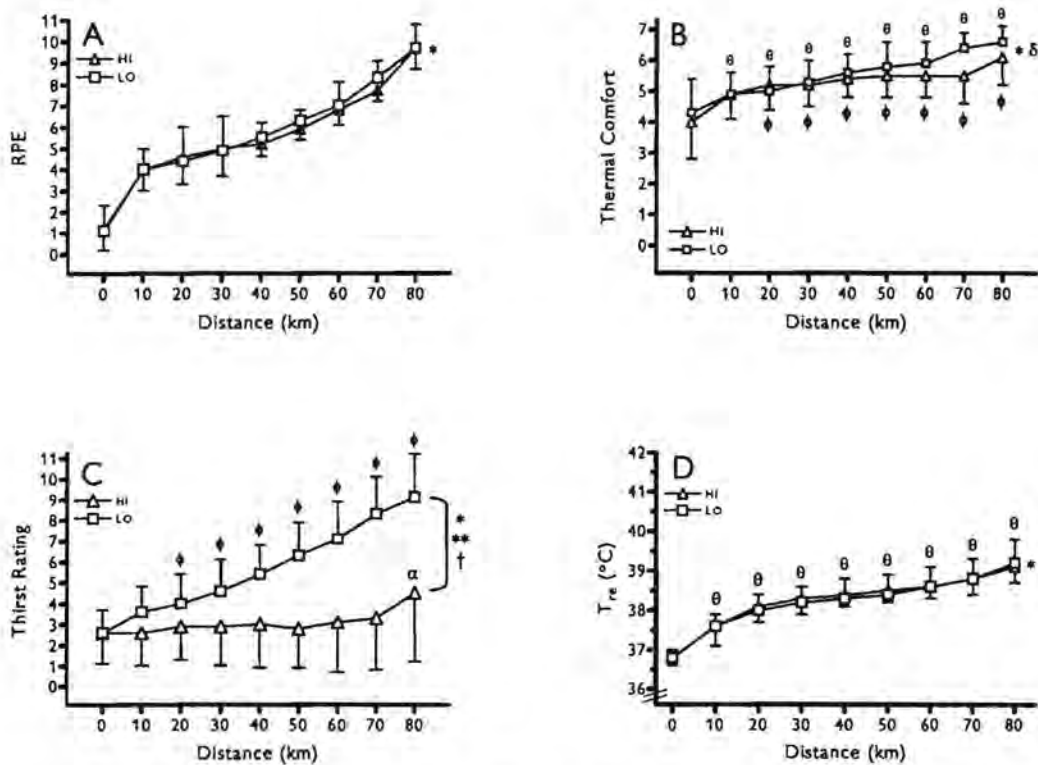


Figure 8. Mean RPE (8a), Thermal Comfort (8b), Thirst Ratings (8c) and rectal temperature (8d) at each 10 km interval during the time trials in both the HI and LO groups.

- | | | |
|----|---|----------------|
| * | Significant time effect | ($p < 0.01$) |
| ** | Significant group effect | ($p < 0.01$) |
| † | Significant group by time effect | ($p < 0.01$) |
| δ | Significant group by time effect | ($p < 0.05$) |
| θ | Significantly different from 0 km | ($p < 0.01$) |
| φ | Significantly different from 0 km and 10 km | ($p < 0.01$) |
| α | Significantly different from 0-60 km | ($p < 0.01$) |

There was a significant time effect ($P < 0.01$), group effect, ($p < 0.01$), and group by time interaction ($p < 0.01$) for the thirst ratings (Figure 8c) so that the fluid ingestion in HI maintained thirst ratings at a lower level compared to LO. In the HI group thirst increased from 2.6 ± 1.5 to 4.5 ± 3.3 , while in the LO group the ratings increased from 2.6 ± 1.1 to 9.1 ± 2.1 (Figure 8c).

HI and LO Temperatures and Q

There was no group effect or group by time effect of fluid ingestion on T_{re} (Figure 8d). Instead, only a time effect was found ($p < 0.01$) so that the two groups responded similarly, with HI increasing from 36.8 ± 0.2 to 39.1 ± 0.4 °C and LO increasing from 36.8 ± 0.2 °C to 39.2 ± 0.6 °C (Figure 8d). Similarly, no significant differences were found in Q_s between groups (454 ± 106 kJ vs. 500 ± 122 kJ, HI vs. LO respectively).

Discussion

The aim of this study was to investigate the effects of graded levels of fluid replacement on exercise performance and thermoregulation in hot and humid environmental conditions. As a consequence of the different rates of fluid ingestion, athletes completed each of the six experimental conditions with different degrees of weight loss (Figure 1). Since their total energy expenditure, and hence substrate utilization would have been essentially the same since they covered the same total distance in each trial, these differences in weight loss during exercise represent proportional differences in total body water content. Therefore each trial was completed at a different level of fluid loss (dehydration). Accordingly this experimental protocol permits an analysis of the effects of graded dehydration on several different performance and temperature-related variables during self-paced 80 km cycling time trials in hot, humid conditions but with appropriate facing wind speeds that more closely simulate out-of-doors cycling.

Performance

The first important finding of this study was that we were unable to detect a dose-dependant effect of the rate of fluid ingestion on performance. Rather, performance appeared to follow an L-shaped curve with the slowest performance in groups ingesting either no fluid (0 and WET) or 33% of their sweat losses and the fastest performance in the *ad libitum* condition (Ad Lib) (Table 2). However, the performance times were not statistically different between trials. But small numbers of subjects would have prevented a significant finding unless the effect of fluid ingestion on performance was extremely large.

Since subjects ingested the same amounts of CHO in all trials, any effect on performance was not due to an increased CHO intake that would have occurred if the ingested fluid had contained CHO. Previous studies (41; 72; 147) do not appear to have controlled for this variable even though CHO has an independent effect on exercise performance and fatigue (21).

Since the absence of a significant effect of the rate of fluid ingestion on performance could have been due to a much smaller effect of dehydration on performance than is currently believed (18; 42) and which our the small sample size could not have detected, we also analyzed the data according to those trials in which subjects drank less (LO) or the same or more than *ad libitum* (HI). When analyzed in this manner a small but significant effect of fluid ingestion on performance could be detected (Table 7).

Power outputs

Thus a group effect on mean power output ($p < 0.05$) and the percentage of PPO maintained during the time trials ($p < 0.05$) was found, and a near group effect on performance time ($p = 0.054$) (Table 7) was shown for the HI and LO groups. Therefore the ingestion of fluid in vol-

umes of *ad libitum* or higher allowed the subjects to maintain higher power outputs during the time trials, and especially during the second half of the time trials (Figure 8a).

In addition, even though the values approached significance ($p = 0.054$), the mean performance time of subjects ingesting 33% or less of their measured sweat losses (LO; $10.8 \pm 15.8\%$ mean replacement) was slower by 3.1% compared to the mean performance time when they replaced ~50% or more (HI; $68.4 \pm 21.8\%$ mean replacement) of their sweat losses. In this study, a 3.1% performance decrement equates to a time loss of approximately 4.25 minutes or a distance of over 2.5 km when cycling at the velocities achieved by these cyclists. Such a difference is likely to be highly significant to competitive cyclists.

A significant group effect was found ($p < 0.05$), but there was no interaction ($p = 0.416$) when comparing the 0-40 km vs. the 40-80 km split times. The 40-80 km split time was slower by 2.46 minutes in the LO group (62.72 ± 3.02 vs. 65.18 ± 4.03 , HI vs. LO respectively). This was not a statistically significant difference, but represents a 3.9% difference between the two groups. Such a difference is similar to the difference in performance time (3.1%) and is likely to be of value to competitive cyclists since, at these velocities and power outputs, 3.1% represents a distance loss of over one km. Interestingly though, the increase in mean power output during the final 10 km was similar between the conditions so that HI increased their mean power output during the final 10 km by an average of 26 W from 208 to 234 W, and LO increased their mean power output by 24 W from 185 to 209 W (Figure 8a), resulting in 10 km split times from 70-80 km of 15.04 ± 0.81 min in HI compared to 15.74 ± 1.15 min in LO.

Thus there appeared to be no effect of fluid ingestion on the ability to increase the power output in anticipation of the end of the time trial (the “end spurt” phenomenon), suggesting

that the body retained a similar reserve capacity between both groups. The presence of the "end spurt" confirms anticipatory regulation of power output during the trials (240; 241) including an effect of the anticipated rate of fluid ingestion on prescient response.

Indeed the difference in power outputs between the HI and LO groups (Figure 7a) was present already after 10 km and was sustained for the rest of the trial. This is consistent with the finding that performance in the heat is impaired shortly after the onset of exercise, in anticipation, in order to prevent the attainment of an abnormal elevation in T_{re} (132; 135; 234; 240). The importance of the present study is to show that the knowledge, prior to the onset of exercise, of the amount of fluid that each cyclist would be allowed to ingest during the exercise bout was enough to produce an anticipatory reduction in power output, already within the first 10 km of the 80 km cycling time trial.

It has been stated previously that dehydration as low as 1-3% creates a performance decrement of up to 20-50% (7; 8; 18; 139; 247). In the current study in the LO trial subjects had already lost approximately 1.4 kg (mean sweating rate of $1.39 \pm 0.18 \text{ L}\cdot\text{h}^{-1}$) at 40 km and thus at the halfway point they were already dehydrated by 2%. Subsequently they cycled slower by 1.7% (1.09 min) during the second half (40-80 km) of the time trial. In contrast the HI group subjects cycled only 0.35% slower during the second half of the time trial (62.50 ± 3.15 vs. 62.72 ± 3.02 min, 0-40 vs. 40-80 km) (Figure 7). This suggests either that dehydration of 2% or the knowledge that inadequate fluid would be provided during the time trial impaired performance in the LO group by approximately 1.3% during the second 40 km (Figure 7b). This effect is substantially smaller than is usually claimed (7; 8; 18; 139; 247).

However, the mechanism for this effect was not disclosed by any of the measurements we made since the T_{re} , RPE, and thermal comfort responses were nearly identical between the HI and LO groups (Figure 8). However sensations of thirst were significantly greater in the LO than HI group (Figure 8c) and could conceivably have contributed to the impaired performance in LO. But it is important to stress that this performance difference was already present at 10 km when the difference in thirst ratings and dehydration levels would have been minimal.

Thus these findings could be interpreted as evidence that the prior knowledge that drinking during the exercise bout would be less than *ad libitum*, rather than the actual level of thirst or dehydration produced, is the real explanation for the impaired performance in the LO group.

Kay and Marino (109) and Kay et al. (111) have shown that performance is not determined by peak (terminal) T_{re} alone, but rather by the pacing strategies used during self-paced exercise. It was concluded that these pacing strategies allowed subjects to anticipate the energy expenditure required to complete the task safely without the achievement of a critically high T_{re} . Indeed other studies from this laboratory suggest that the rate of heat accumulation is an important regulator of exercise performance and the voluntarily chosen pacing strategy (240; 241).

Much like Kay and Marino (109), this study has demonstrated that subjects select a suitable pacing strategy in order to complete a known distance that is presented to them within a specific context, for example in hot and humid environmental conditions. This is represented by the narrow range of percentage of PPO at which each subject completed his 80 km time trials in all the experimental conditions (Table 2). No significant trial effect was found ($p = 0.34$),

and interestingly all subjects chose to cycle between ranges of 45-50% of their PPO regardless of the amount of fluid ingested, suggesting that the pacing strategy and therefore the metabolic rate are maintained in order to produce an individually suitable peak rectal temperature at the end of each time trial (240; 241).

Our next important finding was to show that any effect of fluid ingestion on performance was not due to an effect of the ingested fluid acting on sensory receptors in the mouth since rinsing the mouth with appropriately large volumes of water in WET (1813 ± 232 mL) did not improve performance compared to when no fluid was ingested. Rather, these two trials produced similar performance times (129.4 ± 8.1 vs. 128.3 ± 6.3 , WET vs. 0, respectively). Walsh et al. (247) also showed that mouth rinsing did not influence performance, even though the exercise duration (60 min), mode (fixed workload) and performance measure (open loop time to fatigue) were different from the current trial. This contrasts with the effect of glucose which, at least during shorter duration exercise of 60 min in thermo-neutral conditions, appears to produce an ergogenic effect by acting on sensors in the mouth (31). To our knowledge this is the first study designed to determine whether the beneficial effect of fluid ingestion on exercise performance is due to a biological effect of the absorbed fluid, or to the knowledge that such ingestion will occur *ad libitum* during exercise, and is not due simply to the act of ingesting fluid.

Fluid and weights

The third finding was that there was no effect of fluid ingestion on total sweat losses or sweat rates (Figure 2), or heat storage (Q_s), the rate of Q_s (Figure 3), or the final T_{re} between the original six interventions or between the HI and LO groups (Tables 2 and 7, Figure 8d).

Hence the temperature response between the six original conditions and between the HI and

LO groups was not different because the subjects either consciously or subconsciously (241) selected a pacing strategy that allowed them to complete the time trial with the same physiological (T_{re}) response (Tables 2, 3, and 7, Figure 8d) even though they received different amounts of fluid during each trial. The result was that they cycled at lower power outputs (Table 7 and Figure 7) and hence lower metabolic rates when ingesting smaller fluid volumes. We again emphasize that alterations in power output were present already at 10 km indicating that this effect was anticipatory and occurred specifically to ensure that a “critical” T_{re} was not reached (240; 241).

Temperature responses

Although there appears to be an effect of dehydration on the ability of the thermoregulatory system to conduct heat from the core to the skin (80; 83; 84), the current study suggests that the brain compensates for any effect of anticipated lower rates of fluid ingestion on heat conductance by lowering the metabolic rate so that a critical core temperature is not ever reached, or, rather, that an individually acceptable core temperature is achieved (Table 3). Evidence for this interpretation is the low mean post- T_{re} intra-subject CV ($0.72 \pm 0.27\%$) (Table 3).

Finally, the T_{re} responses between the HI and LO groups were similar (Figure 8d). This finding shows the nature of a homeostatically-regulated system. Thus in the LO trial, the power output and hence the metabolic rate was reduced in anticipation so that performance was impaired, yet the T_{re} response was identical (Figure 8d). Although skin blood flow was not measured in this study, others have shown that dehydration reduces skin blood flow(80). But this study demonstrates that even if skin blood flow was reduced in the LO group so that their capacity to conduct heat from the core to the skin was impaired, by voluntarily reducing

their power outputs in anticipation on the basis of prior knowledge of how much fluid they would be allowed to drink during the time trial, each athlete maintained thermal homeostasis and completed the time trial without achieving a critical elevation in T_{re} (Figure 8d).

Thus in this study there was no risk for “thermal injury” even in athletes who were dehydrated by 2.9-4.3% in the LO group as shown by their similar T_{re} response to that in the HI group (Figure 8d) and as also shown by others (144). Rather, it appears that in the current study the subconscious decision to decrease the power output was made “in anticipation” during the time trials in the LO group so that the heat production and hence the heat storage was similar between the two groups (454 ± 106 kJ vs. 500 ± 122 kJ, HI vs. LO respectively).

Finally, it should be noted that drinking at high rates (“as much as tolerable”) during prolonged exercise could adversely effect performance in competitive cycling, especially during a time trial, in two ways that cannot be detected in laboratory-based testing. First, the act of drinking requires assumption of a less aerodynamic position which will require an increase in power output to maintain the same speed. Second, athletes exercising at high absolute and relative metabolic rates will have correspondingly high sweat rates ($> 1 \text{ L}\cdot\text{h}^{-1}$). If these athletes wish to replace 100% of their sweat losses, they would need to carry approximately one kg of extra weight per hour that they race. Additional mass on either the cyclist or the machine increases the rolling resistance, which in turn elevates the power required to maintain a given velocity. In the current study the differences in weight between Ad Lib ($54.2 \pm 15\%$ of sweat losses) and 100 ($90.7 \pm 4.4\%$ of sweat losses) was 1.1 kg or 1.6% at the completion of the time trial, meaning that the subjects who “drank to replace all their fluid losses” cycled with a potentially superfluous 1.1 kg during the 100 condition.

Kyle (115) calculated the differences in 40 km time trial performance when additional weight is added to the cyclist. One kg of additional mass when cycling between 35-42 km·h⁻¹ creates enough additional rolling resistance to slow the cyclist by 1.4-2.2 s, while the addition of two kg slows the cyclist by 2.8-4.5 s. These calculations assume a flat, straight course with no wind and no turn-around (115). Thus this is the minimal effect which will be substantially increased when cycling uphill.

Thus although 1.1 kg of extra mass represents a small difference in time during a flat time trial, this is a meaningful difference for 1) elite athletes and 2) sub-elite athletes who are trying to perform at their highest level. More importantly, the effect would be substantially amplified during any time trial that includes any uphill section(s).

Conclusion

In conclusion, 80 km cycling time trial performance was not affected by dehydration in a *graded* manner in the same way as thermoregulation appears to be (when the work rate is fixed) (41; 72; 147) since the performance times of all six experimental conditions were similar (Table 2) even though the subjects completed each trial with different levels of fluid loss. However, small numbers may have prevented a significant finding if the effect of dehydration on performance is substantially less than is currently believed (7; 8; 18; 139; 247). Thus when analyzed in the HI and LO groups, prior knowledge of how much fluid would be ingested during the time trial had a small but significant effect on subsequent performance in that time trial.

When subjects ingested fluid at less than *ad libitum* rates their performance times were 3.1% slower, indicating that either dehydration alone or the knowledge that an inadequate fluid

volume would be ingested inducing increased sensations of thirst (Figure 8c) impaired performance. Taken together with the finding that higher rates of fluid ingestion did not enhance performance in a graded manner, these data appear to indicate that *ad libitum* drinking may optimize performance so that it is unnecessary for an athlete to replace 100% of his or her sweat losses to achieve the best possible result.

Furthermore the data support the finding that the body readily copes with any effects of dehydration on temperature regulation by altering the metabolic rate and therefore reducing heat production. This alteration occurs "in anticipation," specifically to ensure that a critical elevation in T_{re} (84) does not occur during prolonged exercise, especially in the heat when athletes are free to pace themselves.

Finally, the rectal temperature responses to the 80 km time trials were similar among all six experimental conditions, although the subjects finished each time trial in different times and at different levels of dehydration. The individual physiological responses were therefore the same between experimental conditions even though subjects ingested quite different amounts of fluid during each condition. This suggests that the amount of work that can be performed safely in these conditions is carefully controlled by the brain (132; 167; 179; 185) so that a critical core temperature is never reached, or alternatively so that an individually acceptable core temperature is always achieved regardless of the drinking behavior and hence the fluid status of each individual.

Chapter 6

Hyponatremic encephalopathy despite a modest rate of fluid intake during a one-day 109 km cycle race

This chapter has been published in its entirety as a Case Report in the British Journal of Sports Medicine (Br J Sports Med 39(10):e38, 2005).

Introduction

There is now a near complete international consensus that hyponatraemic encephalopathy (HE) develops in subjects who ingest excessive volumes of fluid during exercise and who gain weight, as a result developing “water intoxication” (68; 97; 106; 173; 225; 228). Accordingly, fluid ingestion guidelines recently accepted by United States Track and Field (USATF) (173) suggest that athletes should drink “ad libitum” during exercise, that is, according to thirst, since this biologically driven approach, adopted naturally by all creatures other than exercising humans who have been taught otherwise, will likely prevent either drinking too little or drinking too much.

However, the apparent reluctance to accept that a basic physiological drive can produce safe drinking behaviors in humans, as it does in all other creatures, continues to underpin advice that athletes must be provided with specific guidelines for rates of fluid intake during exercise. Therefore the USATF guidelines suggest that rates of fluid intake during exercise should be between 400-800 mL·h⁻¹ (173), although others advise rates as high as 1000 mL·h⁻¹ (42). These values are, however, substantially less than rates of 1200 mL·h⁻¹ advocated by the American College of Sports Medicine(40) and of up to 1800 mL·h⁻¹ originally proposed by the United States Army (153).

Yet there are examples of athletes who have developed water intoxication during exercise despite drinking fluid at rates substantially lower than these currently prescribed rates.

Speedy et al. (224) reported the development of hyponatraemia in two Ironman triathletes whose estimated rates of fluid intake were 733 and 764 mL·h⁻¹ during 13.3 and 12 hours of exercise, respectively. These rates fall within the range of 400-1000 mL·h⁻¹ currently advo-

cated (42; 173). Glace et al. (74) reported that athletes who ingested fluid at a rate of $700 \text{ mL}\cdot\text{h}^{-1}$ developed gastrointestinal symptoms during a 160 km trail race, whereas Stuempfle et al. (229; 230) reported a reduction in serum sodium concentrations ($[\text{Na}^+]$) in athletes completing a 100 mile Iditasport ultra-marathon completed in cold conditions in Alaska, even though they drank only $200\text{-}400 \text{ mL}\cdot\text{h}^{-1}$. Similarly, Twerenbold et al. (242) reported a mean decrease in serum $[\text{Na}^+]$ of $6.2 \text{ mmol}\cdot\text{L}^{-1}$ in women who ingested $1000 \text{ mL}\cdot\text{h}^{-1}$ of water for four hours when covering 40 km at a pace equivalent to a 4:15 (h:mm) 42.2 km marathon. This rate of fluid ingestion was twice the sweat rate of these athletes; as a result the athletes gained two kg during the four hours that they ran.

These data therefore indicate that medical complications can occur in athletes who drink at rates which are less than the maximum "safe" values currently proposed by various authorities.

As part of a cross-sectional study of 193 athletes in the 2003 109 km Cape Argus – Pick 'n' Pay Cycle Tour in Cape Town, South Africa, which attracts > 35,000 participants annually, we identified one subject who developed mild hyponatraemic encephalopathy with a post-race serum $[\text{Na}^+]$ of $129 \text{ mmol}\cdot\text{L}^{-1}$, despite a relatively modest rate of fluid intake during the race. Here we report the physiological basis for the development of her condition. This case study adds further to the evidence showing that drinking to a fixed rate in excess of thirst can produce medical complications even when the rate appears quite modest.

Case Report

A 57 year old female cyclist (pre-race weight of 66.6 kg) who had previously completed the 109 km cycle tour on 16 occasions (best time 3:57, h:mm) volunteered to participate in a

study of fluid balance and serum $[\text{Na}^+]$ during the race according to previously described methods (180; 215; 216).

Environmental conditions during the race were the following: at 6:00 AM the ambient temperature was 18.3 °C and the relative humidity was 99%; at 9:00 AM the ambient temperature was 19.6 °C and the relative humidity was 99%; at 12:00 PM the ambient temperature was 21.7 °C and the relative humidity was 77%; at 4:00 PM the ambient temperature was 23.4 °C and the relative humidity was 75%.

Following her completion of the race in 5:10 (h:mm), she complained of the symptoms of headache, light-headedness and difficulty concentrating. She had experienced these symptoms previously on numerous occasions following rides of > 100 km, and in at least one 42.2 km marathon some 20 years earlier.

She started the race with a weight of 66.6 kg. Her total fluid intake during the race was 3800 mL ($735 \text{ mL}\cdot\text{h}^{-1}$). Her post-race weight was 69.0 kg which indicated a total weight gain during the race of 2.4 kg (3.6% of body weight). To account for this 2.4 kg of weight gain during the race, her estimated sweat rate during the race would have been approximately $270 \text{ mL}\cdot\text{h}^{-1}$. Her post-race serum $[\text{Na}^+]$ was $129 \text{ mmol}\cdot\text{L}^{-1}$ and she was the only athlete in our trial of 196 cyclists who developed a serum $[\text{Na}^+]$ less than $135 \text{ mmol}\cdot\text{L}^{-1}$ (Figure 1) and the only athlete in the study to develop symptoms of hyponatremic encephalopathy.

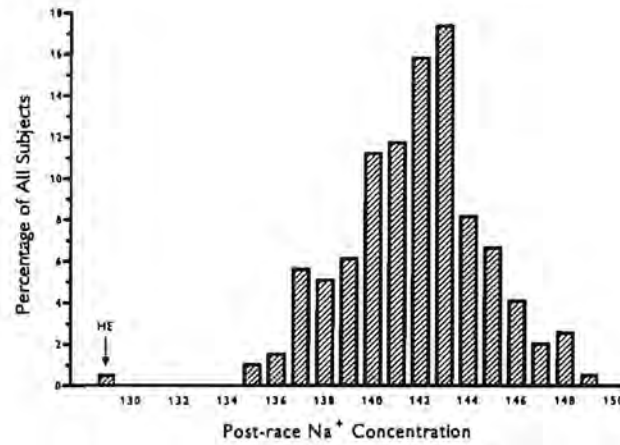


Figure 1. Distribution of post-race serum Na^+ concentrations in 196 ($N = 103$, Men; $N = 93$, Women) cyclists who completed the 109 km cycle race. The athlete reported in this study is identified as "HE" (hyponatraemic encephalopathy) and was the only athlete who developed hyponatraemia (serum Na^+ concentration $< 135 \text{ mmol.L}^{-1}$).

As in all our and other previous studies (97; 180; 215; 216; 221; 222; 224; 225) an inverse linear relationship was found between the change in mass during exercise and the post-race serum $[\text{Na}^+]$ so that those athletes who lost the least weight during the race had the lowest post-race serum $[\text{Na}^+]$ (Figure 2). Furthermore, the subject in this case report ("HE" in Figure 1 and Figure 2) was the only cyclist to gain a substantial amount of weight.

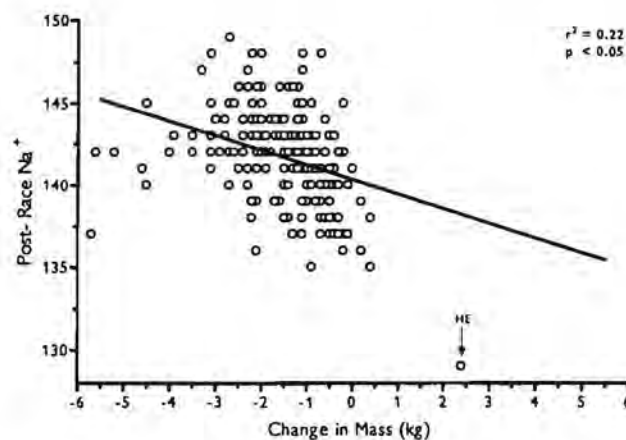


Figure 2. Relationship between change in mass and serum Na^+ concentration in 196 ($N = 103$, Men; $N = 93$, Women) cyclists who completed a 109 km cycle race. The athlete who gained the most weight is identified as "HE" on the graph and was the only athlete to develop hyponatraemia.

Two weeks after the race this athlete performed a series of laboratory experiments for measurement of her sweat rate and the $[\text{Na}^+]$ of the sweat collected from her back, arm, and leg. When cycling in a specially designed environmental chamber at a rate of 120 W in ambient conditions of 28.5 °C, 55% humidity, and with a facing air velocity of 10 km·h⁻¹ wind speed, her sweat rate was 520 mL·h⁻¹ and the sweat $[\text{Na}^+]$ on her back, arm, and leg were 73, 68, and 64 mmol·L⁻¹, respectively (average of 68 mmol·L⁻¹). Therefore, total sweat Na⁺ loss during the race was probably only 70-105 mmol, similar to values either measured (106; 170; 224) or predicted (170) in other athletes who developed this condition during prolonged exercise.

On the basis of the finding that her condition was due to voluntary overhydration without a substantial Na⁺ deficit, she was advised that in future she should always drink "ad libitum" during exercise.

On follow up after the 2004 Cape Argus/Pick 'n' Pay 109 km Cycle Tour, she reported that her *ad libitum* fluid intake rate had been not more than 500 mL·h⁻¹. Her race time was 5:38 for the 109 km course, which was similar to her time of 5:10 in the previous year. She reported being symptom-free following the race, as well as in other rides >100 km completed in the 12 months since she had been evaluated in the laboratory. In fact, she enthusiastically stated that her enjoyment of cycling has increased substantially since she began to drink less fluid during prolonged exercise.

Discussion

The only cyclist to develop hyponatraemia in our prospective study of 196 cyclists in this race was also the only cyclist to gain a substantial amount of weight (2.4 kg; 3.6% of body weight, Figure 2). This mirrors our finding at the 2001 South African Ironman triathlon in which the only athlete to develop hyponatraemic encephalopathy and who required hospitalization after the race, was also the only athlete to gain a significant amount of weight during the race (3.8 kg; 4.7% of body weight) (180).

But, more to the point, the athlete in this study developed hyponatraemic encephalopathy within a relatively short duration of exercise (5.16 h) despite a rate of fluid intake that complies with some of the current guidelines for fluid replacement during exercise (40; 42). Previously, Speedy et al. (228) have reported the development of asymptomatic hyponatraemia in two female Ironman triathletes who ingested fluids at similar rates as did the cyclist in this report. Yet those triathletes developed asymptomatic hyponatraemia only after 13 h as compared to the 5-6 h in this athlete. However, neither had symptoms of central nervous system dysfunction (hyponatraemic encephalopathy).

The aetiology of this condition clearly is the ingestion of free water at rates that exceed the rate of free water clearance by the kidneys, leading to water intoxication as faithfully reproduced in the laboratory (182; 222). The low sweat rate in this cyclist is likely due to the low exercise intensity (average cycling speed $21.1 \text{ km}\cdot\text{h}^{-1}$) combined with cool conditions in which heat losses due to convection and radiation are high. The latter two avenues of heat loss were not considered when the original studies on which the current drinking guidelines (40; 147; 153) are based were conducted (147; 153). As a consequence of the low sweat rate of this cyclist and her low rates of free water clearance, fluid retention and cerebral swelling de-

veloped, causing hyponatraemic encephalopathy according to the mechanisms we have proposed since 1985 (178), and as conclusively proven in 1991 (106), even though her rates of fluid ingestion were modest and well within currently accepted guidelines (40; 42; 153; 173).

Confirmation that fluid overload caused hyponatraemic encephalopathy in this case comes from the predictions, based on the laboratory measurement of her average sweat $[\text{Na}^+]$, that her total Na^+ losses during the race were small (approximately 70-105 mmol). Furthermore simple balance equations show that a positive change in weight of either 3.3% or 4.0% respectively, would be enough to lower a female athlete's serum $[\text{Na}^+]$ to $130 \text{ mmol}\cdot\text{L}^{-1}$, depending on whether the ingested fluid is water (3.3%) or an electrolyte-containing sports drink (4.0%) according to the calculations of Weschler (251). The actual weight change in this athlete was +3.6%, and her post-race serum $[\text{Na}^+]$ was $129 \text{ mmol}\cdot\text{L}^{-1}$, confirming the accuracy of the predictive equations of Weschler (251).

The fact that this athlete did not develop symptoms in subsequent exercise when she drank "ad libitum" and without increasing her Na^+ intake either before, during or after exercise confirms that this condition is due to fluid overload to which any Na^+ deficit plays only a minor contributory role (251) since it merely moderates the serum $[\text{Na}^+]$ at any given level of fluid overload.

Finally this study suggests that fluid replacement guidelines based on absolute rates of fluid intake are inherently flawed since sweat rates can vary so widely during exercise, ranging from $> 2 \text{ L}\cdot\text{h}^{-1}$ in American football players (62) to approximately $500 \text{ mL}\cdot\text{h}^{-1}$ in ultra-marathon runners in cold conditions (229; 230) to $300 \text{ mL}\cdot\text{h}^{-1}$ in this cyclist.

Rather, we continue to argue that no evidence exists to suggest that athletes who drink "ad libitum" as opposed to at fixed rates are at increased risk of ill health during exercise, or that they will perform less than optimally as a result (173; 175). Instead we argue that all the current evidence indicates that athletes perform optimally when they drink "ad libitum" during exercise (34; 44; 141).

Acknowledgements

We wish to acknowledge the help of Dr. Basil Bonner for his logistical support. We would also like to acknowledge the kind assistance and hard work of Ian de Witt for analyzing the sweat samples and Ingeborg Muhrfeldt for her work in analyzing the blood samples. Finally, special thanks to the Cycle Tour Trust for the opportunity to perform this research.

Response by Associate Professor William O. Roberts

(Br J Sports Med, eLetters, 11 Apr 2006):

Dear Editor,

The case study described by Dugas and Noakes (2005:39:e38) demonstrates the wide individual variability in sweat losses that occur in athletes, and there is not much doubt this woman was ill advised for her fluid replacement plan. The conclusions of the both the case report and the commentary that follows place the blame for her problem on the 1996 American College of Sports Medicine (ACSM) Fluid Replacement Position Stand, whereas the problem seems to be misapplication of the ACSM advice. The case report does not detail where she got her fluid replacement advice but her previous experiences imply that she was overhydrating during exercise for many years, and she may be a "non-responder" to a hypo-osmotic, overhydrated state.

I have copied the pertinent sections of the ACSM position stand below: From the abstract. "During exercise, athletes should start drinking early and at regular intervals in an attempt to consume fluids at a rate sufficient to replace all the water lost through sweating (i.e., body weight loss), or consume the maximal amount that can be tolerated." [I have always interpreted the last half of this statement to mean replace up to sweat losses as outlined in the text, but others may have a different view.]

From the main text: "As such, individuals participating in prolonged intense exercise must rely on strategies such as monitoring body weight loss and ingesting volumes of fluid during exercise at a rate equal to that lost from sweating, i.e., body weight reduction, to ensure complete fluid replacement. This can be accomplished by ingesting beverages that enhance drinking at a rate of one pint of fluid per pound of body weight reduction. While gastrointestinal discomfort has been reported by individuals who have attempted to drink at rates equal to their sweat rates, especially in excess of $1 \text{ L}\cdot\text{h}^{-1}$, this response appears to be individual and there is no clear association between the volume of ingested fluid and symptoms of gastrointestinal distress. Further, failure to maintain hydration during exercise by drinking appropriate amounts of fluid may contribute to gastrointestinal symptoms. Therefore, individuals should be encouraged to consume the maximal amount of fluids during exercise that can be tolerated without gastrointestinal discomfort up to a rate equal to that lost from sweating."

From the conclusion. "During exercise, fluid and carbohydrate requirements can be met simultaneously by ingesting $600\text{-}1200 \text{ mL}\cdot\text{h}^{-1}$ of solutions containing 4%-8% carbohydrate. During exercise greater than 1 h, approximately 0.5-0.7 g of sodium per Liter of water would be appropriate to replace that lost from sweating." [The key word in this section is "can," it does not say "should" or even imply that all athletes require $600\text{-}1200 \text{ mL}\cdot\text{h}^{-1}$.]

It appears that she did not get her advice from the American College of Sports Medicine Fluid Replacement Position Stand as suggested by the authors of the manuscript and the commentary, nor did she or her advisors learn from her past experiences. With a sweat rate of 270 ml/hour and a fluid intake of just over 700 ml/hour, she is either going to urinate large volumes during and immediately after her activity or she will become water intoxicated if her kidneys do not respond with appropriate diuresis. Although the wording in the abstract could be misconstrued to mean "drink as much as you can," the main text clarifies the recommendation. The recommendation stresses replacing sweat losses as the first choice in fluid replacement during exercise, and not more. While the 600-1200 mL·h⁻¹ is higher than 400-800 mL·h⁻¹ that Noakes recommends, it represents the range around the middle of the population and does not reflect the problems faced by those (including this woman) who sit at the extremes of sweat loss during activity. Even the 400 mL·h⁻¹ from Noakes' recommendation would have left this woman overhydrated at the end of her activity. To site an example like this from the lower extreme of sweat loss as a failure of the ACSM Fluid Recommendations seems a stretch to me and emphasizes that the wide variation in fluid requirements across the entire athlete population makes it nearly impossible to give a precise recommendation that includes specific fluid volumes. The best advice still remains to "replace most of the sweat losses" as outlined in the ACSM Position Stand. The ACSM Exercise and Fluid Replacement Position Stand is currently in revision and should be published in the near future with updates that reflect changes in the area since 1996.

William O Roberts MD, MS, FACSM

Past President, American College of Sports Medicine

(I have no financial or advisory ties to any water or sports drink companies. I did receive an honorarium from the Gatorade Company for appearing on an educational film clip regarding exertional heat stroke in 2003.)

Reference

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(Please see the Appendix for the original letter as it appeared on the British Journal of Sports Medicine website)

Chapter 7

Addendum 1:

Sodium ingestion and hyponatremia: Sports drinks do not prevent a fall in serum sodium concentration during exercise

Addendum 2:

New use of current technology to measure rectal temperature and heart rate during endurance exercise

The above addendums have been published in their entirety as a Letter to the Editor in the British Journal of Sports Medicine (Br J Sports Med 40(4):372-74, 2006) (49), and as a short report in the South African Journal of Sports Medicine (S Afr J Sports Med. 17(1):29-30, 2005), respectively.

Addendum 1: Sodium ingestion and hyponatremia

Dear Editor-in-Chief,

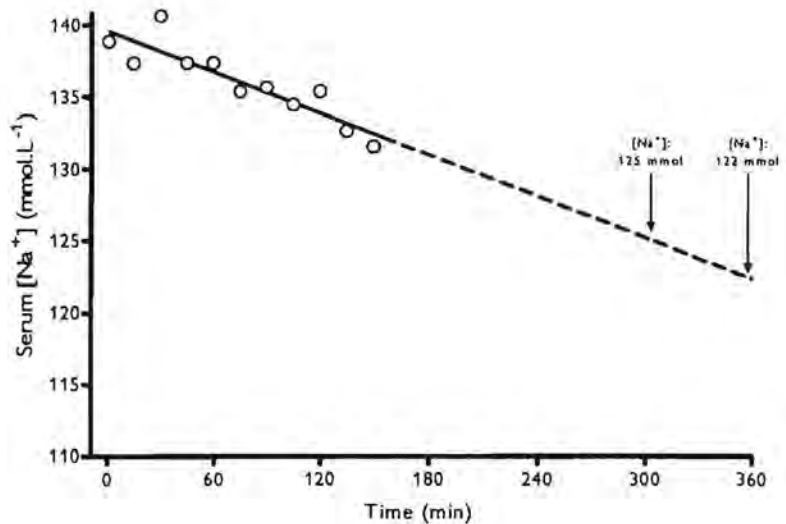
In a well-controlled and designed study recently published in another journal, Baker et al. (17) clearly demonstrated the effects of ingesting large volumes of water or carbohydrate-electrolyte sports drinks (CES) on serum Na^+ concentration ($[\text{Na}^+]$) during exercise in older, active adults (54-70 years). The data show that ingesting any hypotonic fluid, be it water or a Na^+ containing sports drink (approximately $18 \text{ mmol}\cdot\text{L}^{-1}$) in volumes that are similar to or more than weight losses produces a fall in $[\text{Na}^+]$ —a finding which mirrors the study of Twerenbold et al. published in your journal (242). Readers should know that any fluid with a $[\text{Na}^+] < 150 \text{ mmol}\cdot\text{L}^{-1}$ is considered to be hypotonic to the plasma.

In Baker et al. the ingestion of a CES produced a slower rate of fall in $[\text{Na}^+]$, as also found by Twerenbold et al. (242) and also by Barr et al. (19). However, the authors failed to conclude that the consumption of *any* hypotonic fluid at these rates nevertheless causes a steady decline in serum $[\text{Na}^+]$. Instead, Baker et al. concluded that a CES better restores plasma volume and is therefore a more effective fluid replacement in older adults during intermittent exercise (17). This conclusion ignores the fact that in their trial the serum $[\text{Na}^+]$ in both the men and women fell during exercise, regardless of the type of fluid they were ingesting, and that a falling serum $[\text{Na}^+]$ will eventually lead to hyponatremia and its associated symptoms and medical complications.

One female subject in the study of Baker et al. lowered her $[\text{Na}^+]$ by nearly $10 \text{ mmol}\cdot\text{L}^{-1}$ in just 150 minutes when voluntarily consuming 2.7 L of CES. I have re-plotted her data to show

that, had she continued drinking the CES at that rate and given a relatively constant rate of decline, after four hours her serum $[\text{Na}^+]$ would be approximately $128 \text{ mmol}\cdot\text{L}^{-1}$. After five hours she would have reached a $[\text{Na}^+]$ of $125 \text{ mmol}\cdot\text{L}^{-1}$, and after six hours it would likely have been $122 \text{ mmol}\cdot\text{L}^{-1}$ (Figure 1). Since these are serum $[\text{Na}^+]$'s at which cerebral and pulmonary edema develop (14), and at which emergency medical treatment is necessary, this finding is of particular significance.

Figure 1. Linear regression ($r^2 = 0.81$; $p < 0.05$) of the fall in $[\text{Na}^+]$ during exercise in one female subject in Baker et al. The rate of decline is approximately 3.3 mmol Na^+ per hour when ingesting a CES at a rate of approximately $1.1 \text{ L}\cdot\text{h}^{-1}$.



In 2000, the average 42.2 km marathon finishing time for women in North America was 4:57 (h:mm). In the 2005 San Diego marathon alone there were 9069 women finishers, of whom approximately 2985 finished between 4-5 h, 2379 finished between 5-6 h, and 2531 between 6-9 h (39). If these marathon finishers were to consume CES at the same rates advocated by Baker et al. and elsewhere (40) to prevent "voluntary dehydration," the number of hyponatremic cases and their associated medical complications are sure to increase, placing undue pressure on medical directors and their staff who must diagnose and treat this preventable yet complicated condition.

Of particular concern is that sports drink companies, as well as the American College of Sports Medicine (ACSM) drinking guidelines (40), currently advocate that sports drinks which contain Na^+ should be consumed to prevent hyponatremia during prolonged exercise. However, the data of Baker et al. clearly show that the consumption of any hypotonic fluid in quantities to prevent "voluntary dehydration" will inevitably produce a fall in the serum $[\text{Na}^+]$ that can eventually lead to symptomatic hyponatremia.

In order to help prevent any further increase in the number of cases of this preventable condition, I feel that this alternative interpretation of the data of Baker et al. should be brought to the attention of the readers of this journal.

Jonathan Dugas

University of Cape Town

Response by Professor W Larry Kenney

(Br J Sports Med, eLetters, 19 Apr 2006):

Dear Editor,

We appreciated the recent letter by Dugas in this journal (BJSM 2006;40:372) because it provides an excellent teaching tool for students on the dangers of over-extrapolating published data. Most scientifically puzzling is the figure published with Dugas' letter. In that figure, Dugas replots data from an elderly woman exercising intermittently for 150 minutes, then extends a linear regression line based on those data out to 6 h (a vastly different time frame) to make a point about ultra-endurance athletes (a vastly different subject population). Dugas' extrapolation ignores all negative feedback characteristics inherent in physiology (see pub-

lished subjective sensory data for this subject in Baker et al. (1)) that would have prevented this subject from continuing both exercise and her overdrinking behaviour long before 6 h. To actually achieve the 6-h end point of Dugas' linear extrapolation, this petite older woman would have to continue to overdrink at the same rate until she increased her initial body weight by over 14%! This clearly defies both logic and physiology. To see the folly in such over-extrapolation, one only needs to apply Dugas' logic – and extrapolation methodology -- to heart rate data from the same study. If Dugas had chosen heart rate instead of serum sodium concentration to over-extrapolate, many of our elderly men and women would be finishing this imaginary 6-h ultra-marathon with heart rates of ~250 bpm, obviating any concern about hyponatraemia! Such is the danger of blindly extrapolating selected laboratory data from a “well controlled and designed study”. Thus we disagree with Dr. Dugas that “this finding is of particular significance,” as it could never have occurred, but share with him a desire to prevent all athletes from substantial overdrinking.

Also in his letter, Dugas restates several conclusions that were soundly based on empirical data, then (correctly) refutes his re-interpretations. Baker et al. illustrated the dangers of drinking fluids well in excess of sweat rate, especially in smaller subjects who sweat little. In one such subject, a 45.7-kg 65-year old woman who overdrank 2.4 kg more than she sweat, serum sodium fell when she overdrank a sports drink, and fell at a substantially higher rate when she overdrank a similar volume of water. Within the time period studied, she became symptomatically hyponatraemic only in the water trial. Dugas alters that simple observation by inserting such words (note CAPS) as “in volumes that are SIMILAR TO OR more than weight loss” and attributes that statement to Baker et al. Moreover, he suggests that excessive overdrinking is “advocated by Baker et al”. I challenge Dr. Dugas to point out the line in Baker's paper where she advocates overdrinking.

It is unfortunate that several important and valid points made by Dugas and his colleagues over the years become lost when good science is sacrificed and other authors' published words are misrepresented. Drs. Dugas and his colleagues have made important contributions to our understanding of this topic that I believe all reasonable scientists working in this area agree upon: (1) Substantial overdrinking (well in excess of sweat loss) during exercise, regardless of the beverage consumed, is not to be suggested, facilitated, nor condoned; (2) Scientific organizations should not – and in my careful reading of the literature do not – simply tell athletes to “drink as much as possible”. Rather, they tell athletes to learn to drink fluids during exercise in volumes that approximate sweat loss in an effort to prevent BOTH extremes -- significant dehydration and hyponatraemia; and, (3) In order to be palatable, sodium-containing beverages are necessarily hypotonic to plasma. During prolonged exercise, they do not necessarily prevent a fall in serum sodium, but attenuate the decline, as clearly illustrated by Twerenbold et al (2) and others.

Note bene: In a previous letter addressing Baker et al., Noakes (3) took issue with a statement inaccurately referenced to Costill et al. (4) that “...carbohydrate-electrolyte solutions (CES) are more effective than water in stimulating voluntary fluid intake...and attenuating increases in core temperature during exercise-heat stress.” To clarify, when consumed in equal volumes, there is no reported benefit of CES over water. Only when the increased voluntary intake associated with CES significantly limits dehydration do laboratory studies show an attenuated core temperature response. We thank Dr. Noakes for allowing us to clarify this point.

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4. Costill, DL, Kammer WF, Fisher A. Fluid ingestion during distance running. *Arch Environ Health* 1970;21:520-25.

(Please see the Appendix for the original letter as it appeared on the British Journal of Sports Medicine website)

Addendum 2: New use of data logging technology

Introduction

The technology necessary to log data remotely and independently has been available for some years. This technology has been applied mostly to environmental and natural sciences, however, and not in life sciences. This was due primarily to the cost of the technology and the small demand for it in the life sciences, especially in studies of exercise physiology. Our recent collaboration with a local technology company (SyGade Solutions (Pty) Ltd., Johannesburg) has resulted in the use of miniature data loggers to record rectal temperature, heart rate (HR), and altitude during road and cycle racing. This technology has the potential to measure these variables simultaneously and in a free-living situation and therefore will contribute to more innovative research.

Description of the data logger

The data loggers were tested in the laboratory from 35 – 44 °C, and returned an accuracy ranging from - 0.22 – 0.1%. Each logger weighs 79 g and has dimensions of 105 x 58 x 20 mm (length X width X height, respectively). The loggers use a 16-bit micro-processor operating at 13 Mhz, and draw 20 milliamps during operation. Each is powered by a single AAA/1.5v battery that can supply enough current for up to 12 hours of logging. Data are stored on a FLASH device similar to those used currently in digital cameras. This allows for the retention of data even if power is removed during use.

The loggers can record up to 12 hours of data at two-second intervals in the absence of HR. When HR is recorded in addition to rectal temperature, the logging time decreases in direct proportion to the time interval between heart beats so that maximum logging time is short-

ened to seven hours if a HR of 220 beats.min⁻¹ is maintained. The minimum HR that can be logged is 30 beats.min⁻¹.

The loggers measure heart rate in milliseconds from beat to beat. Because the HR transmitters are not suited for beat to beat calculations, and to reduce interference from other HR monitors, the HR transmitters were hard-wired to the data logger for maximal accuracy.

Temperature is measured by supplying the probe with a very accurate reference voltage/current and then measuring the changes with a 20 bit analog to digital converter. This is then converted to temperature by means of a calibration table, as supplied by the temperature probe manufacturer (VHA Plus, Irving, Texas, USA).

The air pressure sensor is a Motorola MPX4115 which is sensitive to pressure changes within one meter of vertical distance. However, for the air pressure sensor to measure altitude accurately in metres above sea level, a sophisticated and accurate calibration is required. Therefore, the loggers were designed not to give accurate meter readings, but instead to give only relative changes in pressure and therefore create a profile of the race course. This allows the user to place an athlete at specific points on a course where changes in altitude are known and frequent.

The software for the micro-processor was compiled initially on a PC and then loaded via a special wire interface to the processor. Data are retrieved by interfacing the logger with a PC via a download cable inserted into the HR transmitter input. The programmer is then able to issue special control commands to the logger via the PC. The data are stored in a raw binary format. The processing and separating of data took place on the PC to produce text files that

were then delimited in a Microsoft Excel workbook and plotted using the GraphPad Prism software package (GraphPad Software Inc., San Diego, California, USA).

The loggers contain a function that allows the user to log an event during the data logging period. The user can log an event by pushing a button on the logger at a point specified by the researcher, for example before, during, or after the race. This allows the researcher to know exactly where the start and finish of the course are, for example, after the data have been downloaded and reduced.

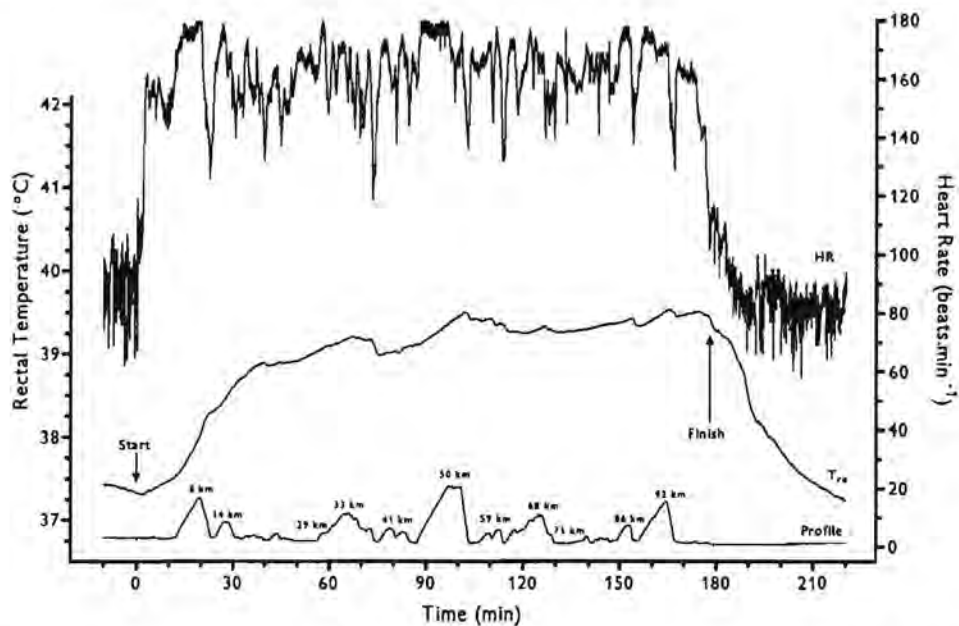


Figure 1. Data from a 100 km cycle race in moderate ambient conditions (16-23 °C). The course profile was produced with an uncalibrated air pressure sensor so that it recorded only relative changes in air pressure, and not meters above sea level. The HR transmitter was wired directly to the data logging unit for maximal accuracy. Note that within 30 minutes the subject's T_{re} had returned to pre-race values without any specific intervention.

Pilot work

Data were logged approximately every 0.03 minutes for the duration of a 100 km cycle race (Figure 1) and a 21.1 km running race (Figure 2). At the start and at the finish of each race, the athlete logged an event to distinguish the starting and finishing points. The event is marked by a "spike" in the data set that allows the researcher to identify the event and then

remove the spike from the data for presentation purposes. The cycling course profile was then compared to a profile provided by the race organizers to determine specific points on the course during the data session, and these points were then noted on the logger's profile (Figure 1). The running course had fewer geographical landmarks compared to the cycling course. Altitude readings from each kilometer on the running course were obtained from the organizers and used to generate a profile of the race course (Figure 2c). This profile was then compared to the profile created by the logger (Figure 2b).

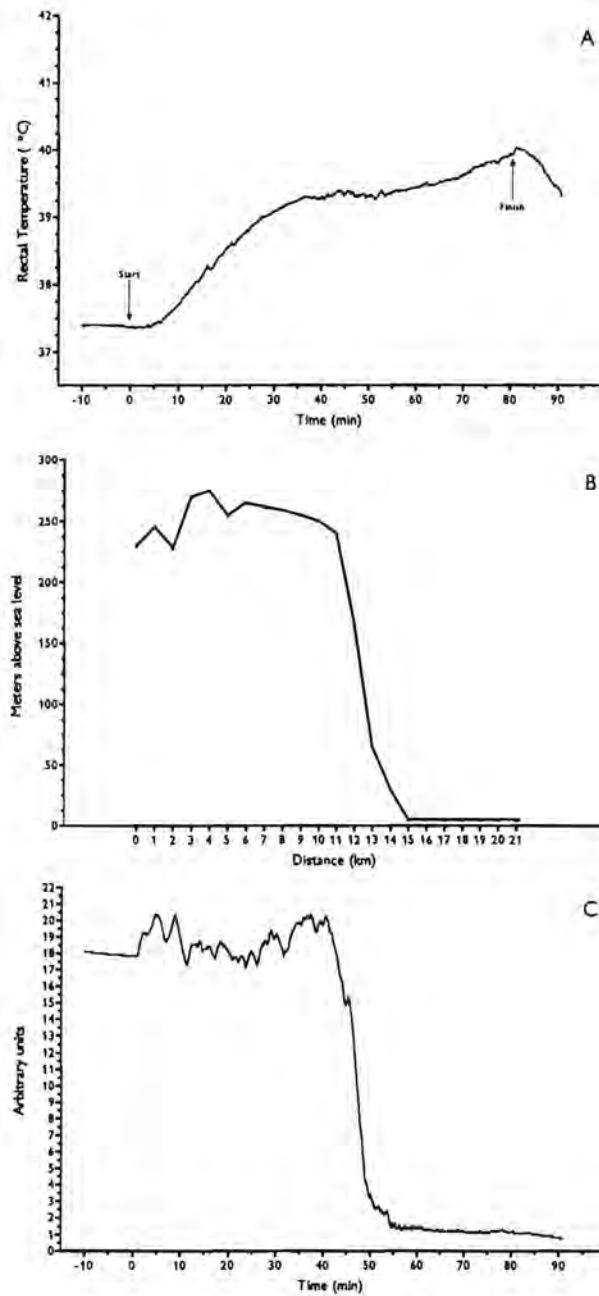


Figure 2. Data from a 21.1 km foot race. Rectal temperature and air pressure were recorded continuously during the race. Altitude at each kilometer was obtained from the race organizers for comparison with the course profile generated by the data logging unit. Note that in this athlete the peak T_{re} was achieved momentarily after the finish of the race, and was 40.7 °C even though the ambient air temperature was ~10-12 °C.

Discussion

The advent of this technology already has yielded a novel finding. The ability to measure the rectal temperature with such high resolution and over relatively long periods of time has allowed us to show that during endurance exercise the rectal temperature is a dynamic variable and appears to change with metabolic rate and course terrain, the former being normally a function of the latter.

In addition, because data of this type have never before been collected, we can see that although the rectal temperature is dynamic in nature as opposed to static, it remains within a range of approximately two degrees Celsius. This sheds new light on thermoregulatory studies, and opens new avenues of research in this area that were previously not available.

Chapter 8

Rectal temperature responses to Arctic and Antarctic swimming

Introduction

The physiological, especially thermoregulatory, response to long distance swimming in water at temperatures of 15°C or greater were first studied Pugh et al. (194; 195). These authors wished to understand how trained swimmers were able to cross safely the English Channel in 12-22 hours “when after shipwrecks persons survive in water in this temperature for only 4-6 hours” (195). They concluded that long-distance swimmers’ resistance to cold depends on superior insulation provided by their increased subcutaneous fat as well as the ability to maintain high rates of energy expenditure ($760-900 \text{ kCal}\cdot\text{h}^{-1}$) for the entire duration of these swims. Despite this, most swimmers complete English Channel swims in water temperatures of 16-18 °C with reduced core body temperatures, in one case as low as 34.0 °C (195).

More recently, besides the subcutaneous fat layer (112; 218), the protective effects of a greater upper body skinfold thickness (218; 246), a larger muscle bulk (78; 239) and a “reactive” metabolic response (96) have also been described (78). It is also now well known that swimming increases the rate of cooling of both the limbs and the body core (94-96; 112; 194), reducing the survival time in cold water of all but the most insulated swimmers.

Presently the survival limits when swimming in very cold water of 0-4 °C are not known, in part because humans do not generally choose to swim for prolonged periods in such cold water. In general it appears that survival time in cold water is an exponential function of the water temperature and becomes less than an hour in water at temperatures of 0-5 °C (236) (p.249; Figure 3), but there are few data to determine the effect of swimming at such low temperature on survival time.

In one of the earliest references, Glaser and Hervey (77) reported that of three subjects who entered a swimming pool at "about 2 °C", two were forced to exit the water in less than two minutes because they were "too breathless to swim." The third subject, a "good swimmer," lasted for nine minutes before being asked to remove himself from the water. In another early study, Glaser (76) exposed a single person "for about half a minute" in an Arctic sea inlet at a temperature of 3 °C. On the basis of this experiment, Glaser concluded that "a man who is swimming hard should produce about as much heat as he loses in water near freezing point and he should not die of cold as long as he is able to swim. Stiffness of the muscles may eventually interfere with efficient swimming; but muscles which are being exercised hard may remain warm enough to prevent them becoming stiff" (p.1068). Glaser also quoted the alleged example of a man "who survived after swimming for more than half a day in water at -1.5 °C".

However, other studies provide a less optimistic outlook. Keatinge and colleagues (113) showed that none of four subjects considered to be good swimmers was able to cover 250 m in water at 4.7 °C even though they were fully clothed and wore life jackets that kept their heads out of water. One subject was unable to cover 30 m and the longest swim time was 11 min. The inability to swim was caused by a progressive, cold-induced hyperventilation that occurred before whole body hypothermia had developed.

Similarly, Wallingford et al. (246) showed that the average distance that fit, young Canadians could swim when wearing their usual clothing in much warmer water of 14 °C was 889 m in an average time of 43.5 minutes. Interestingly, subjects became incapacitated as a result of

fatigue of their arm muscles and before they had reached critical levels of hypothermia (average core temperature at exhaustion was 35.8 °C).

Finally Hayward et al. (94) predicted that a subject wearing a life jacket would likely be able to swim for only 0.85 miles (1.4 km) in water of 12 °C before being incapacitated by hypothermia. In contrast, lying still in the Heat Escape Lessening Posture (HELP) would increase the survival time to three hours in water as cold as 5 °C (94).

In July 2005 we were approached by an experienced long distance cold water swimmer, Lewis Gordon Pugh, who wished to complete long distance (1 km or greater) swims in both Polar regions in August and December 2005 in water temperatures less than 5 °C while clothed only in a swimming costume (Speedo) and wearing a swimming cap and goggles, as dictated by the rules of English Channel Swimming Association. His subsequent, successful completion of three long distance swims in the Polar regions have allowed us (i) to speculate on the physiological adaptations that allowed him successfully to complete these swims and (ii) to define the limiting time for which he was able to swim and the distance he was able to cover in water at temperatures of 2-4 °C. We believe these data to be unique.

Methods

The swimmer in this report participated in a training and research programme that began in July 2005 and continued to December 2005. During the time period from 2003 until the present he completed a number of swims which are shown in Table 1.

Table 1. Recent swims of Lewis Pugh. The swims of interest in this report are those he completed in the Arctic and Antarctic regions.

Date	Location	Distance (km)	Duration (m:ss)	Water Temperature (°C)
18 August 2005	Magdalenefjord (79° N)	1.0	21:30	3-4
19 August 2005	Verlegenuken (80° 03' N)	1.08	20:30	3
15 December 2005	Peterman Island (65° 10' S)	1.0	18:12	0
17 December 2005	Deception Island (62° S)	1.6	30:30	2-3
January 2006	Nelson Mandela Bay, Port Elizabeth, South Africa		297	--
January 2006	Manly Beach to Sydney Opera House, Sydney, Australia	15.0	301	--
12 May 2006	Joostendal glacier, Nigardsbreen, Norway	1.2	23:50	1-2.5

Pugh set the record for the northern-most distance swim in the world in August 2003 by swimming five km around North Cape (Norway) at 71° N in ~8 °C water. On 18 August, 2005, the swimmer completed a 1.07 km swim in 21:30 at Magdalenefjord at 79° N, to break the record for the northern-most distance swim. The water temperature was 3-4 °C. Approximately 12 h later on 19 August, he completed a second swim (1.08 km) in 20:30, this time at Verlegenuken on the island of Spitsbergen in the Arctic Ocean (80° 03' N) in 3 °C water.

On 14 December 2005 the swimmer then set the record for the southern-most distance swim by completing a one km swim in 18:12 at Petermann Island (65 ° 10' S) in 0 °C water. This swim was followed three days later by a 1.6 km swim at Deception Island on 17 December 2005. The swimmer completed this distance in 30:30 in 2-3 °C water. He exited the water with a T_{re} of ~36 °C, which dropped further to 33.6 °C before climbing back to normal values.

Anthropometric measurements were performed at the start of his Antarctic training program and included a DEXA scan for body composition as well as a seven-site skinfold analysis.

Three systems were used to measure rectal temperature during this time period. In the training swims in the portable pool a YSI-compatible tele-thermometer was used to measure and record the temperature values manually. For swims in the Arctic and the Antarctic regions two custom-designed systems were developed. Each system is comprised of a portable and miniature logging unit equipped with both a rectal probe and water temperature probe. This unit transmits a radio signal to a receiver that is connected to a laptop computer. Custom software was built especially for the logging/transmission unit so that rectal temperature and water temperature could be monitored in real time and recorded continuously both locally at the logger and remotely on the laptop computer.

Acclimatisation to swimming in cold water

Since we were unaware of any swimming programs for habituation to ice-cold water, we adopted a progressive approach that allowed us to expose the swimmer incrementally to progressively lower water temperatures in a portable swimming pool over a number of weeks. Each swimming exposure was for 22 minutes and the temperature of the water in the pool was lowered by the addition 1-2 tons of ice before each swim. The training pool was portable in nature and approximately three meters in diameter and 1.5 m high. Approximately one ton of ice was required to lower the water temperature to the desired level before each swim. Due to the small size of the pool the swimmer was attached to an adjacent wall via a harness and a 2-3 m elastic band. This allowed him to swim against the resistance in the elastic band but did not allow for the control of his work rate. Nor was he able to swim crawl with his head under the water, and so instead he used the breast stroke.

The recovery periods after each swim were of varying duration due to the swimmer's personal time constraints, although each was for a minimum of 20 minutes, during which time his T_{re} response was recorded until it reached a nadir or began to rise.

Prior to the Arctic swims, the subject completed two swims in water at a temperature of 5-6 °C (Figure 1). In the final four weeks prior to the Antarctic swims, the subject completed eight cold water swims at progressively lower temperatures starting at 6.5 °C (Figure 2a) and culminating at 1.5 °C (Figure 2h) during which T_{re} was measured continuously (Figures 2a-h).

All temperature measurements during training were performed with YSI 500 series rectal probes (YSI, Yellow Springs, OH, USA). In addition, oxygen consumption and heart rate were measured during cold water exposure before and after the eight cold water exposures (Figure 3a) and compared to the response of a control long distance swimmer accustomed to swimming at temperatures of 10-16 °C but not to lower temperatures. Metabolic rate was recorded with a portable gas analysis system (K4b², Cosmed).

Measurements during cold water swims

Rectal temperature

During record swims in the Arctic and Antarctic regions, the swimmer inserted the rectal probe and donned the data logging unit approximately 30 min prior to each swim. After securing the various wires and equipment, he was left to prepare mentally for his swim.

Swim distance

Distance was measured with at least two Garmin Global Positioning System (GPS) units. In the Arctic, the distance was marked on the shore and the swimmer swam between the shore markers until the 1000 m was achieved. In the Antarctic, it was not possible to access the

shore at Petermann Island. Swims were measured from the shore to a prominent rock that served as the turnabout point. In the Deception Island swim, the distance was tracked in the boat by three separate GPS systems, in addition to a landmark on the beach that served as a guide for the swimmer and the boat.

Post-swim recovery

Following the training swims the swimmer exited the water and went straight into a waiting car in which the heater had been tuned on prior to his entry. He remained there for at least 20 min. Immediately upon exiting the water during the Arctic and Antarctic swims the swimmer was taken directly from the shore to the ship where he was placed in a hot shower while his physiological variables continued to be monitored. Warming continued until his core temperature had returned to >36 °C. During the re-warming procedure the swimmer had voluntary control of the water temperature in the shower.

Approximately 1-2 min following the Deception Island swim, a temperature probe (YSI 500 Series) was inserted into his right vastus lateralis muscle and left in place until the completion of the re-warming procedures.

Results

Preparatory Training Swims

Basic anthropometry and the results of the DEXA scan before and after the Antarctic training program are presented in Table 2. Body mass was 98.5 kg, of which 22.6% (22 kg) was body fat. Body fat distribution as measured with DEXA showed that the fat was distributed primarily around the trunk (13 kg).

Table 2. Basic anthropometry and percentage body fat

	Height (m)	Mass (kg)	% Body Fat
Pre-training	1.86	97.0	22.5
Post-training	1.86	98.5	22.6

The subject's rectal temperature response to the two swims at 5-6 °C prior to the Arctic swim are presented in Figure 1. In both experiments, the subject entered the water with a rectal temperature of ~38 °C. This temperature was maintained for the first 5-10 min after which it fell more steeply, especially in the first exposure (Figure 1). On exiting the water, his temperature had fallen to 36.7 °C after the first swim but only to 37.7 °C on the second swim. Thereafter his temperature fell more steeply reaching the nadir at 35.8 °C after the first swim and at 36.1 °C after the second swim. The nadir was reached nine minutes after the completion of the first swim and 20 min after the second swim.

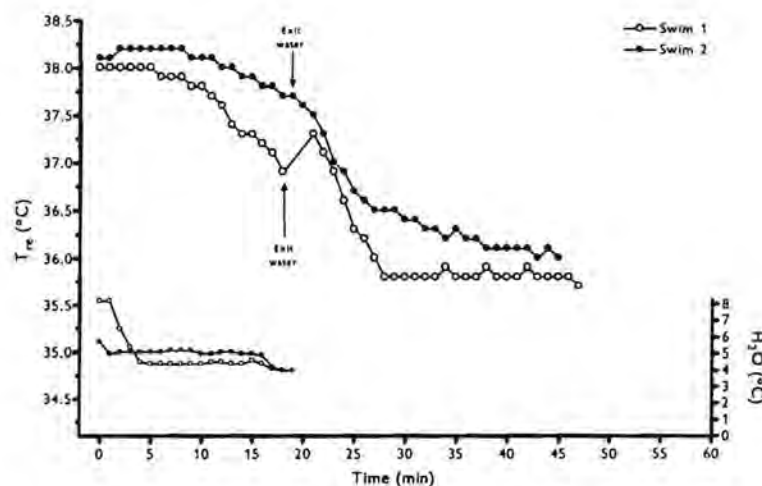


Figure 1. Rectal temperature responses (left y-axis) to two of the swimmer's training swims preceding the Arctic world record. Water temperature is plotted in the right y-axis.

In the eight swims at progressively lower temperatures (6.5 °C to 1.5 °C) before the Antarctic swims (Figures 2a-2h) the same overall pattern was observed as in the training swims before the Arctic expedition. However his temperature on exiting the water and the lowest value measured during recovery both fell with reducing water temperature reaching the lowest values after 22 min swimming at 2 °C, after which his lowest post-exercise rectal temperature was 34.3°C (Figure 2g). When exposed to exercise of four minutes shorter duration at 1.5 °C (Figure 2h), his lowest rectal temperature during recovery was 34.8 °C. It was also notable that during recovery his rectal temperature remained lower for longer in swims at temperatures below 4 °C (Figures 2f-h). In those experiments, the post-exercise rectal temperature was still below 35 °C even 40 min after exiting the water. In addition, there was a trend for his pre-swim rectal temperature to increase from ~37.5°C before the first swim to 38.3°C prior to his final two swims (Figures 2g-h). The swimmer's mean \pm SD pre-swim T_{re} for these eight swims was 37.9 ± 0.3 °C and his mean post-swim T_{re} was 36.5 ± 0.4 °C. The mean lowest T_{re} was 35.0 ± 0.4 °C.

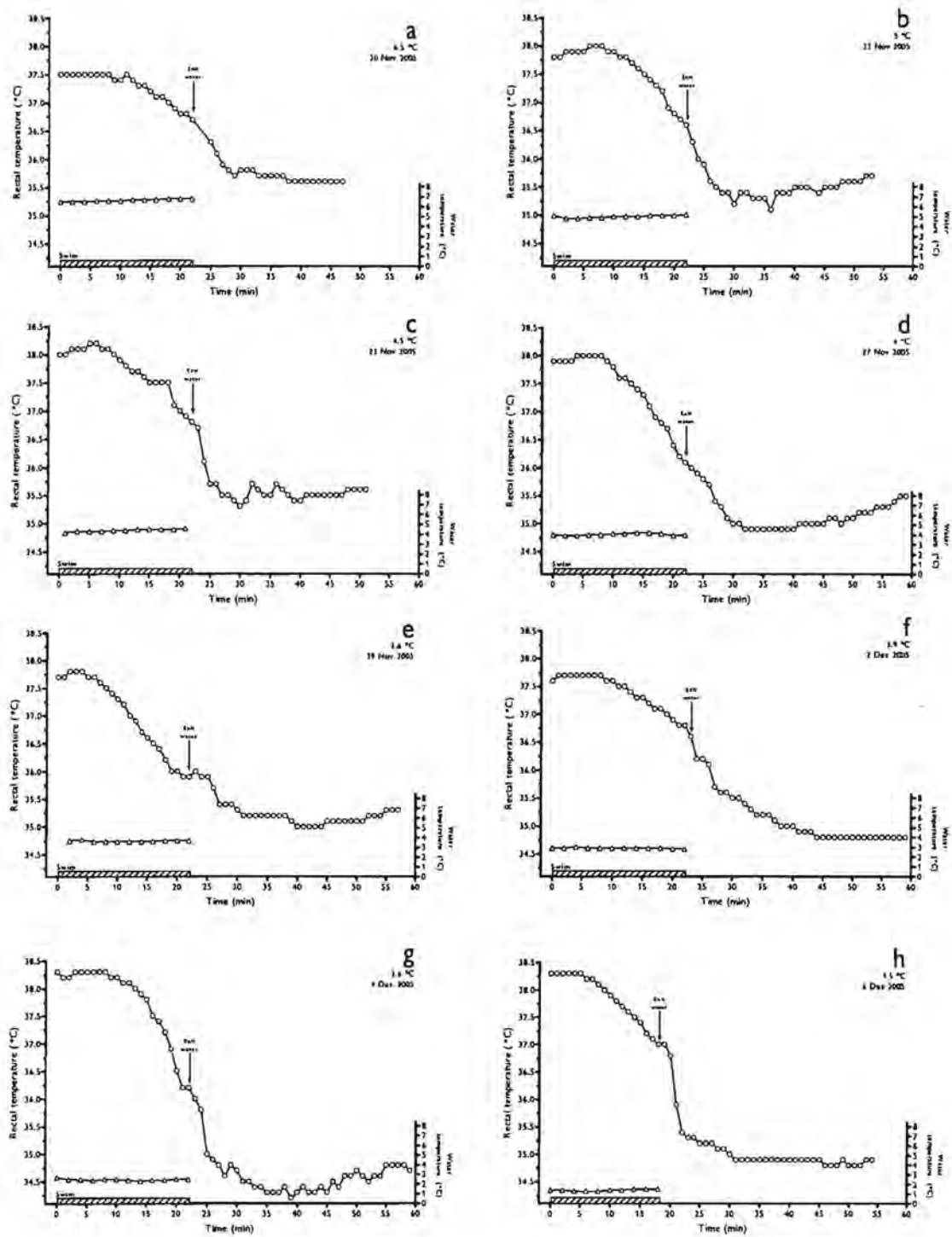


Figure 2. Rectal temperature responses to the eight training swims (a-h) preceding the two Antarctic swims. Swims were separated by at least one day and the water temperature was decreased progressively from swim to swim so that the final swim was completed at 1.5°C . Each swim was of 22 minutes duration except the final swim, which was of 18 minutes duration at 1.5°C .

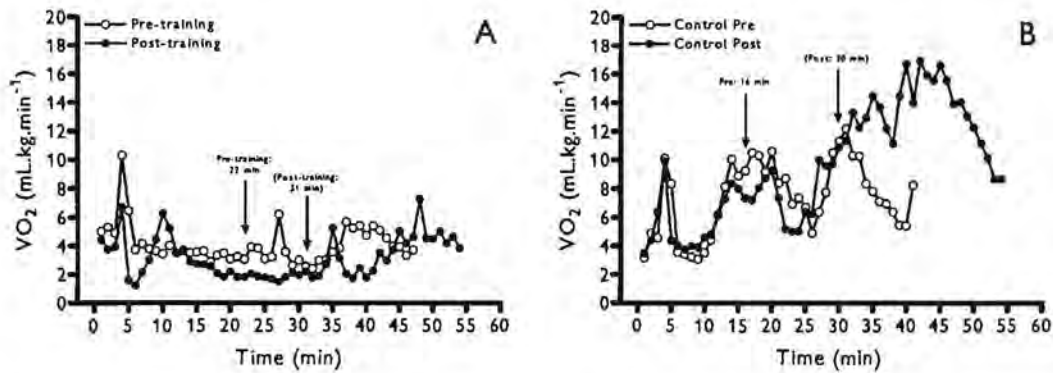


Figure 3. Oxygen uptakes of Pugh (3a) and a control subject (3b) during a cold-water exposure (6°C) both before and after the eight cold water exposures. The control subject was a swimmer but was not exposed to any cold water swims during this time. The duration of each man's exposure was self-determined by each subject. The point at which each man exited the water is noted with the arrows and the corresponding time of the exposure. Note that the duration of each man's second exposure was longer than the first.

During the two passive immersion tests at 6°C , our swimmer maintained a lower metabolic rate compared to the control subject (Figure 3 and Figure 4). While the control subjects O_2 uptake rose gradually during the course of his two immersions, our swimmer's O_2 uptake and HR remained lower at approximately $5\text{ mL.kg}^{-1}.\text{min}^{-1}$ and $70\text{-}90\text{ beats.min}^{-1}$.

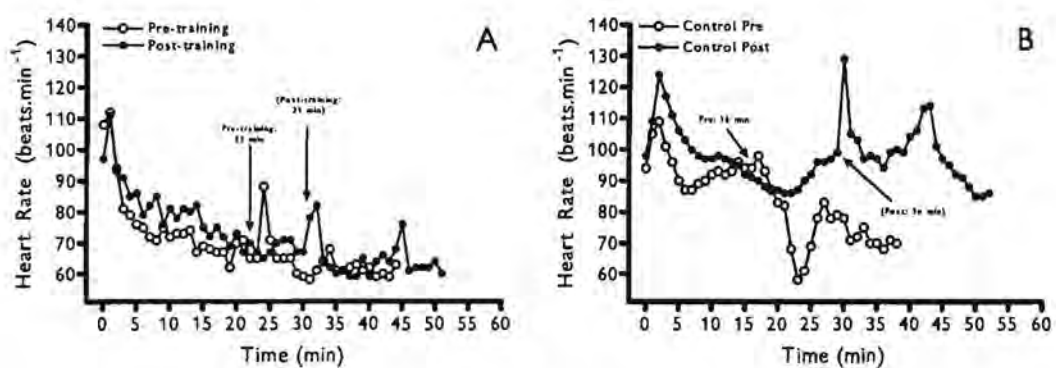


Figure 4. Heart rates of Pugh (4a) and a control subject (4b) during a cold-water exposure (6°C) both before and after the eight cold water exposures. Pugh maintained his HR at a lower level both during and after the immersion test compared to the control swimmer.

The Farthest North and Farthest South Long Distance Swim

On 18 August 2005, the swimmer completed a swim of 1.07 km in 21:30 in the Arctic Ocean at Magdalenefjord, Svalbard, at 79° 00' N, to break his own record (71° N) for the Furthest North Long Distance swim. The water temperature measured at the shore was 3-4 °C. Approximately 12 h later, he completed a second swim of 1.08 km in 20:30, this time at Verleggenhuk on the northern end of the island of Spitzbergen (80° 03' N) in water at a temperature of 3 °C.

On 14 December 2005 the swimmer set the record for the Furthest South Long Distance swim by completing a swim of 1000 m in the Southern Ocean in 18:12 at Petermann Island (65° 10' S) off the Antarctic Peninsula in water of 0 °C. His rectal temperature responses to both these swims are shown in Figure 5.

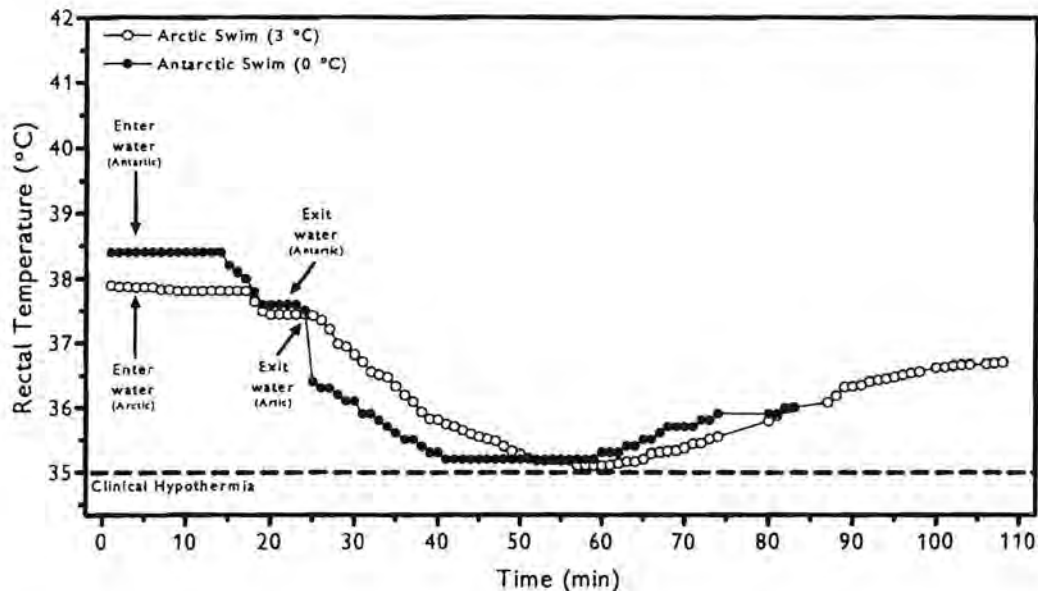


Figure 5. Rectal temperature responses during two world-record long-distance swims in the Arctic (80° 03' N) and Antarctic (65° 10' S). The swimmer took 20:30 to complete the 1080 m swim in 3 °C water in the Arctic and 18:12 to complete the 1000 m swim in 0 °C water in the Antarctic.

In both swims he entered the water with an elevated rectal temperature—37. °C before the Arctic swim and 38.4 °C before the Antarctic swim. Temperatures remained at those elevated values for the first 14 min of the Arctic swim and for the first 10 min during the Antarctic swim, falling to temperatures of 37.4 °C and 37.5 °C after the Arctic and Antarctic swims, respectively. Thereafter there was an essentially identical after-drop which reached a nadir at 35.1 °C at 26 and 19 min after the subject exited the water in the respective swims. The plateau continued for a further 11 and 17 minutes after the respective swims before beginning to rise. The rate of rise from the nadir to a new stable level >36 °C (Arctic = 0.03 °C·min⁻¹, Antarctic = 0.02 °C·min⁻¹) was slower than the rate at which the temperature fell to reach the nadir after exiting the water (Arctic = 0.07 °C·min⁻¹, Antarctic = 0.13 °C·min⁻¹).

The Longest Duration Polar Swim – Deception Island

On 17 December 2005, the swimmer swam a distance of 1650 m in 30:30 in 2-3 °C water at Deception Island (South Shetland Islands 62° S). His pre-swim temperature was 38.4 °C (Figure 5), and his temperature remained at that value for approximately 10 min before falling progressively until he exited the water with a T_{re} of 36 °C. His T_{re} then dropped precipitously to 33.6°C within 14 min of exiting the water (0.17 °C·min⁻¹). It then remained at that value for 5-6 min before rising gradually by 0.13 °C·min⁻¹ for 26 min until his T_{re} reached a plateau at 37 °C. He remained in the shower for an additional 40 min and voluntarily left the warm shower at that time.

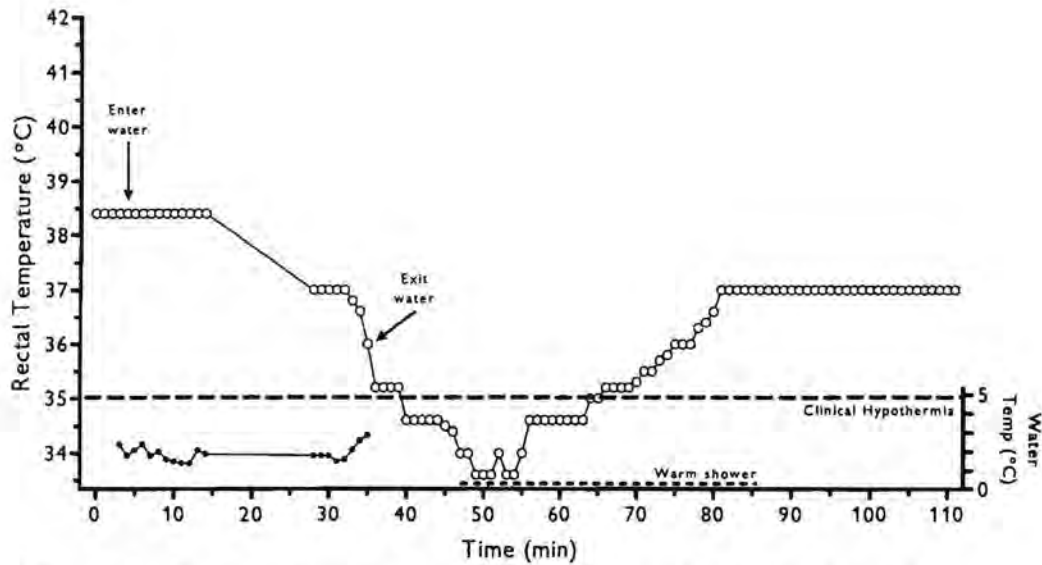


Figure 5. The rectal temperature response to a 1650 m swim across Whaler's Bay at Deception Island in water at 2-3 °C on 17 December 2005. The lowest rectal temperature was 33.6 °C and was reached approximately 14 min after the swimmer exited the water.

Muscle temperature (Figure 6) immediately following the swim was 31.6°C and decreased to 30.4° 40 min after exiting the water despite the fact that the subject had been in a hot shower for 30 min during that time. After a further 10 minutes, his muscle temperature began to rise but was still only 31.5°C at the end of the re-warming period 70 min after he had exited the water at the time when his core temperature had returned to 37°C.

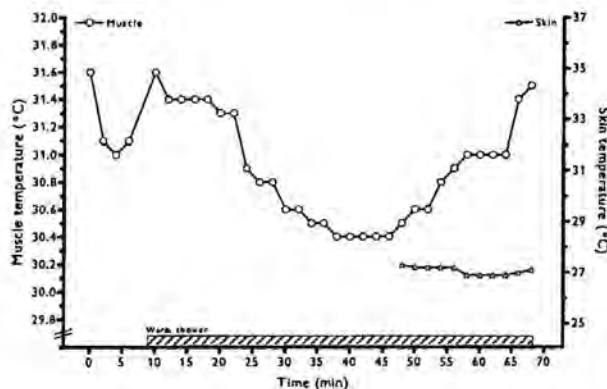


Figure 6. Muscle temperature (left y-axis) and skin temperature of the vastus lateralis following the 1650 m swim on December 17, 2005, across Whaler's Bay at Deception Island in 2-3 °C water. Time "0" is immediately following the swim when the muscle temperature probe was inserted. From 10 minutes following the swim the swimmer was seated in a warm shower, where he remained for the duration of the recovery.

Discussion

This swimmer completed successfully three long distance swims of 1-1.6 km in times ranging from 18-30 min in water at temperatures of 0-4 °C without becoming incapacitated by either hypothermia or hyperventilation, although he showed a marked afterdrop in his core temperature, especially after the 1.6 km swim in 2-3°C water. These performances exceed by a factor of about four those predicted for persons exposed to similarly cold water when fully clothed and wearing a life jacket (113). This swimmer's unusual ability appears to be due to at least three factors.

First, prior to all events including the training swims in Cape Town (Figures 1 and 2), the subject was able to raise his body temperature to between 37.8-38.4 °C without engaging in physical activity. This response appears to have developed progressively during the course of his ten training swims, reaching its maximum in the two final pre-Antarctic swims (Figures 2g-h) and the two Antarctic swims (Figures 4 and 5). Since three independently-calibrated and different measurement systems were used to measure his rectal temperature in the training swims and in the Polar swims and since all three recorded identical results, a measurement error cannot explain this finding.

This adaptation suggests the presence of an anticipatory thermogenesis associated with an acute elevation of his body temperature set point. Thermogenesis alone would not produce a sustained elevation in core body temperature if the thermal set point was not also elevated. Following the swims his set point seems to have returned to ~37.0 °C as this was the highest temperature measured during 65 minutes of recovery in a hot shower (Figure 5).

To our knowledge, an anticipatory elevation of rectal temperature of this magnitude has not previously been reported (1; 23; 107; 244; 257). Pugh and Edholm (194) reported that the

accomplished Channel swimmer, Jason Zirganos, had immediate pre-exercise rectal temperatures of 37.8 and 37.6 °C prior to two swims in Lake Windermere at water temperatures of 13.8-15.8 °C. In addition, one subject in the study of Huttunen et al. (2000) maintained a rectal temperature in excess of 38 °C during swims of 20-30 min in water of 10-14 °C, suggesting an elevated temperature set point both before and during the swim.

Lesna et al. (127) have reported that the resting metabolic rate of habituated cold water swimmers ("polar swimmers") increased by 45% in response to epinephrine infusion—an effect that was 83% greater than the response measured in control subjects. The effect of these infusions on the T_{re} was not reported, however. It is currently believed that epinephrine-induced thermogenesis occurs in human skeletal muscle and white adipose tissue (13; 217), and a similar response has been identified in birds (22). Thus epinephrine-induced thermogenesis associated with an elevated core temperature set point might explain why our subject was able to enter the water with a markedly elevated core temperature despite the fact that he was not shivering and had not engaged in strenuous physical activity.

Interestingly, a pre-exercise elevation in core temperature produced by either external heating or physical activity is usually considered disadvantageous for subsequent cold water exposure in which no physical activity is performed. Passive warming prior to cold water exposure causes has been shown to produce a more rapid fall in core temperature during cold water exposure than occurs without such warming (212). However the effect is lessened when physical activity is used to raise the body temperature before exercise (212).

However that study was performed at rest, not when swimming, and in somewhat warmer water (7 °C). Furthermore esophageal temperature began to fall immediately when the sub-

jects entered the cold water. Our subject was able to maintain his elevated rectal temperature for some time even when his head was immersed in the ice-cold water (Figures 4 and 5) whereas the subjects in the study by Scott et al. (212) did not. Our presumption therefore is that the ability of our subject to raise his body temperature before exercise was beneficial since it allowed him to maintain his core temperature at a higher level for longer than might have been the case otherwise.

Second, the subject was able to maintain his elevated core temperature for between 10 - 15 min while swimming in water at temperatures of 0-4 °C (Figures 4 and 5). Even during his longest swim of 30 min in water of 2-3 °C, his rectal temperature fell by only 2.4 °C during the swim and most of the fall occurred in the last 15 minutes of the swim. In contrast, as already argued, unacclimatized subjects exposed to water below ~10 °C usually show a progressive fall in rectal temperature beginning immediately when the immersion begins, even when they do not swim (212).

There are a number of mechanisms that could explain this unexpected ability. The subject might produce more heat than expected when he swims either as a result of an inefficient swimming technique. Or the subject might have continued high rates of epinephrine-induced thermogenesis during swimming (244). Alternatively, this might be because of superior insulation, particularly of his body core. Pugh and colleagues (194) concluded that the important factor explaining the success of Channel swimmers was the "thermal insulation of their subcutaneous fat". Our data lead us to suggest that an additional factor when swimming in ice-cold water would be a superior ability to protect the core temperature by physiological means while allowing the limbs to cool to very low values.

The vastus lateralis muscle temperature measured immediately after the 1.6 km swim was 31.5 °C, between 2.5 and 4.5 °C lower than the core temperature measured simultaneously (Figure 5). Pugh and Edholm (194) also reported that the temperature of Zirganos's forearm and thigh muscles was ~29 °C, 2.5 cm below the skin surface after a swim of 6:49 (h:mm) at 15.8 °C. The deep (4 cm) forearm muscle temperature was 2 °C lower than the rectal temperature. In contrast, the deep thigh temperature (6 cm below the skin) was higher than the rectal temperature. Pugh and Edholm also reported that the deep muscle temperature "fell rapidly when muscular activity ceased" (p. 762) suggesting an immediate reduction in muscle perfusion on the cessation of exercise. When interpreted with the findings of Pugh and Edholm, our findings suggest that an important adaptation to extreme cold water swimming must be the ability to restrict blood flow to the superficial limb muscles that cool rapidly on cold water immersion while at the same time maintaining (just) sufficient nutritive blood flow to the deep, more insulated portions of those muscles.

The extent to which whole-body cooling had occurred in our subject is confirmed by the marked post-swim afterdrop in his T_{re} (Figures 1, 2, 4, 5) and the prolonged period that was required for his core temperature to return to >36 °C despite exposure to a hot shower and high humidity (Figures 4 and 5). Indeed, even after 75 minutes in a hot shower following the Deception Island swim, his vastus lateralis muscle temperature was still only 31.5 °C, the same as it was when he exited the water, and his skin temperature was only 27 °C (Figure 5). This confirms the extent to which his peripheral limbs had cooled while he had been able to "defend" his core temperature for up to 30 minutes during exposure to water at 2-3 °C.

Thus the key findings are that our subject did not prevent significant whole-body cooling when swimming at 0-3 °C. Rather he was able to prevent excessive cooling of his core even as his muscle and skin temperatures fell markedly below the core temperature. Thus we conclude that a key adaptation allowing this swimmer to complete safely these swims was his ability to insulate his core for up to 30 minutes in ice-cold water while especially his legs cooled to very low temperatures. Interestingly this human response in protecting the core during exposure to ice-cold water contrasts with that of fishing birds like the poorly-insulated cormorants, which increase the time they can swim in cold water by allowing their core temperatures to fall progressively by up to 5 °C when swimming in ~13 °C water for five minutes (254). Cormorants also show a marked (“anticipatory”) increase in body temperature immediately before entering the water and while still on their nests (254). In contrast, well-insulated penguins increase their body temperatures while swimming in cold water and do not show “anticipatory thermogenesis” (254).

We therefore conclude that while increased whole body insulation is clearly important to ensure success by preventing heat loss from the body during cold water swimming, perhaps a more crucial factor must be the ability to insulate the core despite extensive cooling of the periphery by maintaining just sufficient nutritive blood flow to the limbs to ensure their function without the progressive loss of muscular function described by Tipton (Tipton – Lancet).

Third, the subject was able to dive directly into water as low as 0 °C and to begin swimming with his normal crawl stroke and his head under water immediately without any apparent evidence for the cold-shock response (113; 237). Tipton and colleagues (79; 237; 238) have shown that repeated exposure to cold water can produce habituation which may be retained for months without subsequent cold exposure. However, subjects in those trials were ex-

posed to cold water (showers) for only three minutes. The performances reported here suggest that repeated exposure to ice-cold water at 1-6 °C (Figure 2) can produce an adaptation that enhances one's ability to defend the core temperature for up to 30 minutes of swimming in ice-cold water.

When exposed to cold water (6 °C) in which he did not swim, our subject developed a hypometabolic response, maintaining his oxygen consumption at about 5 mL O₂·kg⁻¹·min⁻¹ (Figure 3a). This was in contrast to a long distance swimmer who was unconditioned to swimming in ice-cold water, who developed a shivering response in which the rate of oxygen consumption increased progressively before falling for the final 10 minutes after exiting the water (Figure 3b). This is similar to the more normal response on exposure to warmer (>20°C) water. Jansky et al. (107) have shown that the typical response to repeated exposure to cold water (14°C) in which subjects did not exercise is "hibernatory," meaning that core and peripheral body temperatures are reduced as a result of a reduced cold sensation and a lowering of the threshold at which cold-induced thermogenesis began. Since our subject showed this hypometabolic response to cold water exposure as well as what appears to be a heightened thermogenesis prior to entering the water, it appears that there may be different thermogenic adaptations to either passive or active exposure to ice-cold water.

Fourth, an unexpected finding was the pattern of core and muscle temperature recoveries after the swim. Figure 5 shows that the afterdrop in core temperature reached its lowest value 14 min after our subject exited the water whereas the vastus lateralis muscle temperature reached its nadir 45 minutes after water exit. Furthermore whereas the core temperature had returned to 37 °C at 47 min after exiting the water, the muscle temperature at that time was still only 31.5°C, which was similar to the value measured on water exit. Indeed

only when the core temperature had returned to 37 °C at 47 min after water exit did re-warming of the leg muscles commence (Figure 5).

This indicates that the core temperature is “protected” not only during exercise but also during recovery. It was also notable that after exercise no attempt was made to increase the core temperature to the pre-swim value of ~38 °C. Rather the core temperature seems to have been reset back to ~37 °C as peripheral re-warming commenced only after that core temperature had been reached (Figure 5).

In conclusion we believe that our data show that our subject was at the limits of his thermal tolerance after 30 minutes and 30 seconds swimming at 2-3 °C. Thus, shortly after exiting the water following the Deception Island swim, his core temperature dropped precipitously by 2.4 °C in 14min (Figure 5), the most abrupt fall in core temperature ($-0.17\text{ °C}\cdot\text{min}^{-1}$) that we measured in any of our trials (Figures 1 and 2) but similar to the response after the trial swim at 1.5 °C ($-0.16\text{ °C}\cdot\text{min}^{-1}$) (Figure 2h).

We further conclude that this immediate afterdrop to such a low temperature (34 °C) suggests that at that point our subject was likely at the limit of his cold tolerance and that this sudden drop was caused by (i) the reduction in heat production by his swimming muscles with (ii) an abrupt failure of his unusual capacity to insulate his core by restricting blood flow to his cold limbs. Unrestricted perfusion especially of the superficial and extremely cold portions of the limb muscles would cause convective heat loss from blood with return of cooled blood to the body core as argued by Giesbrecht and Bristow (70).

Cold-induced vasodilation in arterioles can occur. It is possible that this critical temperature at which this vasodilation occurs might have been reached in the subject's superficial muscles. The subject's skin colour was pink on exiting the water, perhaps indicating the presence of cold-induced vasodilation in his cutaneous circulation. We further speculate that if our subject had continued his cold water exposure for even a few more minutes, it is possible that a failure of limb arteriolar vasoconstriction might have occurred causing an abrupt fall in core and brain temperature leading to the sudden loss of consciousness in the water. The danger of a sudden drop in temperature even in those who have reached land, has been emphasized (183).

A particularly disturbing fact was that our swimmer was still swimming reasonably well at this point in that he demonstrated no signs of stopping. The sole indication of his altered state was an apparent change in his mental state as after 1400 m of the Deception Island swim he no longer responded visibly to commands given from the boat travelling with him. This contrasts to studies of either non-swimmers or swimmers unacclimatized to cold water (236). In both of these populations, exercise is limited by muscle failure in the absence of profound hypothermia. Rather it appears that our subject had adapted his muscles to cold water swimming to the point where they would have continued functioning effectively beyond the point at which his core temperature was too low for continued consciousness.

Our contention therefore is that, as a result of his extreme adaptation to ice-cold water, our swimmer had developed the capacity to swim himself to unconsciousness in ice-cold water. We argue that had he attempted to stay in the water at 2-3 °C for even a few minutes longer than 30 min, he would likely have shown a sudden drop in core temperature with the likely sudden onset of unconsciousness.

Our data do not allow us to believe Glaser's optimistic conclusion that a man swimming in ice-cold water "should not die of cold as long as he is able to swim" since his rate of heat production should always match or exceed his rate of heat loss under those conditions. Our experience also makes us question the epochal story of the man who survived in water at -1.5°C for "half a day". The published evidence also tends to rule out this possibility.

Rather we suggest that the limit of tolerance for swimming in ice-cold water was approached by our subject in the Deception Island swim when he swam 1650 m in 30:30 in water at $2-3^{\circ}\text{C}$.

Summary

In summary, we report the first series in which the T_{re} was measured in a subject who voluntarily swam in water at temperatures less than 5°C for up to 30 minutes.

The main findings were that the swimmer entered the water with a substantially increased core temperature which he was able to maintain for up to 10-15 minutes even when swimming in water of 0°C . Maintenance of core temperature sufficient to retain consciousness occurred despite severe whole body cooling which was not corrected even after more than 60 minutes exposure to a hot shower. Re-warming of the limb muscles occurred only after the core temperature had returned to 37°C .

It appears that the limits of his cold tolerance was reached in the 1650 m swim at $2-4^{\circ}\text{C}$ after which his core temperature dropped precipitously by 2.4°C to 33.6°C within 14 min of stopping swimming (Figure 5). This suggests that his ability to defend his core temperature

whilst swimming was approaching its limit. A sudden and catastrophic loss of core temperature regulation secondary to extensive cooling of the limbs might explain the sudden loss of consciousness in some long distance swimmers.

It appears that extensive biological adaptations had allowed our swimmer to limit his hyper-ventilatory response to cold water exposure and to maximize the resistance of his exercising muscles to the effects of cooling to the point where he is likely capable of swimming himself into a state of hypothermic unconsciousness.

By overcoming the two principal factors that limit exercise time in the cold, we suggest that our subject had reached the safe limits for swimming in ice-cold water. We therefore suggest that such limits at water of 2-4 °C are about 30 min swimming or about 1.6 km in highly adapted swimmers. In water at 0 °C, the limits are likely less but still more than one km (Figure 5).

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Chapter 9

Summary and conclusions

Introduction

The broader aims at the outset of this thesis were to investigate the relationship between fluid ingestion and temperature regulation during endurance exercise, and to examine more closely the temperature responses to endurance exercise and performance. More specifically, emphasis was placed on investigating the fluid replacement strategies and the rectal temperature responses to endurance exercise as performed during locally organized and staged endurance races.

An unexpected development during the latter stages of this thesis that was not anticipated at the outset was the work with the cold-water swimmer in Chapter 8. This work, however, added another dimension to the thesis by providing evidence that helps demonstrate the robustness of the human thermoregulatory system.

Research Questions

Answers to the research questions which were asked at the beginning of this thesis are provided below.

Question 1

What are the fluid replacement strategies and post-race plasma $[\text{Na}^+]$ concentrations in endurance cycling?

Although the post-race serum $[\text{Na}^+]$ to marathons and ultra-distance triathlons has been well described, no data were available to describe the post-race $[\text{Na}^+]$ in cycling. Chapter 2 showed that at least this local population of cyclists had a normal and healthy response to endurance cycling since our cohort of finishers completed the race with weight losses of 1.5-2.5% and mild hypernatremia ($141.8 \pm 2.9 \text{ mmol}\cdot\text{L}^{-1}$). The novel finding of this study was that although the women ingested similar volumes of fluid compared to the men, due to their lower mass and therefore lower sweat rates they replaced a larger percentage of their fluid losses. The effect of this was that the women's post-race $[\text{Na}^+]$ was significantly lower com-

pared to the men. These data may help to explain why women present more often with hyponatremia following endurance events compared to men.

Although the neuro-endocrine response in these athletes was not studied, available evidence shows that there are no differences between men and women in the thirst threshold or regulation of plasma osmolality. The fact that women replaced a larger percentages of their weight losses can therefore be explained by behavioral instead of physiological differences. This might help to explain why women present more often with EAH, for if they are ingesting similar volumes of fluid compared to men in all endurance races then since they have smaller masses they will naturally be replacing larger percentages of their weight losses.

Question 2

What are the rectal temperature responses to endurance exercise during actual running and cycling races, and how critical are these temperatures with regards to "heat illness?"

The technology to record continuous temperature values exists in environmental and other life sciences. However it has been largely under utilized in the Exercise Sciences. By taking advantage of this technology we were able to produce two novel studies that showed the continuous temperature responses to both ultra-marathon running and endurance cycling. The data from Chapters 4, 5, and 7 all confirm the conclusions of the early investigators as discussed in the Literature Review. In recreational (Chapter 3) and more competitive (Chapter 4) athletes the T_{re} rises for approximately an hour before reaching a plateau, after which time it is regulated at a higher level in such a way that it remains within a very narrow range.

One exception to this scenario is when an athlete is competing in an individual time trial as in Chapter 7 (Addendum 2). In this situation the T_{re} response is a sigmoidal curve, and the athlete appears to reach the peak T_{re} at the end of the race. This response is similar to that

shown in Chapter 5, in which the subjects were completing 80 km cycling time trials for the best possible result.

In these local endurance events the T_{re} responses were far below any value that might be associated with heat stroke, even in the one athlete who ran a 21.1 km race in <80 min and finished with a T_{re} of 40.3 °C. The ambient conditions were moderate (≤ 20 °C) and solar radiation was low since the races started in the early morning hours of the day. However this is almost always the case in recreational races throughout the world. It is only professional races that have start times after 0800. Except the Boston Marathon which traditionally has a mid-day start, recreational marathons start in the early morning hours. Therefore the evidence from this thesis suggests that the vast majority of recreational athletes completing endurance races in either cool or moderate conditions are unlikely to achieve T_{re} 's above 40.0 °C, and this is further supported by the low incidence of heat stroke in endurance races throughout the world.

Question 3

How does fluid ingestion effect temperature regulation during performance in hot and humid environments?

The effects of fluid ingestion on temperature regulation during fixed-workload exercise is well documented, but its effects on performance longer than one hour is not well described. Chapter 5 investigated this in depth by using a range of fluid volumes to test the influence of the ingested fluid volume on temperature regulation and 80 km time trial performance. The novel and interesting finding from these data was that fluid ingestion affected performance, but not in a graded manner. There was no difference between the six experimental trials in which the subjects ingested varying amounts of fluid so that the subjects did not cycle faster when replacing increasingly higher volumes of fluid. This conflicts with current drinking guidelines and models of fluid replacement which advise athletes to replace 100% of their sweat

losses during prolonged exercise to 1) enhance temperature regulation and 2) improve performance, since performance is thought to be affected negatively by increasing core temperatures. However the data in this thesis and from previous studies suggest that the ability to elevate core temperature and tolerate core temperatures $>39^{\circ}\text{C}$ is perhaps a prerequisite for superior exercise performance.

The effect of fluid ingestion on performance was detected only when the data in Chapter 5 were analyzed in two larger groups consisting of either "HI" (*ad libitum* or greater) or "LO" (33% or less) fluid intake. The subjects did cycle faster in the HI group, suggesting that fluid restriction below *ad libitum* volumes can affect performance. This emphasizes the importance of fluid ingestion during exercise but simultaneously illustrates that larger volumes of fluid are not necessary to achieve the best performance.

The effects of fluid ingestion on temperature regulation during a performance trial are much less profound. The evidence from this thesis suggests that the brain compensates for any effect dehydration has on temperature regulation by adjusting the metabolic rate (and hence the heat production) so that an individually suitable T_{re} is achieved at the end of each trial, independent of the amount of ingested fluid. Furthermore, there appears to be very small intra-subject variation in the T_{re} response to this type of exercise, as indicated by the very low post-trial T_{re} intra-subject CV.

Question 4

Is there a graded effect of dehydration on exercise performance in the heat so that ingesting more fluid will enhance time trial performance?

The data from Chapter 5 show that dehydration does not effect performance in a graded manner in the same way that it affects temperature regulation. Instead, the brain appears to

control the metabolic rate in such a way to produce a highly repeatable post-exercise T_{re} (intra-subject CV $0.72 \pm 0.27\%$). There is an effect of fluid ingestion on exercise performance so that ingesting less than *ad libitum* volumes appears to reduce performance, while ingesting more than *ad libitum* does not appear to have any added benefit on performance or T_{re} .

Considering that the athletes in Chapter 5 were highly-trained and motivated, the data suggest that in hot environments the risk for "heat injury" is low, even when weight losses are in excess of 4%, since the mean post-exercise T_{re} in all conditions and subjects was 39.1 ± 0.5 °C. Two reasons explain this. First, the metabolic rate has been shown to be the best predictor of T_{re} , and therefore small alterations in metabolic rate will produce a large effect on the core temperature. Second, as mentioned above, the brain appears to compensate for any effect of dehydration on temperature regulation by lowering the metabolic rate and therefore limiting the amount of work that an athlete can do, the result of which is a similar T_{re} response even though performance time is slower.

Summary

The collective body of data from this thesis illustrates that the human body has a large capacity to cope with fluid losses in moderate and hot environments. This contrasts with current models used to explain temperature regulation, which suggest that thirst is an inaccurate mechanism and that because of this the human body is exposed to increasing danger as weight losses increase. However, the data from Chapter 5 in particular illustrate that although dehydration may have an effect on temperature regulation, the physiological response to increasing levels of weight loss is to reduce heat production so that a critical core temperature is never reached. The result of this is that the core temperature is regulated successfully at a higher level during exercise, which is a concept first described in 1938.

The data in this thesis demonstrates also that the human body has a large capacity regulate its temperature not only during dynamic endurance exercise in moderate and hot conditions, but even in extremely cold water. During running and cycling the body stores heat for approximately one hour before the T_{re} reaches a new level, after which time it remains within a very narrow range. The data from Chapter 8 suggest that the body makes specific adaptations to repeated and extreme cold-water exposure so that it can better defend its core temperature even in very low (0-4 °C) water temperatures.

Taken together with the data from Chapter 8, this thesis also illustrates that the human body can cope with thermal challenges both cold and hot in nature. Chapter 8 shows that following a progressive training program and the rule of specificity will produce training adaptations that will permit a person to cope with even the most extreme cold for relatively long periods of time (20-30 min). This supports other scientists in this area that have concluded that, as homeotherms, humans have vast amounts of heat in their bodies, thus allowing them to survive in relatively cold water for exposures of this duration of time. Finally, all of these data help to illustrate how robust the human body's temperature regulation mechanisms are, and that the body can regulate successfully its temperature in a wide range of situations and environmental conditions.

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CASE REPORT

Hyponatraemic encephalopathy despite a modest rate of fluid intake during a 109 km cycle race

J P Dugas, T D Noakes

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Objective: To report a case of exertional hyponatraemic encephalopathy that occurred despite a modest rate of fluid intake during a 109 km cycling race.

Methods: Men and women cyclists were weighed before and after the race. All subjects were interviewed and their water bottles measured to quantify fluid ingestion. A blood sample was drawn after the race for the measurement of serum Na⁺ concentration.

Results: From the full set of data (n = 196), one athlete was found to have hyponatraemic encephalopathy (serum [Na⁺], 129 mmol/l). She was studied subsequently in the laboratory for measurement of sweat [Na⁺] and sweat rate.

Conclusions: Despite a modest rate of fluid intake (735 ml/h) and minimal predicted sweat Na⁺ losses, this female athlete developed hyponatraemic encephalopathy. The rate of fluid intake is well below the rate currently prescribed as optimum. Drinking to thirst and not to a set hourly rate would appear to be the more appropriate behaviour.

There is now a near complete international consensus that hyponatraemic encephalopathy develops in subjects who ingest excessive volumes of fluid during exercise and who gain weight, as a result developing "water intoxication".^{1,2} Accordingly, fluid-ingestion guidelines recently accepted by the United States Track and Field³ suggest that athletes should drink ad libitum during exercise—that is, according to thirst—as this biologically driven approach, adopted naturally by all creatures other than exercising humans who have been taught otherwise, will probably prevent too little or too much being drunk.

However, the apparent reluctance to accept that a basic physiological drive can produce safe drinking behaviours in humans, as it does in all other creatures, continues to underpin advice that athletes must be provided with specific guidelines for rates of fluid intake during exercise. Therefore the guidelines of the United States Track and Field suggest that rates of fluid intake during exercise should be 400–800 ml/h,³ although others advise rates as high as 1000 ml/h.⁴ These values are, however, substantially less than rates of 1200 ml/h advocated by the American College of Sports Medicine⁵ and of up to 1800 ml/h originally proposed by the United States Army.⁶

Yet there are examples of athletes who have developed water intoxication during exercise despite drinking fluid at rates substantially lower than these currently prescribed rates. Speedy *et al.*⁷ reported the development of hyponatraemia in two Ironman triathletes whose estimated rates of fluid intake were 733 and 764 ml/h during 13.3 and 12 hours of exercise respectively. These rates fall within the range 400–1000 ml/h currently advocated.^{3–5} Glace *et al.*⁸ reported that athletes who ingested fluid at a rate of 700 ml/h developed gastrointestinal symptoms during a 160 km trail race,

whereas Stuempfle *et al.*¹³ reported a reduction in serum sodium concentrations ([Na⁺]) in athletes completing a 100 mile Iditarod ultramarathon completed in cold conditions in Alaska, even though they drank only 200–400 ml/h. Similarly, Twerenbold *et al.*¹⁴ reported a mean decrease in serum [Na⁺] of 6.2 mmol/l in women who ingested 1000 ml/h of water for four hours when covering 40 km at a pace equivalent to a 4:15 (hours:minutes) 42.2 km marathon. This rate of fluid ingestion was twice the sweat rate of these athletes; as a result the athletes gained 2 kg during the four hours that they ran.

These data therefore indicate that medical complications can occur in athletes who drink at rates that are less than the maximum "safe" values currently proposed by various authorities.

As part of a cross sectional study of 193 athletes in the 2003 109 km Cape Argus/Pick 'n' Pay Cycle Tour in Cape Town, South Africa, which attracts more than 35 000 participants annually, we identified one subject who developed mild hyponatraemic encephalopathy with a serum [Na⁺] of 129 mmol/l after the race, despite a relatively modest rate of fluid intake during the race. Here we report the physiological basis for the development of her condition. This case study adds further to the evidence showing that drinking to a fixed rate in excess of thirst can produce medical complications even when the rate appears quite modest.

CASE REPORT

A 57 year old female cyclist who had previously completed the 109 km cycle tour on 16 occasions (best time 3:57) volunteered to participate in a study of fluid balance and serum [Na⁺] during the race according to previously described methods.^{15,16}

Environmental conditions during the race were the following: at 0600 the ambient temperature was 18.3°C and the relative humidity was 99%; at 0900 the ambient temperature was 19.6°C and the relative humidity was 99%; at 1200 the ambient temperature was 21.7°C and the relative humidity was 77%; at 1600 the ambient temperature was 23.4°C and the relative humidity was 75%.

After completing the race in 5:10, the subject complained of a headache, light headedness, and difficulty concentrating. She had experienced these symptoms previously on numerous occasions after rides of >100 km and in at least one 42.2 km marathon some 20 years earlier.

She started the race with a weight of 66.6 kg. Her total fluid intake during the race was 3800 ml (735 ml/h). Her weight after the race was 69.0 kg, which indicated a total weight gain during the race of 2.4 kg (3.6% of body weight). To account for this weight gain, her estimated sweat rate during the race would have been about 270 ml/h. Her serum [Na⁺] after the race was 129 mmol/l, and she was the only athlete in our trial of 196 cyclists who developed a serum [Na⁺] less than 135 mmol/l (fig 1) and the only athlete in the

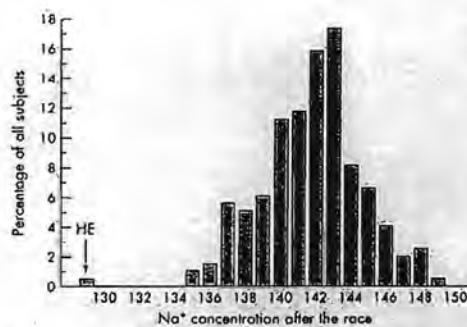


Figure 1 Distribution of serum $[Na^+]$ after a 109 km race in 196 cyclists (103 men and 93 women). The cyclist described in this study is identified as HE (hyponatraemic encephalopathy) and was the only one who developed hyponatraemia (serum $[Na^+] < 135$ mmol/l).

study to develop symptoms of hyponatraemic encephalopathy.

As in all our and other previous studies,^{2, 4, 18-19} an inverse linear relationship was found between the change in mass during exercise and serum $[Na^+]$ after the race, so that those athletes who lost the least weight during the race had the lowest serum $[Na^+]$ after the race (fig 2). Furthermore, the subject in this case report ("HE" in figs 1 and 2) was the only cyclist to gain a substantial amount of weight.

Two weeks after the race, this athlete performed a series of laboratory experiments for measurement of her sweat rate and the $[Na^+]$ of the sweat collected from her back, arm, and leg. When cycling in a specially designed environmental chamber²⁰ at a rate of 120 W in ambient conditions of 28.5°C, 55% humidity, and with a facing air velocity of 10 km/h wind speed, her sweat rate was 520 ml/h and the sweat $[Na^+]$ on her back, arm, and leg were 73, 68, and 64 mmol/l respectively (average of 68 mmol/l). Therefore total Na^+ loss in sweat during the race was probably only 70-105 mmol, similar to values either measured^{18, 21} or predicted²¹ in other athletes who developed this condition during prolonged exercise.

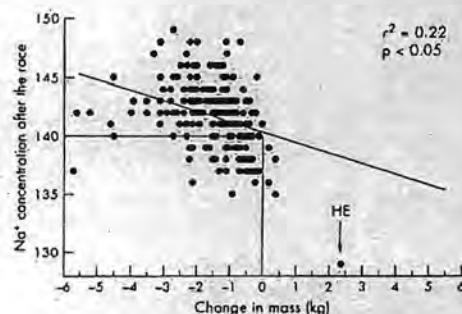


Figure 2 Relationship between change in mass and serum $[Na^+]$ in 196 cyclists (103 men and 93 women) who completed a 109 km cycle race. The cyclist who gained the most weight is identified as HE and was the only one to develop hyponatraemia.

On the basis of the finding that her condition was due to voluntary overhydration without a substantial Na^+ deficit, she was advised that in future she should always drink ad libitum during exercise.

On follow up after the 2004 Cape Argus/Pick 'n' Pay 109 km Cycle Tour, she reported that her ad libitum fluid intake rate had been not more than 500 ml/h. Her race time was 5:38 for the 109 km course, which was similar to her time of 5:10 in the previous year. She reported being symptom-free after the race, as well as in other rides >100 km completed in the 12 months since she had been evaluated in the laboratory. In fact, she enthusiastically stated that her enjoyment of cycling had increased substantially since she began to drink less fluid during prolonged exercise.

DISCUSSION

The only cyclist to develop hyponatraemia in our prospective study of 196 cyclists in this race was also the only one to gain a substantial amount of weight (2.4 kg; 3.6% of body weight; fig 2). This mirrors our finding at the 2001 South African Ironman triathlon in which the only athlete to develop hyponatraemic encephalopathy and who required hospital admission after the race was also the only athlete to gain a significant amount of weight during the race (3.8 kg; 4.7% of body weight).¹⁷

However, more to the point, the athlete in this study developed hyponatraemic encephalopathy within a relatively short time after exercise (5.16 h) despite a rate of fluid intake that complies with some of the current guidelines for fluid replacement during exercise.^{2, 22} Speedy *et al.*²³ have reported the development of asymptomatic hyponatraemia in two female Ironman triathletes who ingested fluids at similar rates to the cyclist in this report. Yet those triathletes developed asymptomatic hyponatraemia only after 13 h compared with 5-6 hours in this case. However, neither had symptoms of central nervous system dysfunction (hyponatraemic encephalopathy).

The aetiology of this condition clearly is the ingestion of free water at rates that exceed the rate of free water clearance by the kidneys, leading to water intoxication as faithfully reproduced in the laboratory.^{18, 24} The low sweat rate in this cyclist is probably due to the low exercise intensity (average cycling speed 21.1 km/h) combined with cool conditions in which heat losses due to convection and radiation are high. The latter two avenues of heat loss were not considered when the original studies on which the current drinking guidelines^{2, 22} are based were conducted.^{9, 22, 24} As a consequence of the low sweat rate of this cyclist and her low rates of free water clearance, fluid retention and cerebral swelling developed, causing hyponatraemic encephalopathy according to the mechanisms we have proposed since 1985,²⁵ and as conclusively proven in 1991,⁹ even though her rates of fluid ingestion were modest and well within currently accepted guidelines.^{2, 7-9, 23}

Confirmation that fluid overload caused hyponatraemic encephalopathy in this case comes from the predictions, based on the laboratory measurement of her average sweat $[Na^+]$, that her total Na^+ losses during the race were small (about 70-105 mmol). Furthermore, simple balance equations show that a positive change in weight of either 3.3% or 4.0% respectively would be enough to lower a female athlete's serum $[Na^+]$ to 130 mmol/l, depending on whether the ingested fluid is water (3.3%) or an electrolyte-containing sports drink (4.0%) according to the calculations of Weschler.²⁶ The actual weight change in this athlete was +3.6%, and her serum $[Na^+]$ after the race was 129 mmol/l, confirming the accuracy of the predictive equations of Weschler.²⁶

The fact that this athlete did not develop symptoms in subsequent exercise when she drank ad libitum and without increasing her Na⁺ intake before, during, or after exercise confirms that this condition is due to fluid overload to which any Na⁺ deficit plays only a minor contributory role²⁴ as it merely moderates the serum [Na⁺] at any given level of fluid overload.

Finally this study suggests that fluid replacement guidelines based on absolute rates of fluid intake are inherently flawed as sweat rates can vary so widely during exercise, ranging from >2 litres/h in American football players²⁷ to about 500 ml/h in ultramarathon runners in cold conditions²²⁻²⁴ to 300 ml/h in this cyclist.

Rather, we continue to argue that no evidence exists to suggest that athletes who drink ad libitum as opposed to at fixed rates are at increased risk of ill health during exercise, or that they will perform less than optimally as a result.^{4,28} Instead we argue that all the current evidence indicates that athletes perform optimally when they drink ad libitum during exercise.²⁹⁻³¹

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COMMENTARY

This is an important paper describing the development of hyponatraemic encephalopathy in a female cyclist. Although similar case studies have been published previously, this one is particularly interesting because the subject developed the disorder over a relatively short time (about five hours) and by ingesting fluids during her event at rates (~700 ml/h) well below those prescribed by the current guidelines of the American College of Sports Medicine (ACSM). The follow up examination is important because it shows that the predicted sweat Na⁺ losses would have been minimal, and lends strong support to the now well established overdrinking theory on why hyponatremia develops. This calculation is important to silence the doubters of this theory, many of whom sit blindly behind the doors of Gatorade and the ACSM.

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PostScript

LETTERS

Sodium ingestion and hyponatraemia: sports drinks do not prevent a fall in serum sodium concentration during exercise

In a well controlled and designed study, Baker *et al.* clearly showed the effects of ingesting large volumes of water or a carbohydrate/electrolyte sports drink (CES) on serum Na^+ concentration ($[\text{Na}^+]$) during exercise in older, active adults (54–70 years). The data show that ingesting any hypotonic fluid, be it water or a Na^+ -containing sports drink (about 18 mmol/l), in volumes that are similar to or more than weight losses produces a fall in $[\text{Na}^+]$ —a finding that mirrors the study of Twerenbold *et al.* published in this journal.¹ Readers should know that any fluid of concentration less than 150 mmol/l is considered to be hypotonic to the plasma.

In the study of Baker *et al.*, the ingestion of a CES produced a lower rate of fall in $[\text{Na}^+]$, as also found by Twerenbold *et al.* and also by Barr *et al.*² However, the authors failed to conclude that the consumption of any hypotonic fluid at these rates nevertheless causes a steady decline in serum $[\text{Na}^+]$. Instead, Baker *et al.* concluded that a CES better restores plasma volume and is therefore a more effective fluid replacement in older adults during intermittent exercise. This conclusion ignores the fact that, in their trial, serum $[\text{Na}^+]$ in both the men and women fell during exercise, regardless of the type of fluid they were ingesting, and that a falling serum $[\text{Na}^+]$ will eventually lead to hyponatraemia and its associated symptoms and medical complications.

One female subject in the study of Baker *et al.* lowered her $[\text{Na}^+]$ by nearly 10 mmol/l in just 150 minutes when voluntarily consuming 2.7 litres of CES. I have replotted her data to show that, had she continued drinking the CES at that rate and given a relatively constant rate of decline, after four hours her serum $[\text{Na}^+]$ would be about 128 mmol/l. After five hours it would have reached 125 mmol/l, and after six hours it would likely have been 122 mmol/l (Fig 1). As these are serum Na^+ concentrations at which cerebral and pulmonary oedema develop,³ and at which emergency medical treatment is necessary, this finding is of particular significance.

In 2000, the average 43.2 km marathon finishing time for women in North America was 4:57 (h:min). In the 2005 San Diego marathon alone there were 9069 women finishers, of whom about 2985 finished in 4–5 h, 2379 finished in 5–6 h, and 2531 in 6–9 h.⁴ If these marathon finishers were to consume CES at the rates advocated by Baker *et al.* and elsewhere⁵ to prevent "voluntary dehydration," the number of hyponatraemic cases and their associated medical complications would be sure to increase, placing undue pressure on medical directors and their staff who must diagnose and treat this preventable yet complicated condition.

Of particular concern is the fact that sports drink companies, as well as the American College of Sports Medicine (ACSM) drinking guidelines,⁶ currently advocate that sports drinks that contain Na^+ should be consumed to prevent hyponatraemia during prolonged exercise. However, the data of Baker *et al.* clearly show that the consumption of any hypotonic fluid in quantities to prevent "voluntary dehydration" will inevitably produce a fall in serum

$[\text{Na}^+]$, which can eventually lead to symptomatic hyponatraemia.

To help prevent any further increase in the number of cases of this preventable condition, I feel that this alternative interpretation of the data of Baker *et al.* should be brought to the attention of the readers of this journal.

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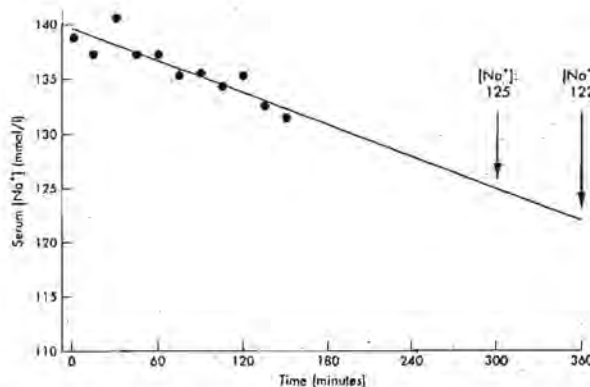


Figure 1 Linear regression ($r^2 = 0.81$; $p < 0.05$) of the fall in $[\text{Na}^+]$ during exercise in one female subject in the study of Baker *et al.* The rate of decline is approximately 3.3 mmol/l Na^+ /per hour when a carbohydrate/electrolyte sports drink is ingested at a rate of approximately 1.1 litres/h.

What do you think?

Since taking a year out of surgical training to investigate this new speciality by way of studying for the MSc, I look forward to the monthly arrival of your journal. The subject matter regularly presents a thought provoking mixture of original ideas and quality trial design.

There appears to be an obvious omission in the journal design though. Without a regular letters section, I think the reader misses out on the extended peer review process that for most journals allows experienced clinicians and scientists the opportunity to add their thoughts. Although I note a section exists on the website, it is more 'useful' in the journal because this process of "rubber stamping" is what often highlights truly important work, at the same time exposing weak articles for what they really are.

Unfortunately it is probably easier for articles on minority subjects to get to print because of the paucity of suitable reviewers. On the few occasions that this happens, I would appreciate the chance to read the other side of the argument, or indeed to vote.

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New use of current technology to measure rectal temperature and heart rate during endurance exercise

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Introduction

The technology necessary to log data remotely and independently has been available for some years. This technology has been applied mostly to environmental and natural sciences, however, and not in life sciences. This was due primarily to the cost of the technology and the small demand for it in the life sciences, especially in studies of exercise physiology. Our recent collaboration with a local technology company (SyGade Solutions (Pty) Ltd., Johannesburg) has resulted in the use of miniature data loggers to record rectal temperature, heart rate (HR), and altitude during road and cycle racing. This technology has the potential to measure these variables simultaneously and in a free-living situation and will therefore contribute to more innovative research.

Description of the data logger

The data loggers were tested in the laboratory from 35 - 44°C, and returned an accuracy ranging from - 0.22% to 0.1%. Each logger weighs 79 g and has dimensions of 105 x 58 x 20 mm (length x width x height, respectively). The loggers use a 16-bit micro-processor operating at 13 Mhz, and draw 20 milliamps during operation. Each is powered by a single AAA/1.5 v battery that can supply enough current for up to 12 hours of logging. Data are stored on a FLASH device similar to those used currently in digital cameras. This allows for the retention of data even if power is removed during use.

The loggers can record up to 12 hours of data at 2-second intervals in the absence of HR. When HR is recorded in addition to rectal temperature, the logging time decreases in direct proportion to the time interval between heart beats so that maximum logging time is shortened to 7 hours if a HR of 220 beats/minute is maintained. The minimum HR that can be

logged is 30 beats/minute.

The loggers measure HR in milliseconds from beat to beat. Because the HR transmitters are not suited to beat-to-beat calculations, and to reduce interference from other HR monitors, the HR transmitters were hard-wired to the data logger for maximal accuracy.

Temperature is measured by supplying the probe with a very accurate reference voltage/current and then measuring the changes with a 20 bit analog to digital converter. This is then converted to temperature by means of a calibration table, as supplied by the temperature probe manufacturer (VHA Plus, Irving, Texas, USA).

The air pressure sensor is a Motorola MPX4115 which is sensitive to pressure changes within 1 m of vertical distance. However, for the air pressure sensor to measure altitude accurately in metres above sea level, a sophisticated and accurate calibration is required. Therefore, the loggers were designed not to give accurate meter readings, but instead to give only relative changes in pressure, thus creating a profile of the race course. This allows the user to place an athlete at specific points on a course where changes in altitude are known and frequent.

The software for the micro-processor was compiled initially on a PC and then loaded via a special wire interface to the processor. Data are retrieved by interfacing the logger with a PC via a download cable inserted into the HR transmitter input. The programmer is then able to issue special control commands to the logger via the PC. The data are stored in a raw binary format. The processing and separating of data took place on the PC to produce text files that were then delimited in a Microsoft Excel workbook and plotted using the GraphPad Prism software package (GraphPad Software Inc., San Diego, California, USA).

The loggers contain a function that allows the user to log an event during the data-logging period. The user can log an event by pushing a button on the logger at a point specified by the researcher, for example before, during, or after the race. This allows the researcher to know exactly where the start and finish of the course are, for example, after the data have been downloaded and reduced.

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Over-extrapolation of research

19 April 2006

W Larry Kenney,
Professor
Penn State University,

Lindsay B. Baker

Send letter to journal:
[Re: Over-extrapolation of research](#)
[Email W Larry Kenney, et al.](#)

Dear Editor,

We appreciated the recent letter by Dugas in this journal (*BJSM* 2006;40:372) because it provides an excellent teaching tool for students on the dangers of over-extrapolating published data. Most scientifically puzzling is the figure published with Dugas' letter. In that figure, Dugas replots data from an elderly woman exercising intermittently for 150 minutes, then extends a linear regression line based on those data out to 6 h (a vastly different time frame) to make a point about ultra-endurance athletes (a vastly different subject population). Dugas' extrapolation ignores all negative feedback characteristics inherent in physiology (see published subjective sensory data for this subject in Baker et al.1) that would have prevented this subject from continuing both exercise and her overdrinking behaviour long before 6 h. To actually achieve the 6-h end point of Dugas' linear extrapolation, this petite older woman would have to continue to over-drink at the same rate until she increased her initial body weight by over 14%! This clearly defies both logic and physiology. To see the folly in such over-extrapolation, one only needs to apply Dugas' logic – and extrapolation methodology -- to heart rate data from the same study. If Dugas had chosen heart rate instead of serum sodium concentration to over-extrapolate, many of our elderly men and women would be finishing this imaginary 6-h ultra-marathon with heart rates of ~250 bpm, obviating any concern about hyponatraemia! Such is the danger of blindly extrapolating selected laboratory data from a "well controlled and designed study". Thus we disagree with Dr. Dugas that "this finding is of particular significance," as it could never have occurred, but share with him a desire to prevent all athletes from substantial overdrinking.

Also in his letter, Dugas restates several conclusions that were soundly based on empirical data, then (correctly) refutes his re-interpretations. Baker et al. illustrated the dangers of drinking fluids well in excess of sweat rate, especially in smaller subjects who sweat little. In one such subject, a 45.7-kg 65-year old woman who overdrank 2.4 kg more than she sweat, serum sodium fell when she overdrank a sports drink, and fell at a substantially higher rate when she overdrank a similar volume of water. Within the time period studied, she became symptomatically hyponatraemic only in the water trial. Dugas alters that simple observation by inserting such words (note CAPS) as "in volumes that are SIMILAR TO OR more than weight loss" and attributes that statement to Baker et al. Moreover, he suggests that excessive overdrinking is "advocated by Baker et al". I challenge Dr. Dugas to point out the line in Baker's paper where she advocates overdrinking.

It is unfortunate that several important and valid points made by Dugas and his colleagues over the years become lost when good science is sacrificed and other authors' published words are misrepresented. Drs. Dugas and his colleagues have made important contributions to our understanding of this topic that I believe all reasonable scientists working in this area agree upon: (1) Substantial overdrinking (well in excess of sweat loss) during exercise, regardless of the beverage consumed, is not to be suggested, facilitated, nor condoned; (2) Scientific organizations should not – and in my careful reading of the literature do not – simply tell athletes to "drink as much as possible". Rather, they tell athletes to learn to drink fluids during exercise in volumes that approximate sweat loss in an effort to prevent BOTH extremes -- significant dehydration and hyponatraemia; and, (3) In order to be palatable, sodium-containing beverages are necessarily hypotonic to plasma. During prolonged exercise, they do not necessarily prevent a fall in serum sodium, but attenuate the decline, as clearly illustrated by Twerenbold et al2 and others.

Note bene: In a previous letter addressing Baker et al., Noakes (3) took issue with a statement inaccurately referenced to Costill et al.(4) that "...carbohydrate-electrolyte solutions (CES) are more effective than water in stimulating voluntary fluid intake...and attenuating increases in core temperature during exercise-heat stress." To clarify, when consumed in equal volumes, there is no reported benefit of CES over water. Only when the increased voluntary intake associated with CES significantly limits dehydration do laboratory studies show an attenuated core temperature response. We thank Dr. Noakes for allowing us to clarify this point.

References

- 1 Baker LB, Munce TA, Kenney WL. Sex differences in voluntary fluid intake by older adults during exercise. *Med Sci Sports Exerc* 2005;37:789-96.
- 2 Twerenbold R, Knechtle B, Kakebeeke TH, et al. Effects of different sodium concentrations in replacement fluids during prolonged exercise in women. *Br J Sports Med* 2003;37:300-3.
- 3 Noakes T. *Med Sci Sports Exerc* 2005;38:193 (letter).
- 4 Costill, DL, Kammer WF, Fisher A. Fluid ingestion during distance running. *Arch Environ Health* 1970;21:520-25.

ACSM Fluid Recommendations: Replace Sweat, not "Drink as much as you can"

11 April 2006

William O Roberts,
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University of Minnesota

Send letter to Journal:

Re: ACSM Fluid Recommendations: Replace Sweat, not "Drink as much as you can"

Email William O Roberts

Dear Editor,

The case study described by Dugas and Noakes (2005:39;e38) demonstrates the wide individual variability in sweat losses that occur in athletes, and there is not much doubt this woman was ill advised for her fluid replacement plan. The conclusions of the both the case report and the commentary that follows place the blame for her problem on the 1996 American College of Sports Medicine (ACSM) Fluid Replacement Position Stand, whereas the problem seems to be misapplication of the ACSM advice. The case report does not detail where she got her fluid replacement advice but her previous experiences imply that she was overhydrating during exercise for many years, and she may be a "non-responder" to a hypo-osmotic, overhydrated state.

I have copied the pertinent sections of the ACSM position stand below: From the abstract. "During exercise, athletes should start drinking early and at regular intervals in an attempt to consume fluids at a rate sufficient to replace all the water lost through sweating (i.e., body weight loss), or consume the maximal amount that can be tolerated." [I have always interpreted the last half of this statement to mean replace up to sweat losses as outlined in the text, but others may have a different view.]

From the main text. "As such, individuals participating in prolonged intense exercise must rely on strategies such as monitoring body weight loss and ingesting volumes of fluid during exercise at a rate equal to that lost from sweating, i.e., body weight reduction, to ensure complete fluid replacement. This can be accomplished by ingesting beverages that enhance drinking at a rate of one pint of fluid per pound of body weight reduction. While gastrointestinal discomfort has been reported by individuals who have attempted to drink at rates equal to their sweat rates, especially in excess of $1 \text{ L}\cdot\text{h}^{-1}$, this response appears to be individual and there is no clear association between the volume of ingested fluid and symptoms of gastrointestinal distress. Further, failure to maintain hydration during exercise by drinking appropriate amounts of fluid may contribute to gastrointestinal symptoms. Therefore, individuals should be encouraged to consume the maximal amount of fluids during exercise that can be tolerated without gastrointestinal discomfort up to a rate equal to that lost from sweating."

From the conclusion. "During exercise, fluid and carbohydrate requirements can be met simultaneously by ingesting $600\text{-}1200 \text{ m L}\cdot\text{h}^{-1}$ of solutions containing 4%-8% carbohydrate. During exercise greater than 1 h, approximately 0.5-0.7 g of sodium per liter of water would be appropriate to replace that lost from sweating." [The key word in this section is "can," it does not say "should" or even imply that all athletes require $600\text{-}1200 \text{ m L}\cdot\text{h}^{-1}$.]

It appears that she did not get her advice from the American College of Sports Medicine Fluid Replacement Position Stand as suggested by the authors of the manuscript and the commentary, nor did she or her advisors learn from her past experiences. With a sweat rate of 270 ml/hour and a fluid intake of just over 700 ml/hour, she is either going to urinate large volumes during and immediately after her activity or she will become water intoxicated if her kidneys do not respond with appropriate diuresis. Although the wording in the abstract could be misconstrued to mean "drink as much as you can," the main text clarifies the recommendation. The recommendation stresses replacing sweat losses as the first choice in fluid replacement during exercise, and not more. While the $600\text{-}1200 \text{ m L}\cdot\text{h}^{-1}$ is higher than $400\text{-}800 \text{ m L}\cdot\text{h}^{-1}$ that Noakes recommends, it represents the range around the middle of the population and does not reflect the problems faced by those (including this woman) who sit at the extremes of sweat loss during activity. Even the $400 \text{ m L}\cdot\text{h}^{-1}$ from Noakes' recommendation would have left this woman overhydrated at the end of her activity. To cite an example like this from the lower extreme of sweat loss as a failure of the ACSM Fluid Recommendations seems a stretch to me and emphasizes that the wide variation in fluid requirements across the entire athlete population makes it nearly impossible to give a precise recommendation that includes specific fluid volumes. The best advice still remains to "replace most of the sweat losses" as outlined in the ACSM Position Stand. The ACSM Exercise and Fluid Replacement Position Stand is currently in revision and should be published in the near future with updates that reflect changes in the area since 1996.

William O Roberts MD, MS, FACSM Past President, American College of Sports Medicine (I have no financial or advisory ties to any water or sports drink companies. I did receive an honorarium from the Gatorade Company for appearing on an educational film clip regarding exertional heat stroke in 2003.)

Reference

Convertino VA, Armstrong LE, Coyle EF, et al. American College of Sports Medicine Position Stand: Exercise and Fluid Replacement. *Med Sci Sports Exerc* 1996; 28: R1-7.