Pacing strategies during exercise in the heat
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In May 2013, the Tour of California, a multi-stage professional cycling race took place for the 8th time. During the third stage of this race, which finished in Palm Springs, ambient temperature reached maximum values of approximately 43°C. This extremely high temperature, combined with considerable solar radiation and the low cycling speed because of the uphill finish, resulted in two cyclists abandoning the race as a result of heat exhaustion. Moreover, the majority of the cyclists required medical attention after finishing. It was nearly impossible for the caretakers of the teams to adequately provide the cyclists with bottles of cold drinks, cold wet towels, and bags of ice to cool down.

Afterwards, the race leader at the start of the third stage, Lieuwe Westra, from the Vacansoleil-DCM Pro cycling team posted the following quote on Twitter (Figure 1.1):

"Lost the leaders jersey today but I had no choice! I wasn’t feeling that good. If I would have gone full throttle, I’d be laying on the side of the road right now."

#Abittoohot

This scenario nicely shows the impact that heat stress can have on health and exercise performance. In rest, the internal temperature of the human body is maintained within a range of approximately 36-38°C (Weller 2005). However, when exercising, core temperature often surpasses this ‘safe’ range and can even get as high as 40°C. Fortunately, a complex interplay between heat production and heat loss mechanisms
generally ensure that the temperature returns to ‘normal’ values after termination of
exercise and as a result, most of the times, no serious health issues arise. The other way
around, a high body temperature can impair performance (Westra’s quote). Therefore,
when exercising, individuals have to find an optimum between performance on one hand
and an acceptable rise in body temperature on the other hand.

In chapter 1 of this thesis, an introduction will be given to the human heat balance, the
regulation of exercise intensity and the impact that heat stress can have on health and
exercise performance. Moreover, methods to reduce the effect of heat stress on
performance are reviewed.

**BODY TEMPERATURE REGULATION**

Humans, just like other warm-blooded (endothermic) animals, are able to maintain a
stable internal temperature in an environment that can exhibit large thermal fluctuations.
Deviations from the normothermic range (36-38°C) may lead to impaired bodily
functioning and even serious health issues such as organ failure and malfunctioning of the
nervous system (Lim et al. 2008). To maintain a stable internal body temperature, humans
need to produce and conserve heat when the outside temperature is cooler than the
internal temperature and loose heat when the internal temperature becomes too high.
The balance between heat production and heat loss eventually determines the core body
temperature. This heat balance can be expressed in equation 1.1 (Åstrand and Rodahl
1986).

\[ M - C - R - E = S \]  (equation 1.1)

In this equation M represents the metabolic heat that is produced by active tissues
(mainly muscles).
C represents conduction and convection. Conduction is the transfer of heat from one material to another through direct contact. An example of conduction is the heat that is transferred from the hands to the handle bars of a bike when cycling on a cool day. Convection is the transfer of heat from one material to another by the movement of gasses or fluids. An example of convection is the cooling of the body when running outside. The air movement over the body continually replaces the relatively warm air around the body by cool air.

R represents radiation, which is the transfer of heat through electromagnetic radiation generated by the thermal motion of charged particles in matter. A simple example of radiation is the warming of the skin by sunlight.

E represents heat loss by evaporation of moisture. The main contributing factor to this kind of heat loss is evaporation of moisture from the skin, but also evaporation of moisture from the respiratory tract contributes to E.

Finally, S represents the total body heat storage. If the body temperature increases, S is positive, when the body temperature decreases, S is negative.

The main control center for the regulation of body temperature is the hypothalamus. This organ with approximately the size of an almond is located just above the brain stem and (like the name indicates) below the thalamus. Its function in thermoregulation can be compared with that of the thermostat in houses. The actual body temperature is compared with a ‘setpoint’ temperature and responses in heat loss or heat gain mechanisms are initiated if the actual body temperature deviates from this setpoint (Sawka and Wenger 1988). The first part of the hypothalamic regulation of body temperature is the reception of temperature by peripheral thermal receptors in the skin and by central thermal receptors mainly located within the hypothalamus itself. The second part is hypothalamic integration of the information from these thermal receptors and comparing this with the setpoint. The third and final step in body temperature regulation is the actual response. The goal of this step is to restore the actual body
temperature to the setpoint. There are several effector mechanisms to accomplish this and these all facilitate or hinder aspects of heat gain/dissipation.

When the temperature sensed by the thermal receptors is lower than the setpoint temperature, heat loss needs to be reduced or heat production has to increase. When it comes to reducing heat loss, the most effective way is to increase the insulative capacity of the skin. This is accomplished by increasing the vasomotor tone of the smooth muscles around peripheral blood vessels in the skin which leads to constriction of these vessels. The result of this vasoconstriction is a redistribution of blood from the ‘cold’ periphery to the ‘warm’ body core and reduced heat loss (Sawka and Wenger 1988). Together with the skin, muscles and subcutaneous fat form an effective insulative buffer that is generally sufficient for effective thermoregulation if ambient temperature is not extremely low, although this strongly depends on body composition (Wilmore and Costill 1999). If reducing body heat by peripheral vasoconstriction is insufficient, heat needs to be produced to maintain the body temperature within the preferred range. The main mechanism to produce heat in the cold is shivering, which can lead to a fivefold increase in heat production compared to the basic metabolic rate (Eyolfson et al. 2001). Heat can also be produced in the absence of shivering. This so called non-shivering thermogenesis can increase heat production in rest up to 20% (van Ooijen et al. 2005). However, the absolute heat production in Watts is relatively small. The exact mechanisms of non-shivering thermogenesis are still being debated, but it likely takes place in brown adipose tissue and skeletal muscle as a result of cold exposure (Wijers et al. 2010).

When body temperature is higher than the setpoint value, the human body needs to dissipate heat. One way to facilitate heat loss is peripheral vasodilatation which, in contrast to heat conservation by peripheral vasoconstriction, redistributes blood from the core to the skin. Vasodilatation is mainly passive in humans: the vasoconstrictor tone is reduced. A very powerful mechanism to dissipate heat is the evaporation of sweat secreted by sweat glands, especially in combination with the enhanced cutaneous blood flow. By evaporating one liter of sweat, 2428 kJ of heat can be dissipated (Wilmore and
Humans can evaporate over 2 liters of sweat per hour, leading to approximately 1350 W of cooling power.

The aforementioned physiological adaptations to either cold or heat are important defense mechanisms against body temperature fluctuations, but the most effective mechanism is perhaps the most logical one: behavior. Simple actions such as wearing appropriate clothing, avoiding exposure to extreme climates, and adjusting the activity level are often sufficient to prevent disturbances in body temperature.

Although core temperature is generally maintained around 37°C (Weller 2005), more pronounced deviations often occur. Fever, for example, is a symptom of infection and characterized by a core temperature exceeding 38°C. The higher temperature is caused by a higher setpoint of the hypothalamic thermostat. Although the increased core temperature is generally unwanted, it also stimulates the immune response and inhibits certain pathogens (Lim et al. 2008). Also, exposure to extreme climatic conditions can lead to pronounced deviations in core temperature. Thermoregulation and behavior will generally be sufficient to adequately deal with most conditions, but sometimes the effect will be too extreme to compensate for. Exercise is probably the most common cause of an increased core temperature since heat loss mechanisms show a time lag compared to heat production mechanisms. In cycling for example, close to 80% of the energy that is generated is liberated as heat (Ettema and Loras 2009). Depending on several factors such as exercise intensity and training status, this can lead to exercise-induced hyperthermia. Usually, core temperature returns to 'normal' values as soon as the exercise is terminated.

The next paragraph will discuss exercise and its effect on body temperature.

**EXERCISE AND BODY TEMPERATURE**

In 2006, the annual International Four Day Marches Nijmegen, with approximately 40,000 participants the largest multi-day walking event in the world, was cancelled after one day because of the death of two participants. Because of the high environmental
temperatures and absence of wind, these two casualties were linked to heat-related illnesses. Although it was later confirmed that the cause of deaths was cardiovascular, heat stress was likely a provoking factor (Saanen et al. 2007). Moreover, 300 participants had to quit the march because of problems associated with exercise in the heat. This example illustrates that exercise, especially when performed in hot and humid conditions and with no or little wind, can lead to serious health issues. The three prominent ‘classic’ exercise-related heat illnesses are heat cramps, heat exhaustion and heat stroke.

Heat cramps are involuntary muscle contractions and occur during or after exercise. However, because body core temperature not necessarily needs to be increased for heat cramps to occur, the direct link between exercise-induced hyperthermia and muscle cramps appears to be lacking (Schwellnus 2007).

Heat exhaustion is the most observed heat-related illness and is characterized by collapsing of an athlete during or after exercise (Holtzhausen et al. 1994). It often occurs when individuals perform exercise in a hot environment while being unacclimatized. The cause for heat exhaustion is believed to be an increased body core temperature caused by extracellular fluid or electrolyte deficits as a result of excessive sweating and inadequate rehydration (Saanen et al. 2007). When exercise is stopped, blood tends to pool in the dilated peripheral vessels and the resulting reduced central blood volume leads to a weak pulse and low blood pressure and this might ultimately result in collapse. Other signs and symptoms of heat exhaustion are non-specific (Table 1.1). Heat exhaustion is sometimes referred to as a mild form of the dangerous condition heat stroke, but as core body temperature is usually < 40°C, the direct link with an elevated core temperature is still unclear (Wendt et al. 2007). The treatment of heat exhaustion is simple and straightforward: removing the person from the heat source and providing rehydration (Glazer 2005).

Heat stroke is the most dangerous of the three ‘classic’ heat-related illnesses and is without a doubt related to an elevated core temperature (Wendt et al. 2007). The diagnosis is based on three factors: a core temperature exceeding 40°C, malfunctioning of
the central nervous system (CNS), and a preceding period of heat exposure (Saanen et al. 2007). The symptoms of heat stroke are stated in Table 1.1. Similar to heat exhaustion, a reduction in central blood volume can result in a pronounced rise in core temperature. Moreover a reduced visceral blood flow can lead to cellular hypoxia in the intestine and liver (Hall et al. 1999), which may eventually lead to leakage of endotoxins into the bloodstream (Hall et al. 2001). When these endotoxins activate leukocytes and endothelial cells, and these cells excrete inflammatory cytokines, the hypothalamic setpoint for sweating and peripheral vasodilatation can be increased, impairing heat loss even more (Bouchama and Knochel 2002). The most important goal of the treatment of heat stroke is a quick reduction of core body temperature, as the main predictor of outcome is the duration of hyperthermia (Lugo-Amador et al. 2004). Submersion in (ice)cold water appears to be the fastest way to reduce core temperature and should therefore be the treatment of choice for heat stroke (Smith 2005).

<table>
<thead>
<tr>
<th>Heat exhaustion</th>
<th>Heat stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td>General weakness</td>
<td>Same as heat exhaustion plus:</td>
</tr>
<tr>
<td>Muscle weakness</td>
<td>• Absence of sweating</td>
</tr>
<tr>
<td>Muscle cramps</td>
<td>• Hot, dry skin</td>
</tr>
<tr>
<td>Nausea</td>
<td>• CNS abnormalities (delirium, coma)</td>
</tr>
<tr>
<td>Vomiting</td>
<td>• Rapid pulse</td>
</tr>
<tr>
<td>Lightheadedness</td>
<td>• Difficulty breathing</td>
</tr>
<tr>
<td>Headache</td>
<td>• Disorientation</td>
</tr>
<tr>
<td>Profuse sweating</td>
<td>• Agitation</td>
</tr>
</tbody>
</table>

Table 1.1 Symptoms and signs of heat exhaustion and heat stroke (Bouchama and Knochel 2002; Glazer 2005).

The aforementioned heat related illnesses are examples of failures in thermoregulation that can occur when metabolic heat production is high (e.g. during sport activities) and heat dissipation to the environment is difficult. However, there is one powerful mechanism that usually prevents the excessive rise of core body temperature during exercise and that is behavior. The human body appears to be programmed to avoid the
surpassing of physiological limits. When such a limit is approached, or it is anticipated that this will happen, the exercise intensity is reduced to prevent harmful consequences (Noakes et al. 2004, 2005; St Clair Gibson and Noakes 2004). A prerequisite for this modulation of exercise intensity is of course that the intensity can be changed by the individuals, which is the case in most real-life competitions. The next sections of the introduction will focus on the regulation of exercise intensity and the specific effects of heat-related signals on this regulation.

**REGULATION OF EXERCISE INTENSITY**

The regulation of exercise intensity was already subject of studies in the early 20th century. The 1922 Nobel Prize in Physiology or Medicine winner A.V. Hill was a pioneer within the field of exercise physiology and the model he proposed for factors limiting exercise performance (Figure 1.2) has been the basis for many further studies.

![Figure 1.2 Model of A.V. Hill that describes factors limiting performance.](image)

From Noakes (2011).
In short, it describes fatigue as a result of physiological failure. This failure can be found in several systems and includes – but is not limited to – glycogen depletion in the muscles, too high body temperature, and a failure of the circulation to provide enough oxygen to the muscles.

In recent years, the focus within exercise physiology has shifted from the limitation of exercise performance to the regulation of performance (Noakes 2011; Noakes et al. 2004; St Clair Gibson and Noakes 2004; Tucker 2009). Within these studies, fatigue is not seen as a result of physiological failure but more as a result of the regulation to prevent physiological limits to be surpassed. In this chapter, current models to explain fatigue during exercise are described. These models describe fatigue as a result of physiological failure and generally differentiate between systems that are failing. Examples of ‘failure models’ are:

- Cardiovascular failure model
- Energy depletion model
- Neuromuscular fatigue model
- Thermoregulatory failure model

Within the cardiovascular model, fatigue is described as a failure of the heart to supply enough oxygen to the exercising muscles and/or to adequately remove waste products from these muscles. As these aspects depend on the blood flow to and from the muscles, the flow rate determines the amount of oxygen delivery and waste products removal. Because there is a limitation of the increase in cardiac output (stroke volume x heart rate), there is a limit to the blood flow as well and this might impair exercise performance.

As muscles require energy to contract, the availability of sufficient ATP is essential for optimal exercise performance. As energy is liberated by means of glycolysis (glucose degradation), lipolysis (breakdown of lipids), or phosphocreatine re-phosphorylation, a failure of these systems to produce the required amount of energy will lead to fatigue.
This can also be the case when there are not sufficient substrates available for these metabolic pathways, like glycogen within the muscles and liver.

The neuromuscular fatigue model describes how fatigue is caused by a reduced muscle activation and/or contraction. This can be a result of a failure of central activation (Millet et al. 2003), a reduced response of the muscle to a stimulus (Lepers et al. 2002), or reduced effectiveness of excitation-contraction coupling within the muscle (Hill et al. 2001).

The thermoregulatory failure model describes how exercise intensity is reduced when core body temperature becomes too high. When the core temperature rises to approximately 40°C, muscle activation is reduced and exercise is terminated. Moreover, β-waves in the brain are reduced, causing a reduction in arousal levels, leading to a reduced exercise performance (Nielsen et al. 2001; Nybo and Nielsen 2001b). The effects of a high core temperature on exercise performance are described in more detail in the next section of the introduction.

Collectively, these models describe that failure in different physiological systems can cause fatigue and thereby limit exercise performance. However, one important aspect is not taken into account within these models: the possibility to reduce exercise intensity before actually stopping exercise. When individuals are forced to continue exercising at a fixed intensity, logically they cannot continue with this forever. In these situations, likely one or more systems will fail and exercise will be terminated. When individuals are able to self-select their exercise intensity, they will slow down to prevent the surpassing of physiological limits and the resulting severe disturbance of body homeostasis.

The central governor model (CGM) (Noakes et al. 2004; St Clair Gibson and Noakes 2004; Noakes et al. 2005) was the first model to describe fatigue as a result of the regulation of the avoidance of physiological failure. Within this model, both anticipation and feedback play important roles. The intensity of exercise is continuously adjusted by changing the number of motor units that is recruited by central activation via the motor cortex. The
resulting muscle activity results in a so-called pacing pattern during exercise. Another term to describe this model is the anticipatory/feedback model of exercise regulation (Figure 1.3 (Tucker 2009)). The anticipatory component within this model is suggested to consist of physiological inputs (such as skin temperature, core temperature, muscle glycogen content), the expected exercise duration, previous experience, motivation, and external competition. Based on this anticipatory information a template is created for an acceptable rise in the rating of perceived exertion (RPE) during exercise. When exercising, feedback from physiological signals results in a conscious RPE. This conscious RPE can be seen as an integration of all physiological feedback and reflects how hard an individual perceives to be exercising. This conscious RPE is continually compared with the on forearm constructed RPE template and if there are differences, the exercise intensity is adjusted. By making changes to the exercise intensity, also physiological signals are regulated to avoid dangerous homeostatic disturbances. This implicates that individuals exercise with a physiological reserve to prevent surpassing of physiological limits (Swart et al. 2009). This is especially true for exercise bouts without a clear endpoint (Tucker 2009).

It is proposed that small changes in physiological signals result in exercise intensity adjustments that are executed without conscious awareness, whereas greater physiological disturbances attract conscious awareness and an associated response (Edwards and Polman 2013).

Although this anticipatory/feedback model of the regulation of exercise is a simple representation of exercise in real-life competition, it can explain certain situations that cannot be explained by the failure-models. One of these situations is the occurrence of an end-spurt near the finish of an event, which is associated with increased electromyographic (EMG) activity in exercising muscles (Ansley et al. 2004; Hettinga et al. 2006; Tucker et al. 2004). This increase in EMG activity indicates an increased neural activation of motor units. According to the failure-models, it is impossible to speed-up towards the end of a race, because athletes should be the most tired at that moment, oxygen need should be greatest, and the amount of waste product accumulation in the muscles should be greatest. Contrary to the failure models, the end-spurt phenomenon can easily be explained by the anticipatory/feedback model of exercise intensity.
regulation. Towards the end of exercise there is more certainty about the endpoint and the chance that physiological limits are surpassed becomes smaller. Therefore, there is no need to maintain a physiological reserve and exercise intensity can be increased.

Signals about the thermal environment and physiological signals are important for the regulation of exercise intensity (Tucker and Noakes 2009). Based on inputs before exercise, a pacing strategy is selected, and changes in thermal physiological afferents during exercise may result in changes in exercise intensity. Therefore, the next section will focus on the effects of thermal signals (core temperature, skin temperature, rate of heat storage, and thermal perceptions) on the selection and modulation of exercise intensity.

![Figure 1.3 Schematic overview of the anticipatory/feedback regulation of exercise intensity. From Tucker (2009).](image-url)
EXERCISE PERFORMANCE IN THE HEAT

When exercise is conducted in the heat, a reduction in performance is generally observed, both in fixed-intensity (Galloway and Maughan 1997) and free-paced exercise (Peiffer and Abbiss 2011; Tatterson et al. 2000). The metabolic heat production in combination with the impaired heat dissipation to the environment poses great stress on the thermoregulatory system and cardiovascular, metabolic and neurological functioning may be hampered. Two views have emerged that describe how high temperature affects exercise performance: the critical core temperature theory, which is usually linked to fixed-intensity exercise, and the effect of the rate of increase of body heat on the anticipatory/feedback regulation of exercise intensity.

Figure 1.4 Esophageal temperature during exercise to exhaustion in 40°C and 17% relative humidity during pre-cooling, control, and pre-heating trials. Data from Gonzalez-Alonso et al. (1999).

When individuals exercise without having control over the intensity, they only have control of the moment to stop exercising. In several studies it is shown that individuals stop exercising when they reach a core temperature of approximately 40°C (Nielsen et al.
This mechanism can be considered to be a safety switch, ensuring that exercise is terminated before serious hyperthermia-related health issues occur. The core temperature at exercise termination appears to be unaffected by ambient temperature, skin temperature and the rate of increase in body temperature during exercise (Figure 1.4; (Gonzalez-Alonso et al. 1999)). However, the critical core temperature does appear to be influenced by exercise mode, motivation, and aerobic training status since well-trained individuals can tolerate higher final core temperatures than untrained individuals (Cheung and McLellan 1998).

Several studies reported exercise termination at a temperature of approximately 40°C, but Ely et al. (2009) observed that athletes could tolerate temperatures well above this value as long as their skin temperature was relatively low. The resulting large core-skin temperature gradient resulted in a relatively small amount of blood in the skin and therefore greater central blood volume facilitating aerobic exercise performance. For this reason, the authors concluded that there is not a critical core temperature that serves as a safety switch, but rather, fatigue is explained as a continuum with inputs from multiple physiological systems.

Although the termination of exercise at a core body temperature of approximately 40°C has been observed frequently, the precise mechanisms by which hyperthermia leads to exercise termination are not complete understood. Active heating (Nybo and Nielsen 2001a) as well as passive heating protocols (Morrison et al. 2004) have shown that central activation was reduced when core temperature was high. This can be caused by inhibitory signals from hypothalamic thermoreceptors (Caputa et al. 1986), a reduced cerebral blood flow leading to a reduced cerebral oxygenation (Rasmussen et al. 2010a; Rasmussen et al. 2010b), or an increased α/β ratio which is linked to a reduced state of arousal and higher RPE (Nielsen et al. 2001). Also peripheral factors that are not related to the central nervous system appear to be involved in fatigue caused by exercise hyperthermia. It is shown that power production following repetitive isometric contractions is substantially reduced when a muscle is passively heated (De Ruiter and De Haan 2000) or core temperature is passively increased (Todd et al. 2005). Moreover, a reduced visceral blood
flow can result in endotoxemia (the leakage of endotoxins from the gastrointestinal tract into the bloodstream) and this can lead to an increase in the hypothalamic setpoint for sweating and peripheral vasodilatation (Bouchama and Knochel 2002; Hall et al. 2001). Together, these studies suggest that exercise hyperthermia may lead to both central and peripheral fatigue causing exercise termination. In Figure 1.5 an overview is given of the potential triggers of fatigue.

![Diagram of potential triggers of fatigue induced by exercise hyperthermia.](image)

**Figure 1.5** Overview of potential triggers of fatigue induced by exercise hyperthermia. From Cheung and Sleivert (2004).

During exercise in which individuals are able to self-select the intensity, core temperature generally remains below nominally critical values and the anticipatory/feedback regulation of exercise intensity also appears to apply to exercise performed in the heat. Exercise intensity is reduced with the goal to prevent a potentially harmful state of hyperthermia, resulting in decreased exercise intensity at temperatures well below critical values. By reducing the intensity, metabolic heat production is decreased and consequently, the
increase in core temperature is blunted. Within the anticipatory/feedback model of exercise intensity regulation, thermal signals at the start and during exercise may play important roles. In the heat, these signals become of special interest. Afferent information regarding core temperature, skin temperature and body heat storage as well as thermal perceptions may all affect performance.

Many studies investigated the effect of heat stress on self-paced exercise performance (Altareki et al. 2009; Gonzalez-Alonso et al. 1999; Tucker et al. 2006; Peiffer and Abbiss 2011; Tucker et al. 2004; Tatterson et al. 2000; Marino et al. 2004). One of the first studies investigating the effect of heat stress on self-paced exercise performance was conducted by Tatterson et al. (2000). They observed that power output was reduced while exercising in the heat well before core temperature became close to possibly dangerous values. This down-regulation of exercise intensity resulted in a similar increase in core temperature in the heat (32°C) and in thermoneutral (23°C) conditions (Figure 1.6).

It is suggested that power output is used as a tool to allow for an acceptable rise in core temperature during exercise to prevent catastrophic failure. This notion is confirmed in a study on the effect of body mass on pacing pattern during exercise in the heat (Marino et al. 2000). Within this study it was found that body mass was inversely correlated with running speed in 35°C. The greater surface-to-weight ratio of the lighter runners facilitated heat dissipation and allowed them to have a greater running speed and still maintain an acceptable rise in core temperature compared to heavier runners. Four years later, a study of the same research group reaffirmed the effect of body mass, and more specifically the surface-to-weight ratio on self-selected exercise intensity in runners (Marino et al. 2004). In this study, African runners were found to perform better than Caucasian runners in the heat, but not in cooler conditions. The greater body mass of the Caucasian runners made them reduce their speed in the heat to ensure an acceptable rate of rise in core temperature to avoid possibly dangerous hyperthermia. This was evidenced by the similar pattern of core temperature increase of African and Caucasian runners despite the difference in performance. Although the exact mechanism of action of core temperature on the regulation of exercise intensity is still unclear, it is suggested that the
rate of rise in core temperature combined with the (anticipated) remaining exercise duration is an important signal for the selection and modulation of exercise intensity. This is enforced by the observation that starting with a lower core temperature generally yields improvements in aerobic exercise performance (Ross et al. 2013), possibly because of a greater capacity for heat storage (Lee and Haymes 1995).

**Figure 1.6** Power output and rectal temperature during a 30-min cycling trial at 32°C (HT) and 23°C (NT) in elite road cyclists. * Denotes difference (P<0.05) with NT values. Values are mean ± standard error (n=11). Data reproduced from Tatterson et al. (2000).
Another factor that is proposed as input for the anticipatory/feedback model of exercise regulation is skin temperature (Schlader et al. 2011b). This is an especially relevant signal since the skin is in direct contact with the (thermal) environment and the main location for heat exchange. The temperature of the environment has a direct effect on the temperature of the skin and can therefore be important for exercise performance. High skin temperature and an accompanying low gradient between core and skin temperature results in a high skin blood flow (Charkoudian 2003) and is associated with cardiovascular strain (Kenefick et al. 2010). Also, high skin blood flow can lead to a reduced muscle blood flow resulting in decreased effectiveness of exercise (Hettinga et al. 2007). Because of the high skin blood flow, less blood is available for oxygen delivery to the brain (Rasmussen et al. 2010b) which may lead to a decreased motor unit recruitment and impaired exercise performance (Rasmussen et al. 2010a). Cooling of the skin before the start of aerobic self-paced exercise has been shown to be beneficial for pacing pattern and performance (Kay et al. 1999; Ross et al. 2011), and Schlader et al. (2011b) showed that skin temperature at the start of a 60-min cycling time trial was important for the selection of starting exercise intensity. A lower skin temperature at the start of exercise resulted in higher initial exercise intensity and this resulted in a higher average intensity. However, another study showed that changes in skin temperature did not affect pacing pattern during a 40-km cycling time trial (Barwood et al. 2012). Therefore, at this moment the precise effect of skin temperature on the selection and modulation of exercise intensity is not completely understood and appears to differ with the duration of exercise.

The combination of core temperature and skin temperature gives an indication of mean body temperature. This estimation of total body heat content may also be of importance in the regulation of exercise intensity. Tucker et al. (2006) suggested that exercise intensity is controlled by feedback about the rate of heat storage during the early stages of exercise. Based on this feedback, exercise intensity is regulated to prevent an excessive rise of body temperature. Although the calculation of heat storage within this study has been shown to be erroneous (Jay and Kenny 2009), the concept itself is still interesting as it integrates information from different thermal afferents.
Together with information regarding body heat content, thermal perceptions constitute
the thermal state of the human body. These perceptions are usually questioned as
thermal sensation (how hot or cold are you feeling now?) and thermal comfort (how
comfortable are you with the temperature?) (ISO10551 1993). Thermal sensation is mainly
determined by skin temperature (Cotter et al. 2001), whereas thermal comfort is
determined by core temperature as well as skin temperature (Kato et al. 2001). Thermal
perceptions have been suggested to be relevant for the selection and modulation of
exercise intensity (Schlader et al. 2011a), although this has been questioned as well
(Barwood et al. 2012).

As the appliance of heat stress generally leads to differences in many thermal measures, it
is often complicated to determine the isolated effect of a thermal afferent. In chapters 2
and 3 of this thesis, an attempt is made to manipulate a single thermal afferent (skin
temperature) and investigate its effect on pacing pattern during time trials of different
lengths.

On first sight, the critical core temperature paradigm and the anticipatory regulation of
exercise intensity in the heat appear to be conflicting. However, Cheung (Cheung 2007)
pointed out that these theories test different concepts and are actually complementary.
Both mechanisms can be seen as protective mechanisms against potentially harmful
hyperthermia. The anticipatory down-regulation of exercise intensity prevents an
excessive rise in body temperature, whereas the critical core temperature is a safety
switch for when the core temperature does get critically high.

**IMPROVING PERFORMANCE DURING EXERCISE IN THE HEAT**

Heat stress has a negative impact on (prolonged) exercise performance. As many recent
sports events took place in hot conditions (e.g. the Olympic Games in Athens and Beijing),
sports physiology researchers focused on methods to alleviate the detrimental effect of
heat stress on performance. The simplest method is to remove the heat source or shield
oneself from the heat source, but as this is often not possible during exercise, other strategies can be used by individuals to reduce the performance degradation caused by heat stress. These strategies include: acclimatization / acclimation, cooling (before and during exercise), fluid ingestion (before and during exercise) and the adoption of pacing strategies.

**Acclimation / acclimatization**

The terms acclimation and acclimatization are both used to describe adaptive changes in response to changes in particular climatic factors. When these changes are experimentally induced, this is called acclimation whereas the term acclimatization is used when these changes occur in the natural climate (Eagan 1963; Hart 1957). Exercise-heat acclimation/acclimatization can be used as a method to induce physiological changes that allows individuals to improve performance in the heat by allowing the body to get used to exercising in the heat. The first physiological adaptation to occur is cardiovascular stability and involves plasma volume expansion and a lower exercise heart rate (Patterson et al. 2004; Senay et al. 1976). This is generally followed by a greater skin blood flow (Wyndham 1973), increased sweating rate/sensitivity (Lorenzo and Minson 2010), lower muscle glycogen use (Febbraio et al. 1994), and a lower core body temperature (Nielsen et al. 1993). Generally, complete exercise-heat acclimation is accomplished within 7-10 days, but after 4-6 days almost two-thirds of the physiological adaptations have already occurred (Pandolf 1998). Heat acclimation has been shown to be an effective strategy to reduce the detrimental effect of heat stress on exercise performance, but the effects will disappear if the repeated exercise-heat exposure is not repeated (Lorenzo et al. 2010; Nielsen et al. 1993).

**Cooling before exercise**

As one might expect, body cooling can reduce the effect of heat stress on exercise performance. Cooling can for example be accomplished by: cooling vests, cooling caps, cooling neck collars, head ventilation devices, the ingestion of ice slurry, or submerging body parts in cold water. Most of the studies on cooling strategies focused on cooling
before exercise (pre-cooling) rather than on cooling during exercise. The main reason for this is the practical difficulties that are associated with cooling during exercise. Cooling during exercise often requires an external energy source which is prohibited during professional sports events or adds extra weight, which can act out negatively on performance.

Cooling before exercise can be applied by several methods that either reduce core temperature (e.g. cold water ingestion, ice slurry ingestion, cold intravenous saline), skin temperature (e.g. cool air exposure, ice jackets, cooling caps), or both (e.g. immersion of body parts in cold water, cold room). These methods have in common that they all reduce the body heat content prior to exercise and increase the heat storage capacity of the body. A meta-analysis of pre-cooling effects on sports performance demonstrated that pre-cooling improves performance during fixed-pace (open end) exercise by 8.6%, performance of graded exercise tests by 6.0%, and performance during (closed end) time trial protocols by 4.2% (Wegmann et al. 2012). Especially during long-lasting exercise, pre-cooling becomes advantageous, because body heat content is usually greater during this type of exercise than during short exercise protocols. When looking at pacing patterns after pre-cooling, generally a greater exercise intensity is observed towards the end of exercise (compared to no pre-cooling) whereas relevant physiological changes have disappeared at that moment (Duffield et al. 2010; Ihsan et al. 2010). Several mechanisms have been proposed for how pre-cooling improves performance. One mechanism is that pre-cooling increases the capacity for heat storage and that a greater rate of heat gain is possible during exercise. In fixed-pace exercise this results in a longer time until core temperature becomes critically high, whereas in self-paced exercise, a reduction in exercise intensity can be delayed or prevented. Another potential mechanism is the lower cardiovascular strain after pre-cooling. Because heat stress increases the blood flow to the skin to dissipate heat, less blood is available for oxygen delivery to, and waste product removal from the exercising muscles. This results in greater cardiovascular strain during exercise (Galloway and Maughan 1997). Pre-cooling reduces the need for a greater blood flow to the skin for heat dissipation and has been shown to reduce cardiovascular strain during exercise (Lee and Haymes 1995). Moreover, when the skin is cooled, peripheral
vasoconstriction increases the central blood volume and this leads to improved perfusion of the exercising muscles (Booth et al. 1997; Duffield et al. 2003). Together with a reduced sweat rate (Duffield et al. 2010; Lee and Haymes 1995), these adaptations to pre-cooling can substantially reduce cardiovascular strain. Pre-cooling may also positively affect thermal sensation and the RPE (Yeargin 2008; Marsh and Sleivert 1999), although other studies did not observe this (Arngrimsson et al. 2004; Kay et al. 1999).

When using pre-cooling strategies in practice, a couple of factors should be considered. The added value of pre-cooling differs for example with type and duration of exercise, environmental temperature, and gender (Wegmann et al. 2012). Moreover, individuals need to get accustomed to pre-cooling and ideally practice the incorporation of pre-cooling within the regular preparation regime. Also, when using pre-cooling as a preparation regime for exercise in the heat it is important to achieve an optimal combination of a cool body and warm exercising muscles. From a practical point of view, combining the standard warming-up regime combined with pre-cooling by ice slurry might be optimal. Chapter 4 of the thesis further elaborates on this topic.

**Cooling during exercise**

As mentioned earlier, cooling during exercise is often complicated because of practical considerations. However, the effectiveness of several cooling modalities on performance has been investigated in sport and occupational settings. General consensus of these studies is that cooling during exercise is beneficial for reducing heat stress and improving performance. This was found for wind cooling (Ansley et al. 2008; Teunissen et al. 2013), wearing a cooling vest (Nunneley and Maldonado 1983), neck cooling (Tyler and Sunderland 2011b; Tyler et al. 2010) and hand palm cooling (Hsu et al. 2005). Of these cooling methods, wind cooling is common during out-of-doors exercise. Especially in exercise modalities involving relatively high speeds like cycling, the associated high wind speed can substantially improve convective heat dissipation and reduce the need for other (pre-)cooling (Saunders et al. 2005). The other cited methods for cooling during exercise have the disadvantage that they are uncomfortable for the user and/or add weight.
Therefore, the ingestion of ice slurry during exercise has recently become of interest to researchers. When ice slurry replaces fluid consumption during exercise, it does not add weight and may therefore improve performance in the heat by increasing the heat storage capacity of the body.

**Drinking during exercise**

During exercise, sweat production increases to allow evaporative cooling of the body, and this causes a loss of body water. A person’s sweating rate depends on exercise duration, personal factors (i.e. genetic predisposition and fitness level), environmental conditions (i.e. temperature, humidity and relative air velocity), and clothing worn (Shapiro et al. 1982). Because of these factors there are great differences in sweat rate within and between individuals. A person will dehydrate – technically but not necessarily functionally - if fluid intake is not high enough to compensate for the fluid loss. The decrease in plasma volume observed with dehydration, increases thermal and cardiovascular strain (Gonzalez-Alonso et al. 1995; Montain and Coyle 1992), glycogen use (Hargreaves et al. 1996), and perceived exertion (Mudambo et al. 1997), and the combination of these effects can possibly impair exercise performance (Cheuvront et al. 2003; Murray 2007). However, there is considerable debate whether or not hypohydration (=reduced body water content) during exercise is actually detrimental to exercise performance. Several studies have shown that hypohydration between 1 and 2% of body mass already results in performance decrements (Armstrong et al. 1985; Bardis et al. 2013; Walsh et al. 1994). Moreover, the most recent position stand of the American College of Sports Medicine (ACSM) (Sawka et al. 2007) states that hypohydration by more than 2% of body mass should be avoided. However, a review by Goulet (2011) showed that dehydration up to 4% body mass did not impair exercise performance in moderate-to-warm conditions (20-33°C), as long as cyclists were allowed to drink according to their thirst sensation during exercise. The discussion regarding the importance of body water content is also reflected in the literature about drinking guidelines during exercise. Should athletes replace all fluid lost by sweating, drink to avoid > 2% body mass loss, drink according to their thirst sensation, or not drink at all? No simple answer can be given to this question because the
preferred strategy differs with exercise mode and duration, environmental conditions and personal factors. The chapters 6 and 7 of this thesis further explore the effect of hydration status and rehydration (drinking) behavior on self-paced exercise performance in the heat.

**Drinking before exercise**

Another method to increase body water content is drinking before exercise (pre-loading). Especially because the sweat rate of athletes during exercise in a hot environment can exceed 2 L·h\(^{-1}\) (which is well above maximal gastric emptying rate) (Coyle and Hamilton 1990), and there is often little opportunity for drinking during events. The main goal of preloading is to increase body water (induce hyperhydration) to delay, limit or prevent dehydration. Inducing hyperhydration to create a ‘reservoir’ of body water that can be used for sweating and delaying or preventing dehydration has been shown to reduce thermoregulatory strain and to provide a meaningful physiological performance advantage (Anderson et al. 2001; Coutts et al. 2002; Greenleaf et al. 1998; Greenleaf et al. 1997; Sims et al. 2007a; Sims et al. 2007b). However, inducing hyperhydration can be counterproductive if the excessive fluid intake results in hyponatremia (water intoxication). The combination of electrolyte loss via sweating and drinking of an excessive amount of hypotonic fluid can cause a severe drop in serum sodium concentration, with or without complete rehydration. This drop can lead to headache, confusion, nausea, and in extreme cases even coma or death. Other disadvantages of preloading with fluid are that body weight is higher from the start onwards and that there is a higher chance that an athlete has to urinate during an event.

**Adoption of pacing strategies**

Earlier in this introduction, the effects of thermal signals on pacing pattern during exercise and the implications for overall performance were described. It is clear that heat stress reduces exercise intensity during self-paced exercise. When pacing patterns during exercise in the heat are compared with patterns during exercise in thermoneutral environments, the inability to maintain a desired power output until the end is a frequently observed phenomenon (Tatterson et al. 2000). This reduction in power output
has been observed when core temperatures were not even close to critical values (Altareki et al. 2009; Tucker et al. 2004; Ely et al. 2010). As the reduction in exercise intensity in the heat is such a common observation, it is proposed that pacing is not only a resultant of the anticipatory and feedback regulation, but it can also be used as a strategy to minimize performance decrements in the heat. If the rate of increase in body heat content determines pacing pattern in the heat, then it could be that a pacing strategy leading to a lower rate of heat storage may be beneficial for performance. A slower initial pace results in a lower metabolic heat production and resulting lower rate of heat storage. However, if this also leads to a less pronounced reduction in muscle activation and drop in exercise intensity is still unknown.

CONCLUDING REMARKS

Exercising in the heat can lead to serious hyperthermia-related health issues, especially when the intensity is high. The human body has several defense mechanisms aimed at the prevention of a possibly dangerous hyperthermic state. These mechanisms include an anticipatory reduction of exercise intensity when feedback from thermal signals indicates that successful exercise completion is at stake and a ‘safety switch’ that causes exercise termination when the core temperature becomes (too) high. Also, several techniques, including (pre-)cooling, fluid ingestion, and adoption of pacing strategies can be used to counteract the detrimental effects of heat stress on exercise performance. The main goals of this thesis are (1) to investigate the importance of different thermal signals on the selection and modulation of exercise intensity during self-paced exercise in the heat and (2) to explore the ergogenic effects of thermal interventions on performance during self-paced exercise in the heat.
Chapter 1

OUTLINE OF THE THESIS

This thesis aims to evaluate the effect of thermal signals on pacing pattern and explore the ergogenic effect of thermal interventions on performance during self-paced exercise in the heat. More specifically, Chapter 2 describes the effect of a radiant heat exposure on pacing and performance of a 7.5-km cycling time trial.

Chapter 3 evaluates the effect of two durations of radiant heat exposure on pacing during a 15-km cycling time trial. By comparing results from this chapter with chapter 2 of the thesis, conclusions can be drawn about the importance of skin temperature in the regulation of exercise intensity relative to the length of the time trial.

Chapter 4 compares the effects of four different thermal preparation regimes on pacing pattern during 15-km cycling time trials in the heat.

Chapter 5 aims to evaluate the influence of the anticipated thermal load on pacing pattern during a 20-km time trial.

Chapter 6 investigates the interactive effect of heat stress and hypohydration on pacing pattern and performance during a 40-km cycling time trial in the heat.

Chapter 7 aims to determine if ad-libitum drinking during a 40-km cycling time trial in the heat can counteract the potential negative effects of starting hypohydrated.

Chapter 8 evaluates the effect of an active warm-up on simulated firefighting performance.

Chapter 9 provides an overview of the main results of the thesis, the additive scientific value, directions for future research and recommendations for sport and occupational settings.