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Review

Temperature and neuromuscular function

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This review focuses on the effects of different environmental temperatures on the neuromuscular system. During short duration exercise, performance improves from 2% to 5% with a 1 °C increase in muscle temperature. However, if central temperature increases (i.e., hyperthermia), this positive relation ceases and performance becomes impaired. Performance impairments in both cold and hot environment are related to a modification in neural drive due to protective adaptations, central and peripheral failures. This review highlights, to some extent, the different effects of hot and cold environments on the supraspinal, spinal and peripheral components of the neural drive involved in the up- and down-regulation of neuromuscular function and shows that temperature also affects the neural drive transmission to the muscle and the excitation-contraction coupling.

From a cardio-vascular to a neuromuscular approach in environmental physiology

In the years following (and even preceding) the Second World War, there has been a large amount of research on the effect of thermal conditions on exercise capacity in humans (e.g., Dill et al., 1938; Eichna et al., 1945; Adolph et al., 1947; Bazett, 1949; Hardy, 1949). During the following 50 years, most of researches examining the consequences of exercising in a cold environment – or cold water – focused on health and safety issues (e.g., Shephard, 1985; Lin, 1988; Doubt, 1991); whereas the research in hot environments focused on cardiovascular adaptations (e.g., Williams et al., 1962; Leithead & Lind, 1964; Strydom et al., 1966; Wyndham et al., 1968; Rowell, 1974; Brengelmann, 1983; Sawka et al., 1983). However, the 1990s saw a slight shift in research focus from a health and cardiovascular model to a neural model. Indeed, the cardiovascular adaptations were unable to explain some observations, such as the decrement in muscle activation observed with passive hyperthermia in the 2000s (Morrison et al., 2004; Thomas et al., 2006; Racinais et al., 2008).

One of the first studies to investigate the effect of environmental conditions on the central nervous system (CNS) concluded that there was an alteration in cortical excitability of hyperthermic (central temperature increased up to 44–45 °C) anesthetized cats (Bulochnik & Ziablov, 1977). In humans, cerebral adaptations to environmental temperature have been studied from both a circulatory (e.g., Nybo & Nielsen, 2001b; Nybo et al., 2002; Rasmussen et al., 2004) and neural (e.g., Nielsen et al., 2001; Nybo & Nielsen, 2001c; Todd et al., 2005) perspectives during this last decade. Briefly, hyperthermia has been shown to reduce brain perfusion (Nybo & Nielsen, 2001b; Nybo et al., 2002; Rasmussen et al., 2004) and increase or decrease metabolic rate in different brain areas (Nunneley et al., 2002). These modifications impair heat loss from the brain (Nybo et al., 2002) and reduce cerebral oxygenation during hyperthermic strenuous exercise (Rasmussen et al., 2010) but may not affect prefrontal cortex oxygenation during passive hyperthermia (Morrison et al., 2009). In parallel, modifications in electroencephalographic (EEG) activity have been observed with hyperthermia (Nielsen et al., 2001; Nybo & Nielsen, 2001c; Rasmussen et al., 2004). These modifications suggest a decrement in arousal in humans exercising up to hyperthermia (i.e., central temperature higher than 39 °C) (Nielsen et al., 2001) and were associated with an increase in perceived exertion (Nybo & Nielsen, 2001c; Rasmussen et al., 2004). The modification in EEG activity and the increase in perceived exertion do not appear to be a simple effect of the reduced cerebral perfusion but may be related with the fatigue that arises concomitantly with the increase in central and brain temperatures during exercise-induced hyperthermia (Rasmussen et al., 2004).

However, neural drive investigations cannot be limited to cerebral adaptation. For example, it was observed that hyperthermia decreased voluntary activation without affecting motor cortical excitability (Todd et al., 2005). The authors pointed to a failure of descending voluntary drive to compensate for changed muscle properties, despite the availability of additional cortical output (Todd et al., 2005). Thus, investigating cerebral changes without taking into account the peripheral modifications in the neuromuscular system does not capture the complexity of the supraspinal adaptation. In addition, it has recently been shown that the decrement in muscle activation with hyperthermia was partly linked to some peripheral failure in the transmission of the neural drive (Racinais et al., 2008).

Therefore, we believe that peripheral neuromuscular adaptation has to be taken into account when investigating the effect of environmental conditions. In addition, the relationship between temperature and both neural and muscular function implies that both hot and cold environments should be considered. After discussing the effect of environmental temperature on "*Neuromuscular performance*" this review focuses on the temperature dependency of the "*Neural drive*", spinal adaptations (*Spinal modulation*), peripheral nerves (*Peripheral nerve adaptations*) and the muscular system (*Muscle function*).

Neuromuscular performance

The effect of the environmental temperature on neuromuscular performance is related to exercise duration, especially in a hot environment (*Exercise duration*). Performance during short duration exercise (e.g., jump, sprint, muscle force and power) can be considered as an indicator of the integrated functioning of the neuromuscular system. This kind of performance is widely recognized to deteriorate in a cold environment but can be improved in a hot environment, which acts as a passive warm-up (Bishop, 2003; Racinais, 2010). From the data obtained in both cold and hot conditions, a relationship between muscle temperature and performance can be established (*Dose/response relationship*). This relation is a function of contraction type and velocity (*Contraction velocity*). However, an improvement in performance in hot environment has not always been observed. Hot environments only seem to improve muscle performance in the morning, when the body temperature is at its lowest; however, if central temperature continue to increase there is a reduction in voluntary force production (*Local vs central temperatures*).

Exercise duration

It has been well established that a decrease in muscle temperature in a cold environment induces profound changes in neuromuscular function (Denys, 1991). Practically all aspects of neuromuscular function, (i.e. mechanical, biochemical and neural), are deteriorated with lowered muscle temperature. Conversely, warming-up the muscle can improve muscle function and performance (Bishop, 2003) but also reduce the time to fatigue (Thornley et al., 2003). In addition, although an increase in muscle temperature may improve power production during a short duration cycling sprint, this difference disappears if the sprints are repeated (Linnane et al., 2004) suggesting that the effect of a hot environment are dependent of exercise duration. Power decrement during a repeated-sprints exercise was observed to be greater with hyperthermia (induced by exercise in hot environment, 40 °C) than in control condition (Drust et al., 2005) leading the authors to conclude that the attainment of an elevated central temperature could negatively affect performance when sprints have to be repeated (Drust et al., 2005). In continuous activity as in repeated sprints, the shift from improvement to impairment in human ability to exercise in hot environment seems to be related with the increase in central temperature when exercise is prolonged. As presented in Table 1, this shift is related with exercise duration (Table 1, Parts A, B and C). The studies reported in the Part A observed an improvement in human performance in a hot environment or following passive whole body warm-ups. In all instances, when performances improved test duration ranged from <1 s (i.e., vertical jump) to 30 s (i.e., cycling exercise). The studies in the Part B failed to observe a marked effect of a hot environment on human performance. All these performances involved repetition of short duration exercise or exercise of ~ 5 min in duration. The studies reported in the Part C are based on exercise lasting more than 10 min and observed a decrement in human performance in hot environment. From the studies presented in the Part A of the Table 1 – showing a consistent temperature/performance relationship – it is possible to determine a dose/response relationship.

Dose/response relationship

The rate of deterioration in muscle performance is strongly associated with decreasing muscle temperature. The more muscle temperature decreases the more muscular performance decreases and thus, there is a dose-dependent relationship between these factors. In the work of Oksa et al. (1997) eight subjects were exposed to 27 °C, 20 °C, 15 °C and

Table 1. Different effects of an increase in body temperatures on human physical abilities as a function of exercise characteristics and duration

Reference	Effect of body temperatures increase	Exercise duration	Environmental condition
Part A: positive effect on human physical abilities			
Segal et al. (1986)	↑ muscle contractility	<1 s	Muscle *in vitro*, water bath
Davies and Young (1985)	↑ muscle force ↑ muscle contractility	<1 s	Water bath
Binkhrorst et al. (1977)	↑ muscle force	Few seconds	Water bath
Bergh and Ekblom (1979)	↑ power output ↑ muscle force	Few seconds	Water bath and exercise
Falk et al. (1998)	↑ peak power output	5 × 15 s	Warm environment
Sargeant (1987)	↑ power output	20 s	Water bath
Linnane et al. (2004)	↑ mean power output	30 s	Water bath and warm environment
Ball et al. (1999)	↑ peak power output	2 × 30 s	Warm environment
Part B: no effect on human physical abilities			
Dotan and Bar-Or (1980)	= power output	30 s	Warm environment
Backx et al. (2000)	= power output	2 × (3 × 30 s)	Warm environment
Linnane et al. (2004)	= mean power output	2nd sprint of 30 s	Water bath before warm environment
Falk et al. (1998)	= peak power output	2nd series of 5 × 15 s	Warm environment
Hue et al. (2003)	= power production	5 min	Warm environment
Part C: negative effect on human physical abilities			
Voltaire et al. (2002)	↓ incremental exercise duration	~ 10–15 min	Warm environment
Gonzalez-Alonso et al. (1999)	↓ time to exhaustion	~ 28–63 min	Water bath before warm environment
Nybo and Nielsen (2001a)	↓ time to exhaustion	~ 50 min or more	Warm environment

↑, improvement; =, no significant effect; ↓, decrement.

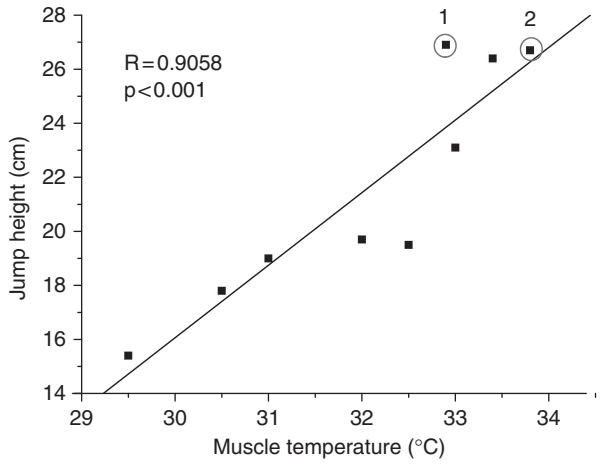

Fig. 1. Correlation between muscle temperature and jump height of the drop jump. Each point represents eight subjects except at 1, $n = 7$ and 2, $n = 3$. Modified with kind permission of Springer Science+Business Media from Oksa et al. (1997). Eur J Appl Physiol 75: 484–490 and Oksa et al. (1996a, b). Human Mov Sci 15: 591–603.

10 °C air for 60 min wearing minimal clothing (shorts and jogging shoes). After each exposure the subjects performed maximal drop jump and the flight time of the jump (corresponding to the height of the jump) was measured. During the exposures the muscle temperature from calf (m. gastrocnemius medialis) was measured from the depth of 3 cm. Figure 1 summarizes the relationship between muscle temperature and height of the jump and shows a significant correlation.

Passive rewarming has been used as a method to return muscle force back to thermoneutral level and Oliver et al. (1979) found that at room temperature 1 h is enough to restore cooling induced decrement in muscle force. However, active rewarming is more efficient way to restore reduced performance capacity. In the study of Oksa et al. (1996a, b) eight subjects were allowed to do rewarming exercise after being cooled at 10 °C for 60 min. After cooling, they performed a drop jump and then walked on a treadmill for 5 min at a velocity of 5 km/h, then performed another drop jump and walked again. After three walking bouts (15 min) the thermoneutral muscle temperature and after four walking bouts (20 min) thermoneutral jump flight time was obtained. Thus, there is a dose-dependent relationship also between active rewarming (exercise) and recovery in performance. It may be concluded that the most important factor in determining the outcome of performance is muscle temperature (Fig. 1).

Muscle power production or jump height was reduced after immersion of the leg in a cold bath (Bergh & Ekblom, 1979; Sargeant, 1987) but improved if the leg was immersed in a hot bath (Bergh & Ekblom, 1979; Davies & Young, 1985; Sargeant, 1987). From the studies relating modifications in short-duration exercise performance associated with muscle temperature recording, it is possible to estimate a range of variation in muscle force and/or power per degree of variation in muscle temperature (Table 2). From the data presented in the Table 2, a variation in muscle temperature of 1 °C can modify performance by 2–5% depending on the contraction

Table 2. Variation (%) in muscle force/power for 1°C variation in muscle temperature

Reference	Variation (%) in muscle force/power for 1°C of variation in muscle temperature	Test	Methodology used to modify muscle temperature
Oksa et al. (1995)	2.4	Balls throw	60 min in a room at 10°C
Sargeant (1987)	3	Cycle ergo meter	Water bath 12–18°C (45 min)
	4		Water bath 44°C (45 min)
Bergh and Ekblom (1979)	2.1	Leg force 0°/s	Cooling by water bath and warming by exercise
	4.2	Vertical jump	
	4.4	Peak velocity	
	4.7	Mean velocity	
	4.7	Leg force 90°/s	
	4.9	Leg force 180°/s	
	5.1	Cycle power	
Oksa et al. (1996a, b)	5.4	Drop jump	Room at 10°C (60 min)

The relation within muscle force/power and muscle temperature is positive: an increase in muscle temperature improves muscle force/power whereas a decrease in muscle temperature impairs muscle function. This relation can be quantified by the percentage of variation in muscle force/power for 1°C variation in muscle temperature.

type and velocity. Generally speaking, there is a positive relationship between the magnitude of the effect of temperature and the movement velocity.

Contraction velocity

Following water immersion in water bath ranging from 18 °C to 39 °C, muscle power and the velocity of contraction increased in parallel with muscle temperature whereas muscle force was not affected (Binkhorst et al. 1977). This suggests that high velocity movements are more temperature dependent than low velocity movement. This assumption is supported by the fact that dynamic exercises are more affected by temperature than isometric contractions (Bergh & Ekblom, 1979). In addition, optimal pedalling velocity during a 20 s cycling sprint is also positively linked with temperature (Sargeant, 1987). Several studies on muscle contractile properties have shown that the rate of contraction is slower with subnormal muscle temperatures, leading to less powerful contractions (e.g. Bigland-Ritchie et al., 1992; De Ruiter & De Haan, 2000). During exercise, this translates to power decrements when temperature decreases and power increments when temperature increases and more so at high (cycling at 140 rev/min) than slow (cycling at 54 rev/min) movement velocities (Sargeant, 1987). This was also confirmed in upper body muscles (Oksa et al., 1995) where cooling-induced decrements in ball throwing exercise was higher with light ball (0.3 kg, faster movement, 9.4% reduction in performance) than with heavier ball (2.0 kg, slower movement, 5.6% reduction in performance).

Cooling also affects the relationship between force production and velocity. The force–velocity curve is shifted to the left (De Ruiter & De Haan, 2001), which means that at a given force the velocity of movements or muscle contraction decreases after cooling. It also means that maximal power and force in a cooled muscle occurs with a slower muscle contraction velocity than in a thermoneutral muscle (De Ruiter & De Haan, 2001). Regarding the force–time curve a similar shift has been observed meaning that in a given time less force is produced after cooling the muscle (Clarke & Royce, 1962). That could be explained by slower cross-bridges cycling, thus lowering the rate of force development without affecting maximal isometric force.

Local vs central temperatures

In line with the experiments based on water immersion of the exercising muscle, a cold environment markedly decreases muscle power (Hackney et al., 1991; Oksa et al., 1995, 1996a, b, 2000). However, the effects of a hot environment are equivocal. Some studies observed similar power output in neutral and hot environments (Dotan & Bar-Or, 1980; Backx et al., 2000), whereas others have observed a higher power output in hot environments than in neutral conditions (Falk et al., 1998; Ball et al., 1999). It was recently hypothesized that one of the confounding factors responsible for these different observations could be the circadian rhythm of the body temperature (Racinais, 2010). Indeed, in the morning, when body temperature is at its lowest, a hot environment can improve muscle contractility (Racinais et al., 2005), muscle force (Racinais et al., 2005) and short-duration performance (Racinais et al., 2004). Cold exposure; however, decreases performance in both the morning and the afternoon (Racinais et al., 2009).

Another explanation for the variability of the effect of a hot environment on muscle function is the level and the kind of temperature considered.

Following the dose/response relationship presented in section ''Dose/response relationship'', an increase in muscle temperature can increase muscle power. However, this is not a linear relationship as there is a ceiling above which increasing the environmental temperature does not improve maximal power output (Racinais et al., 2006). In addition, exposure to hot environments increases central temperature, whereas, it is an increase in local temperature that improves power output (Falk et al., 1998). As presented in the section ''Exercise duration'', an increase in central temperature when exercise is prolonged can even reduce the maximal power output during sprint cycling (Drust et al., 2005). It is central temperature rather than local thermal afferent input from the skin that seems to be responsible for the alteration in neural drive (Thomas et al., 2006). This is supported by animal models that have observed rats to stop exercising in a hot environment at the same abdominal and cerebral temperatures regardless of the modification made to their initial temperature (Fuller et al., 1998) and goats to reduce velocity or refuse to move when cerebral temperature increases close to 42 °C (Caputa et al., 1986). In the human brain, heat loss is inadequate during prolonged exercise with hyperthermia, leading to higher brain than central temperature (Nybo et al., 2002). Combined, these studies suggest that cerebral temperature could also be a key factor leading to an alteration in physical activity.

It is important to note that a reduction in voluntary maximal force has also been observed after passive hyperthermia (Morrison et al., 2004; Thomas et al., 2006; Racinais et al., 2008) suggesting that central temperature is the key factor, independently of any exercise-induced fatigue. When passive exposure to a hot environment increases central temperature close to 39 °C force production is reduced due to decrement in voluntary activation (Morrison et al., 2004; Thomas et al., 2006; Racinais et al., 2008), i.e. decrement in the neural drive to the muscle.

Neural drive

Both hot and cold environments influence muscle electrical activity (*Environmental condition and muscle electrical activity*). A hot environment decreases voluntary activation (*Voluntary activation failure in hot environment*) whereas a cold environment alters co-activation and the agonist–antagonist ratio (*Coactivation changes in cold environment*). These adaptations can represent a physiological alteration but can also represent a protective adaptation (*Failure or modulation*?).

Environmental condition and muscle electrical activity

The electrical activity (EMG) of the muscle may be substantially affected by cooling (e.g. Rissanen et al., 1996). The literature is not unanimous is regards to the changes in EMG amplitude as some studies report decreased amplitude due to cooling (Bell, 1993; Petrofsky & Laymon, 2005) while others report increased amplitude (Piedrahita et al. 2008). Although the difference between studies may be explained by different exercise types and cooling procedures, Petrofsky and Laymon (2005) reported that EMG amplitude correlates positively with increasing force production regardless of temperature variation and may therefore be considered as reliable indicator of muscle strain in various conditions. The literature more uniformly reports that cooling decreases the frequency component EMG and that the decrement seems to depend fairly linearly on the level of cooling (Petrofsky & Lind, 1980). For example, a 30 min exposure of the forearm to 10 °C water in comparison with 40 °C water decreased the frequency from approximately 180 to 100 Hz (Petrofsky & Lind, 1980). The decrement in frequency component has been connected with simultaneous decrease in nerve conduction velocity (Mucke & Heuer, 1989).

During exercise in the heat, EMG activity has been observed to decrease (Kay et al., 2001; Tucker et al., 2004, 2006) or remain unchanged (Ftaiti et al., 2001; Hunter et al., 2002). However, it is difficult to draw conclusions from these data as muscle drive during exercise depends on exercise intensity (i.e., maximal vs sub-maximal) and protocol type (i.e., self-paced vs fixed intensity). From the data obtained during a maximal voluntary contraction (MVC) performed following active hyperthermia, EMG is not affected during brief ($\sim$ 4 to 5 s) MVC (Nybo & Nielsen, 2001a; Saboisky et al., 2003) but is decreased with prolonged (Nybo & Nielsen, 2001a) or repeated contractions (Martin et al., 2005). However, as a reduction in EMG has also been reported following exercise in a neutral environment (Racinais et al., 2007), only data from passive heat exposure should be considered to conclude on the effect of a hot environment on neural drive. In addition, environmental temperature can also affect surface EMG data by changing the electrophysiological properties of the skin–electrode pair (e.g., sweating, gel melting). Therefore, only data on voluntary activation obtained during passive hyperthermia and with electrical stimulation of the motor nerve should be considered.

Voluntary activation failure in hot environment

A decrement in the voluntary activation has been observed in the leg extensors without alteration in the

forearm flexors after cycling exercise inducing a heat stress (Saboisky et al., 2003), which suggests a selective reduction in the voluntary activation of the exercising muscle (Saboisky et al., 2003). However, others observed similar impairments in the voluntary activation of both the exercising (knee extensors) and non-exercising (handgrip) muscles after cycling to exhaustion in hot environment (Nybo & Nielsen, 2001a). This suggests that the reduction in voluntary activation is linked with the temperature rather than exercise-induced fatigue (Nybo & Nielsen, 2001a). This second hypothesis is supported by several studies that have observed a decrement in voluntary activation with passive hyperthermia (Morrison et al., 2004; Thomas et al., 2006; Racinais et al., 2008), which confirm that a high body temperature *per se* can reduce voluntary muscle activation.

Voluntary activation is not altered more after exercising in hot than in neutral environment during the first seconds (~ 10 s) of contraction but further decreases as the contraction becomes more prolonged (Nybo & Nielsen, 2001a). This suggests that the capacity of the CNS to maximally activate the muscle may be altered by hyperthermia only if force output has to be sustained for more than a few seconds (Nybo & Nielsen, 2001a; Martin et al., 2005). Given that comparable data have been observed in a neutral environment with an earlier and larger central activation deficit during a continuous than an intermittent elbow extension task (Bilodeau, 2006), it is difficult to discriminate the relative role of the temperature and the exercise in these previous data. In the absence of exercise, passive hyperthermia has been shown to reduce voluntary activation even during contraction of 10 or less seconds (Morrison et al., 2004; Thomas et al., 2006; Racinais et al., 2008), In addition, Racinais et al. (2008) showed that during a 120 s contraction passive hyperthermia induces an additional decrease in voluntary activation. This additional decrease was not explained by additional failure in the peripheral transmission of the neural drive suggesting the occurrence of supraspinal failure when contraction is prolonged (Racinais et al., 2008). The possibility of a supraspinal failure during a prolonged contraction with hyperthermia is supported by the observation that hyperthermia does not affect the amplitude of a superimposed twitch evoked by motor cortex stimulation during a brief MVC but this amplitude increases more during a sustained maximal effort in hyperthermia than in neutral environment (Todd et al., 2005).

Coactivation changes in cold environment

Neural drive alterations in cold environment differ from those in hot environment. Whereas hyperthermia seems to decrease the amount of activation of the agonist muscle, a cold environment mainly acts on the agonist–antagonist relation. During human locomotion the antagonist muscle contracts simultaneously with the agonist muscle, a phenomenon called co-activation. In normal conditions co-activation is desired and helps to keep movement "smooth." However, literature reports that cooling has a clearly modulating input on the co-activation by increasing it. Bawa et al. (1987) found that during extension of the elbow the antagonist muscle (*m. biceps brachii*) co-activated significantly more while subjects were cooled, whereas in thermoneutrality mainly the agonist (*m. triceps brachii*) was active. More recently, Oksa et al. (2002) found that during wrist flexion extension work in cold the level of co-activation was significantly higher when compared with thermoneutral conditions. A similar phenomenon was previously found in the studies of Oksa et al., (1995, 1997) where during the concentric phase of the muscle contraction the activity of the antagonist muscle was significantly increased during cooling and at the same time the activity of the agonist decreases significantly, a phenomenon named as "braking effect". These two changes may be the mechanistic reasons for the decreased muscular performance in the cold. In addition, it seems that another reason underlying these changes is the reduced activity of the muscle spindles (Oksa et al., 2000). However, it has been shown that a cooling induced increase in the level of co-activation can be reduced by intermittently changing work intensity from low to moderate or high (Oksa et al., 2006).

Humans can adapt to cold, especially in terms of thermal responses, but also at the level of the neuromuscular system. Few efforts have been made to see if repeated local (forearm) cold water exposure (8 °C water, 30 min/day/10 days) can induce neuromuscular adaptations (Geurts et al., 2005, 2006). These studies failed to show any adaptation in the time to peak tension or relaxation rate. However, very recently the study of Westerlund et al. (2009) showed that 3 months of to whole body cryotherapy (WBC, extremely cold air, -110 °C) was able to induce adaptations in neuromuscular performance. In that study 14 subjects were exposed to WBC three times a week for 2 min minimally clad. Before and after the exposure period, the subjects performed dynamic drop jump exercise. EMG was simultaneously recorded from ankle extensor (m. gastrocnemius medialis, agonist during shortening phase) and flexor (m. tibialis anterior, antagonist during shortening phase) muscles. At the beginning of the WBC period there was a significant reduction in the flight time of the drop jump (from 436 ± 17 to 420 ± 16 ms, $P < 0.05$) but this difference was not significant after 3-month WBC (from 434 ± 17 to 427 ± 17, ns)

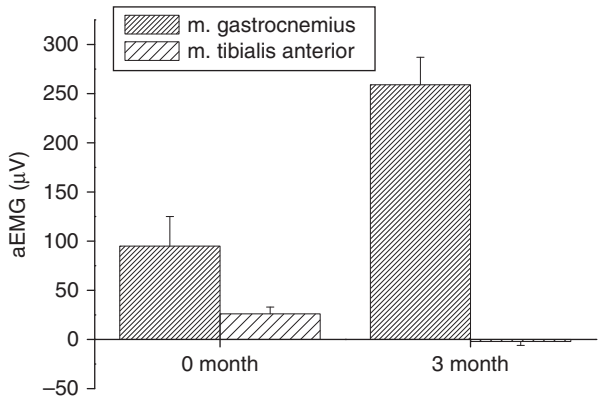

Fig. 2. The difference in averaged EMG activity before and after whole body cryotherapy (WBC) of m.gastrocnemius (agonist) and m. tibialis anterior (antagonist) muscles during the shortening phase of the drop jump in the beginning (0 month) and after (3 month) 3-month exposure period. The values are mean ± SE. Modified with permission from Westerlund et al. (2009). J Therm Biol 34: 226–231.

(Westerlund et al., 2009). Similarly, during the shortening phase of the drop jump the agonist activity increased and antagonist activity decreased after the 3-month WBC indicating significantly reduced co-activation (Fig. 2). The reduced co-activation possibly induced the observed smaller difference in flight time (increased performance). These results point towards the possibility that neuromuscular system is able to adapt when systematic cold exposure of sufficient duration is applied.

Failure or modulation?

The reduced activity of the agonist muscle associated with an increased activity of the antagonist muscle in cold environments could be interpreted as a strategy to prevent cold tissues from injury (Oksa et al., 1995, 1996a, b). The decrement in voluntary activation in hot environment has also been hypothesized to represent a conscious or unconscious anticipatory decrement in voluntary motor drive to avoid further heat production and protect the body (and the brain itself) from an additional rise in temperature (Marino, 2004; Tucker et al., 2004, 2006). Supporting this hypothesis, it has been observed that both power output and EMG activity during a self-paced time-trial decreases in hot conditions before body temperatures becomes extremely high, suggesting an anticipatory response adjusting muscle recruitment to reduce heat production (Tucker et al., 2004). Exercise intensity has been proposed to be down-regulated by the rate of heat storage to maintain homeostasis by reducing the exercise work rate (Tucker et al., 2006; Altareki et al., 2009). The theory of an adaptation from the CNS to protect the body was first developed to explain the force decrement of respiratory muscles following exercise due to decrement in neural drive (Verin et al., 2004), which could represent a protective mechanism in order that muscle fatigue does not exceed a critical threshold (Amann et al., 2006). However, the theory of an anticipatory decrement in exercise intensity cannot be transposed to passively induced hyperthermia and other sources of modulation and/or failure have to be considered. On one hand, it can be argued that the neural adaptations to both cold and hot environments can be linked to a modulation occurring upstream of the motor cortex, commonly referred to as "Motivation," "Arousal" or "Will." On the other hand, neural drive modifications can also be a consequence of some peripheral adaptations occurring at any stage from the motor cortex to the sarcolemma, including the spinal cord.

Spinal modulation

As detailed in "*Neural drive*", environmental conditions have been shown to alter the neural drive of the exercising muscle. This alteration is generally evidenced by changes in EMG activity and can be considered as a physiological failure or a protective mechanism. However, a modification in CNS output is not proof of cortical adaptation but can also be a consequence of a spinal modulation of the supraspinal outputs. There is numerous evidence that spinal loop properties are modified in both cold (*Spinal modulation in cold environment*) and hot (*Spinal modulation in hot environment*) environments as compared with neutral conditions.

Spinal modulation in cold environment

Peripheral regulation of the neural drive is mainly conducted through reflex pathways, with the stretch reflex (T-reflex) playing a major role. Stretch reflex is a monosynaptic, ipsilateral spinal reflex which is activated by stretching the muscle spindles (during the stretch phase of stretch-shortening cycle, tapping the tendon or causing a flexion of a joint), which in turn facilitates the following contraction of the agonist muscle and inhibits the contraction of the antagonist muscle (Matthews, 1964). Both α-motoneuron excitability and γ-motoneuron sensitivity affect stretch reflex responses, which may be divided into short (SL), medium (ML) and long (LL) latency responses. SL is a spinal response, ML is considered to be a delayed spinal response (because of use of slower nerve fibers, e.g. spindle group II afferents) and LL a central (supraspinal) response (Duchateau & Hainaut, 1993; Schieppati & Nardone, 1997). Many studies concerning the effects of cooling on stretch reflex have shown that cooling suppresses

stretch reflex amplitude (e.g. Denys, 1991). There is evidence that the suppressed T-reflex amplitude is due to decreased activity of the muscle spindles and thus decreased γ-motoneuron excitability (Bell & Lehmann, 1987) and these changes may reduce muscle force production. However, it also has been shown that during low-intensity repetitive work in cold (when forearm muscle strain is higher in relation to same work in thermoneutral condition), stretch reflex responses are enhanced. When applying local cooling on the forearm, SL and ML responses are enhanced and when applying whole body cooling also LL response is enhanced (Oksa et al., 2002). This probably indicates that the increased strain of the working muscles were met by increasing the reflex activity during local cooling, therefore, in the cold more muscle fibers are recruited in order to maintain the given work level. However, in the case of whole body cooling also increased supraspinal neural drive from the CNS was required (Oksa et al., 2000) in order to perform the required work. In addition to changes in stretch reflex, substantial alteration in H-reflex and M-wave has been reported (Oksa et al., 2000; Coulange et al., 2006). In the cold air exposure (10 °C, 60 min) Oksa et al. (2000) found increased H_{max}/M_{max}-ratio and H-reflex amplitude reflecting increased excitability of the motoneuronal pool, both γ and α. This increased excitability of the motoneuron pool may possibly be explained by increased sensory input from the cutaneous afferents and/or increased supraspinal drive from the CNS (Oksa et al., 2000, 2002).

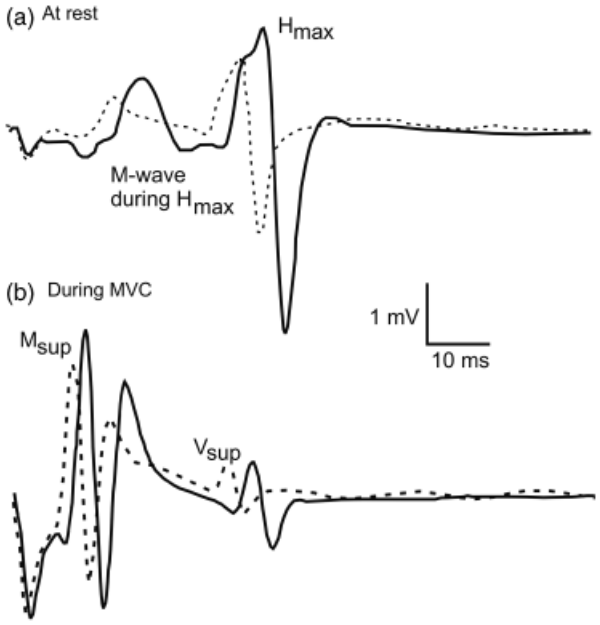

Fig. 3. Example of electrically evoked action potentials recorded at rest (top graph) and during maximal voluntary contraction (MVC, bottom graph) in one subject in both neutral (—) and hot (- - - -) environment. A hot environment significantly decreased the maximal amplitude of an electrically induced action potential (M_{sup}) as well as the maximal amplitude of an electrically induced H-reflex (H_{max}) or its electrophysiological variant during contraction (V_{sup}). Reproduced with permission from Racinais et al. (2008).

Spinal modulation in hot environment

One of the major sites for modulation in the motor drive is the spinal cord. In neutral environments, spinal modulation of the neural drive has been observed after an isometric contraction maintained until exhaustion (Duchateau & Hainaut, 1993; Duchateau et al., 2002) or after submaximal running exercise (Racinais et al., 2007). As this modulation can be partly related to presynaptic inhibition mediated by temperature sensitive group III and IV afferents (Bigland-Ritchie et al., 1986; Woods et al., 1987; Garland & McComas, 1990; Garland, 1991; Duchateau et al., 2002; Avela et al., 2006) a spinal modulation of the neural drive is likely to occur in a hot environment. This is supported by recent findings showing that passive hyperthermia reduces the amplitude of the reflex waves (H-reflex) whether expressed absolutely or normalized by the change occurring at sarcolemmal level (Fig. 3) (Racinais et al., 2008). H-reflex decrements were confirmed by a decrease in V-waves, an electrophysiological variant of the H-reflex (Racinais et al., 2008). These decrements in electrically evoked reflex waves can be linked to both the inhibition/control loop acting presynaptically and alterations in the excitability of the postsynaptic element, i.e. the motoneuron (Racinais et al., 2008). Presynaptically, the decline in transmission from the Ia afferent stimulation to α-motoneuron excitation could be a consequence of a presynaptic inhibition mediated by groups III and IV afferents (Bigland-Ritchie et al., 1986; Woods et al., 1987; Garland & McComas, 1990; Garland, 1991; Duchateau et al., 2002; Avela et al., 2006). The duration of this presynaptic inhibition will depend on whether the input that is producing the inhibition is ongoing or has ceased. As these afferents are sensitive to temperature, which is an ongoing factor when exercise is performed in hot environments, input of groups III and IV muscle afferents could represent a valuable explanation for a decrement in spinal reflexes amplitude (Racinais et al., 2008). However, maintaining the muscle in an ischemic state does not affect the altered responses to transcranial stimulation (Gandevia et al., 1996; Taylor et al., 2000; Andersen et al., 2003) suggesting that groups III and IV muscle afferents do not directly inhibit motoneurons but act upstream of the motor cortex to impair voluntary descending drive (Taylor et al., 2006). Therefore, also alterations in the excitability of the postsynaptic element (i.e., the

motoneuron) has to be considered to explain the decrements previously observed in both muscle electrical activity and H-reflex amplitude.

Peripheral nerve adaptations

A hot environment can increase maximal muscle force via an improvement of the muscular contractile properties (*Neuromuscular performance*); however, hyperthermia reduces muscular performance by decreasing motor drive (*Neural drive*). As detailed above, the decrements previously observed in both muscle electrical activity (*Neural drive*) and H-reflex amplitude (*Spinal modulation*) could be a consequence of an altered excitability of the α-motoneurons and/or the sarcolemma. Various studies in humans and animals suggest that high temperature could reduce the amplitude of the action potential (*Neural and sarcolemmal excitability*). There is also a positive relationship between temperature and nerve conduction velocity with a slowing down of the nerve conduction velocity in cold environment (*Conduction velocity*). In addition, the effect of temperature on a motor unit seems to be dependent on the characteristic of the motor unit (Effect of nerve diameter and motor unit type) and it can be difficult to differentiate *in vivo* the adaptation occurring at the level of the nerve from the muscle.

Neural and sarcolemmal excitability

The decrement in muscle electrical activity generally observed in hot environment has been partly attributed to supraspinal adaptations (see *Neural drive*). However, the decrement in the amplitude of an electrically evoked H-reflex (*Spinal modulation*) suggests that a hot environment also impairs the transmission of the neural drive at peripheral level (i.e., spinal cord or peripherally). Results from animal experiments support this hypothesis. A decrease in the amplitude of an electrically evoked action potential when temperature increases was described 60 years ago in squid axon (Hodgkin & Katz, 1949; Huxley, 1959), frog myelinated fibers (Schaepfle & Erlanger, 1941) or single myelinated fiber of rat ventral root (Bostock et al., 1978). In humans, a negative linear correlation has been observed between temperature and the latency, amplitude, duration and area of the action potential; all these parameters are decreasing when temperature rises (Bolton et al., 1981). At low temperatures, the voltage-gated sodium channel remains open for a longer period of time, increasing the amplitude, duration and area of a single axon potential (Rutkove et al., 1997). The opposite occurs at high temperature, thus reducing the amplitude of the action potential. However, although the effects of temperature are well understood at the single fiber level, complexities arise when studying compound action potentials *in vivo* (Rutkove et al., 1997). For example, opposite effects have been observed depending on whether the entire segment was cooled or only a small area around the recording site (Lang & Puusa, 1980).

The amplitude of an evoked action potential has been observed to be reduced by passive heating of the upper limb following the same pattern as a single motor unit; probably as a consequence of temperature-dependant variations in the ion channel function of both muscle and nerve membrane (Rutkove et al., 1997). Following whole body passive hyperthermia, Racinais et al. (2008) recorded muscle action potentials (M-wave) of humans' in hot environment and found that both the maximal M-wave at rest (M_{max}) and during contraction (M_{sup}) were significantly decreased with hyperthermia (Fig. 3). This suggests that a given amount of (electrically induced) neural drive failed to induce an equivalent sarcolemmal action potential in hyperthermia. This decrement can be explained by failures in the motor nerve and the sarcolemma themselves as well as in the synaptic transmission at neuromuscular junction. *In vitro* studies confirm this by showing modification in synaptic transmission at high temperature (Kelty et al., 2002). The stimulation of a nerve causes the release of a variable number of quanta per impulse during a train of stimuli, but if post-synaptic quantal units go undetected following a stimulus, this is termed as a failure (Karunanithi et al., 1999). It was previously observed that at 22 °C all the synapses produced one or more quantal events for each nerve impulse without any failure. But, as temperature increased, the amplitude of the response declined and failures became evident until transmission completely failed when the nerve temperature reached 35 °C (Karunanithi et al., 1999). However, these observations were carried out *in vitro* in Drosophila synapses with a range of temperatures lower than those recorded *in vivo* in hyperthermic humans.

Interestingly, a decrement in resting M-waves has also been observed after locally heating the leg (Dewhurst et al., 2005), which was attributed to a shortening of the depolarization time when temperature increased, consequently allowing less Na+ to enter the cell (Rutkove, 2001). In addition, cooling the head during whole body hyperthermia does not protect from M-waves decrements in a hot environment (Racinais et al., 2008). Collectively, these data suggest that the decrement in M-wave is not a central adaptation but represents a peripheral failure in the transmission of the neural drive from the α-motoneuron to the sarcolemma or in the motor

nerve and/or the sarcolemma themselves. Given that Rutkove et al. (1997) observed that local warming of the arm reduced the amplitude of the action potential in both sensory and motor nerve without altering the neuromuscular transmission, changes in the α-motoneuron and the sarcolemma have to be considered with hyperthermia. The neuromuscular junction function seems poorly influenced by temperature due to a very high safety factor, with far more acetylcholine released with a given stimulus than necessary to induce a muscle fiber depolarization (Rutkove, 2001). In addition, the synaptic transmission can be partly protected by preconditioning and heat shock proteins (Karunanithi et al., 1999; Kelty et al., 2002).

Conduction velocity

As presented above, a decrease in the amplitude of the action potentials when temperature increases has been described in both animals and humans. However, the average decrease is generally modest (Stegeman & De Weerd, 1982) and a decrease in the duration of these action potentials has even been a more consistent observation and is more marked than the fall in amplitude when temperature increases (Bolton et al., 1981). Inversely, an increase in the duration of the action potential (broadness) has been reported when temperature decreases (Buchthal & Rosenfalck, 1966; Ludin & Beyeler, 1977). A relationship between temperature and the duration of the action potential has been observed even without modification in the amplitude of the response (Todnem et al., 1989).

In addition, temperature also affects the velocity of propagation (De Jesus et al., 1973). Conduction velocity in both sensory and motor fibers increase non-linearly when temperature increases (Todnem et al., 1989). Similarly, distal motor latencies increase also non-linearly when temperature decreases (Todnem et al., 1989). Conduction velocity increases with increasing temperature (Rutkove et al., 1997) but the effect of temperature is most pronounced in the low temperature range (Todnem et al., 1989). This slowing-down in nerve conduction velocity with cooling could induce a slower and weaker muscle contraction. However, the reduced nerve conduction velocity in cold environments may result in an increased temporal summation leading to increased EMG amplitude response (Oksa et al., 2000), which in part may explain the differences found in the literature regarding EMG amplitude responses. The decrease in nerve conduction velocity has been reported to have a Q_{10} (an indicator of temperature sensitivity per every 10 °C change in temperature of a tissue) of approximately 1.4 and the absolute decrement in velocity to vary between 1.1 and 2.4 m/s/ °C (Denys, 1991).

Effect of nerve diameter and motor unit type

The decrement in conduction velocity is dependent on the fiber class, with larger-diameter, faster conducting fibers having higher temperature sensitivity (Gasser & Grundfest, 1939; Douglas & Malcolm, 1955; Goldman & Albus, 1968; Rutkove, 2001). At the level of the muscle, the shortening velocity is also more influenced by temperature changes in fast fibers than in slow fibers (Bennett, 1984) and the fast motor units have been suggested to be more temperature dependant than slow motor units (Faulkner et al., 1990). However, fiber type does not seem to influence the effect of temperature on the time to peak tension (Ranatunga, 1980; Petrofsky & Lind, 1981) or on isometric force (Ranatunga, 1980; Petrofsky & Lind, 1981). As presented above, the effects of temperature on an isolated cell (e.g., nerve or muscle fiber) are easier to characterize than the effect of temperature on humans *in vivo*; and some neuromuscular changes due to environmental conditions could reflect either neural or muscular modifications. For example, cooling can change motor unit recruitment pattern so that with a certain submaximal work level more and faster motor units are recruited in order to maintain the given work level (Rome, 1990; Oksa et al., 2002). This can be seen as significantly increased EMG activity for a given amount of work. Oksa et al. (2002) showed that, during a 10%MVC wrist flexion–extension work, EMG activity was approximately 30% higher during whole body or local forearm cooling than in thermoneutral working condition.

Muscle function

In vivo, the peripheral neural modifications detailed above affect the whole neuromuscular system and the motor nerve cannot be considered without the innervated muscle. A cold environment can slow down the contraction of the muscle (*Slowing of the muscle contraction in cold environment*) and has a potential effect of muscle strain (*Muscle strain in cold environment*). Hot temperatures modify the mechanical properties of the muscle fiber (*Muscle function and temperature* in vitro) but the effects of hot environment on exercising humans are more complex (*Muscle function in hot environment* in vivo).

Slowing of the muscle contraction in cold environment

The decrement in muscular performance in a cold environment can also be explained by alterations in the functional properties of skeletal muscle. Litera-

ture reports that the rate of tension development in the beginning of muscle contraction i.e. the time to maximum force level (twitch or tetanic tension) is temperature dependent (De Ruiter & De Haan, 2000). The temperature sensitivity (Q_{10}) of the rate of tension development was reported to be approximately 1.5 (Ranatunga et al., 1987) but later higher values have also been reported to vary from 2.0 to 3.6 (De Ruiter & De Haan, 2000). Recently it was reported that electromechanical delay (EMD) is also longer in cold. After 30 min immersion on 5 °C water, the EMD was increased by 24.6% (Kubo et al., 2005). Similar temperature dependence has been found also for the rate of relaxation at the end of muscle contraction. It is generally described as half relaxation time i.e. the time from the maximum tension to 50% of the maximum tension and also as late half relaxation time from 50% to 25%MVC. The Q_{10} of the rate of relaxation in humans has been reported to be approximately between 1.7 and 2.3 (Ranatunga et al., 1987) but the more sensitive late half relaxation time has Q_{10} values from 2.1 up to 6.9 (De Ruiter et al., 1999; De Ruiter & De Haan, 2000).

Two other factors that are also important for muscle function are elasticity and stiffness, and the knowledge regarding the effect of cooling on them is sparse. Asmussen et al. (1976) showed that the stiffness (i.e. the ratio between force and length changes) of the muscle–tendon entity does not significantly change due to cooling and more recently Kubo et al. (2005) reported similar results. However, previously it has been reported that at lower temperatures the stiffness of tendons and joints increase (Hunter et al., 1952). Asmussen et al. (1976) studied also the effect of cooling on the capacity of the muscles to utilize their elastic properties by comparing the jump height of static and countermovement jump. It was found that the "gain" in height i.e. the increase in the countermovement jump height in relation to the height of the static jump increased after cooling. Simultaneously, the EMG activity of the working muscles during countermovement jump increased. These results led to the conclusion that the utilization of elastic components of the muscle is enhanced after cooling. However, indirect but to some extent contradictory results have also been presented. Large reductions in performance capacity (17% decrease per 1 °C decrease in muscle temperature) have been reported during a drop jump, an exercise mode that effectively utilizes the elastic components during muscle contraction (Oksa et al., 1997). When comparing this with an exercise of similar nature and duration (upper body exercise, ball throwing) a reduction of only 2.4% was found (Oksa et al., 1995). Therefore, it may be premature to draw definitive conclusions on how cooling effects the ability of the muscles to utilize their elastic properties.

Cooling can also affect muscle co-ordination thus altering the accuracy of motion trajectory and maintenance of force at a desired level. The studies of Meigal et al. (1998, 2003) showed that coefficient of force variation (CoFv) increased in cold conditions and especially when shivering. For example, during shoulder flexion the CoFv in thermoneutral condition was ca. 5%, whereas during shivering it increased to 10% ($P<0.05$). Even though the subjects were able to keep the desired force level fairly accurately the variability of force production around the desired level was greater in the cold and during shivering (Meigal et al., 1998, 2003). More recently, Dewhurst et al. (2007) compared the CoFv between young and older subjects and found that in older subjects CoFv was changed in low (0–3 Hz), middle (4–6 Hz) and high (8–12 Hz) frequency force signal bands, whereas in the young there was only a change in high frequency band. This implies that similarly as performance capacity is gradually decreased when aging proceeds so is the ability to accurately control force production.

It has also been shown that motion trajectory can change due to low ambient temperature. The study of Piedrahita et al. (2008) showed that during simulated packing work the vertical amplitude of the motion trajectory of the shoulder, elbow and wrist were significantly higher when performing the work in cold as compared with a thermoneutral condition. The result was the same regardless whether the work was performed with whole body cooling or in the cold but adequately dressed (Piedrahita et al., 2008). This indicates that the inter-joint coordination of the upper arm in cold is altered in relation to thermoneutral condition and may increase the strain of work.

Muscle strain in cold environment

Recently, it has been found that the so called EMG gaps (a short period, less than a second, of very low muscle activity or even rest) are very sensitive to cold ambient temperature as well as to muscle cooling (Oksa et al., 2006; Piedrahita et al., 2008). These two studies showed that during low intensity repetitive work in the cold the occurrence of EMG gaps was reduced between 2% and 90%, depending on the muscle studied, the great variation being possibly due to different tasks and composition of the muscle. This probably reflects that the normal variation in fiber recruitment has diminished and that only very low threshold fibers may be active throughout the work (Hägg, 1991; Sjögaard & Sögaard, 1998). Previously it has been found that future trapezius myalgia patients had a lower frequency of EMG gaps (<10.8 gaps per minute) than nonpatients

(more than 10.8 gaps per minute) and a regression analysis revealed that low rate of EMG gaps predicts future patient status (Veiersted et al. 1993). It also has been shown that low intensity repetitive work (at 10%MVC level) in cold induces approximately 30% higher strain and fatigue to the working muscles (Oksa et al., 2002) and that, together with lower frequency of EMG gaps, may pose a substantial risk for overuse complaints, symptoms and disorders in the cold.

However, it has been shown that intermittently increasing the work intensity from low to moderate (10% to 30%MVC, every fourth minute) the amount of EMG gaps can be restored from an average of 6.9 ± 1.1 (work only with 10%MVC) to 12.4 ± 1.0 gaps/minute (work intermittently increased to 30%MVC, $P<0.05$) (Oksa et al., 2006). Interestingly, Veiersted et al. (1993) showed that those who were predicted to be at risk for future trapezius myalgia patient status experienced <10.8 gaps/minute. It may be concluded that an increased amount of EMG gaps reflects more evenly distributed work load (Oksa et al., 2006) thus, possibly reducing the risk for overuse symptoms.

Muscle function and temperature *in vitro*

Low muscle temperature potentially slows-down chemical reactions in the muscle (Oksa et al., 1996a, b), delays the cross-bridge cycle (Asmussen et al., 1976) and decreases actomyosin sensibility to calcium (Hartshorne et al., 1972). In hot environments the effects of temperature on time to peak tension, half-relaxation time or maximal shortening velocity are less marked (Bennett, 1984). However, *in vitro* experiments showed that increasing temperature shorten the time to peak twitch tension and the half relaxation time of the muscle in an exponential fashion (Segal et al., 1986). Consequently, the force–time curve is shifted to the right when temperature increases. This shift has been related to the accompanying increases in myosin adenosine triphosphatase (ATPase) activity (Barany, 1967) and calcium sequestration by the sarcoplasmic reticulum (Stein et al., 1982), and increasing the kinetic of the isometric twitch (Close & Hoh, 1968; Ranatunga, 1982). Conversely, decreasing temperature from 35 to 10 °C, *in vitro* (with 5 °C intervals) results in almost a linear decrease in maximum shortening velocity and rate of tension development. When comparing fast (extensor digtorum longum) and slow (soleus) muscles the slow muscle seems to be more temperature sensitive, especially in the lower temperature half (from 20 to 10 °C), than fast muscle (Ranatunga, 1982, 1984). The maximum tetanic force can also be improved by increasing temperature (Close & Hoh, 1968; Stephenson & Williams, 1981; Segal et al., 1986) possibly by improving contractile protein binding (Stephenson & Williams, 1981); however, this may not be the case in all muscles (Segal et al., 1986). However, an increase in temperature across the standard range experienced *in vivo* (i.e., from 37 to 43 °C) has not been observed to modify the absolute force of the muscle fiber (Place et al., 2009).

The temporal characteristics of contraction and relaxation are shortened when temperature is increased (Close & Hoh, 1968; Petrofsky & Lind, 1981; Ranatunga, 1982; Segal & Faulkner, 1985). Thus the stimulation frequency required to sustain a given level of force increased (Segal et al., 1986), increasing the energy cost of the contraction (Rome & Kushmerick, 1983). Consequently, the time to reach fatigue is inversely related to temperature (Segal et al., 1986). However, relationship between fatigue and temperature observed *in situ* (Petrofsky & Lind, 1981) and *in vivo* (Clarke et al., 1958; Edwards et al., 1972) is not linear but bell-shaped. Interestingly, if the number of stimuli required to reach fatigue *in vitro* is analyzed, it also shows a bell-shaped relation with temperature (Segal et al., 1986). Experiments on skinned muscle fiber have shown that inorganic phosphate accumulation decreases force production (Cooke et al., 1988; Godt & Nosek, 1989) and this has been proposed to be as one of the main triggers for muscle fatigue. In addition, the sensitivity of active force to inorganic phosphate decreases when temperature increases from low temperature (e.g., laboratory room temperature) to physiological temperature (Coupland et al., 2001). This suggests that, if inorganic phosphate is the underlying cause of fatigue, fatigue would be less at physiological temperature than at low temperature and, therefore, explain the ascending part of the bell-shaped relation discussed above. Following the same model, while acidosis has large negative effects on contractile function at low temperatures, the effects are limited at higher, more physiological temperatures (Ranatunga, 1987; Pate et al. 1995; Westerblad et al., 1997). However, an increase in muscle temperature increases reactive oxygen and nitrogen species production (Zuo et al., 2000; Van der Poel & Stephenson, 2002; Arbogast & Reid, 2004; Edwards et al., 2007). Recent studies confirm that muscle fiber fatigability decrease when its temperature is increased from low temperature to more physiological values (Roots et al., 2009). However, increasing temperature in the range of the physiological values (i.e., from 37 to 43 °C) does not seem to affect the fatigability of the muscle fiber (Place et al., 2009).

Muscle function in hot environment *in vivo*

In vitro experiments are suitable to determine the intrinsic properties of the muscle. However, *in vivo*,

muscle mechanical responses cannot be limited to the muscle fiber as it also involves the non-contractile material as well as interactions between the different fibers. For example, an increase in temperature decreases the viscous resistance of muscle and joints (Hill, 1927; Buchthal et al., 1944; Wright & Johns, 1961). Increasing temperature can also affect muscle metabolism by improving the release of oxygen from haemoglobin (McCutcheon, 1999) and myoglobin (Theorell, 1934). The rate of ATP utilization has also been proposed to be increased when exercising in the heat; matched by an increase in anaerobic glycolysis and creatinine phosphate hydrolysis (Febbraio et al., 1994, 1996). Lastly, whereas *in vitro* experiments are performed in a controlled environment independently of blood flow or innervations; *in vivo*, hot temperatures will affect the muscle environment by increasing the local vasodilatation (Karvonen, 1978) or changing the properties of the nerve and sarcolemmal action potential (see above).

The outcome is that muscle efficiency (i.e., the level of force for a given quantity of neural drive) can be improved in hot environment as compared with neutral temperature (Racinais et al., 2005). However, given that circadian rhythms can affect the response to a hot environment (for a review see Racinais, 2010), a hot environment only improves muscle contractility in the morning but not in the afternoon (Racinais et al., 2005).

In addition, during exercise, heat production occurs in the muscle leading the temperature of the working mammalian muscle up to 1–2 °C higher than the central temperature (Baracos et al., 1984). When muscle temperature increases, protein degradation increase more than protein synthesis (Baracos et al., 1984) and hyperthermia stimulates the degradation of muscle proteins (Luo et al., 2000). Proteolysis and ultrastructural damage have been observed *in vitro* when muscle temperature increases from 32 to 42 °C (Baracos et al., 1984; Essig et al., 1985) potentially impairing force development. However, tetanic force in *soleus* fibers is not altered when temperature increased from 37 to 43 °C suggesting that muscle fibers can tolerate a temperature $\sim$ 6 °C above *in situ* temperature without displaying any decrease in tetanic force production (Place et al., 2009). Elevating muscle temperature to the extreme physiological range decreases its contractility (Van der Poel & Stephenson, 2002), but this change is reversible (Van der Poel & Stephenson, 2002).

Summary

Exercise capacity has been widely related to both the environmental and body temperatures. Numerous studies have focused on the negative effect of hot environments on the capacity to sustain exercise but temperature is also modifying the neuromuscular function during short duration activity. During short duration exercise, there is a positive relationship between performance and muscle temperature; i.e., neuromuscular function is impaired by cold temperature and improved by hot temperature. The variation in performance ranges from 2% to 5% by 1 °C of change in muscle temperature and is more marked for fast than slow movement. However, if central temperature increases (i.e., hyperthermia), this positive relation stops and performance becomes impaired.

Performance impairments in both cold and hot environments are partly related to a modification in neural drive. The amount of voluntary neural drive is reduced with hyperthermia whereas cold temperatures alter coactivation and coordination. These adaptations are partly linked to physiological failures but could also be partly linked to protective adaptations to reduce the risk of injury or hyperthermia. Neural drive in both cold and hot environments is also affected by changes of the spinal loop properties.

Peripherally, there is a positive relationship between temperature and nerve conduction velocity; i.e., a negative relation between temperature and depolarization time. Consequently, there is also a negative relationship between temperature and the latency, duration (broadness) and the area of an action potential, as well as its amplitude. A positive relation between temperature and contraction velocity has also been observed at the level of the muscle; i.e., a decrement in the rate of both muscle contraction and relaxation when temperature decrease. Increasing temperature can potentially increase the tetanic force by improving contractile protein binding but the shortening of the contraction and relaxation time lead the necessity of a higher stimulation frequency to sustain a given level of force. That potentially increases the energy cost of the contraction and the fatigability of the muscle, but maybe not in the *in vivo* temperature range.

Key words: thermoregulation, muscle, nerve, spinal reflex, central nervous system (CNS).

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