Cold Stress Effects on Exposure Tolerance and Exercise Performance

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ABSTRACT

Cold weather can have deleterious effects on health, tolerance, and performance. This paper will review the physiological responses and external factors that impact cold tolerance and physical performance. Tolerance is defined as the ability to withstand cold stress with minimal changes in physiological strain. Physiological and pathophysiological responses to short-term (cold shock) and long-term cold water and air exposure are presented. Factors (habituation, anthropometry, sex, race, and fitness) that influence cold tolerance are also reviewed. The impact of cold exposure on physical performance, especially aerobic performance, has not been thoroughly studied. The few studies that have been done suggest that aerobic performance is degraded in cold environments. Potential physiological mechanisms (decreases in deep body and muscle temperature, cardiovascular, and metabolism) are discussed. Likewise, strength and power are also degraded during cold exposure, primarily through a decline in muscle temperature. The review also discusses the concept of thermoregulatory fatigue, a reduction in the thermal effector responses of shivering and vasoconstriction, as a result of multistressor factors, including exhaustive exercise. Published 2016. *Compr Physiol* 6:443-469, 2016.

Introduction

People exercise and work in many cold-weather environments (e.g., low temperature, high winds, and immersion). For the most part, cold-weather is not a barrier to performing physical activity. Successful and safe exploration to high altitude, polar regions, and swimming for hours across the English Channel are clearly indicative that human beings can perform in extreme cold (38). Tolerating and performing in cold-weather environments has typically not received as much academic interest as other environmental extremes, even though a lack of tolerance during cold exposure can lead to life-long, life-altering injuries, and even death.

There are scenarios (immersion, rain, and low ambient temperature with wind) in which whole-body or local thermal balance cannot be maintained during exercise-cold stress, contributing to cold-weather injuries, survivability, and diminished exercise capability and performance. This article does not deal with the pathophysiological consequences of cooling (cold injury, hypothermia, and drowning); instead it focuses on the cold-evoked physiological mechanisms, and internal and external factors that alter physiological responses, tolerance, and performance in the cold.

Biophysics of Heat Exchange and Balance

Changes in deep body temperature are simply caused by either a positive or negative change in heat storage. If the body produces more heat than it dissipates, deep body temperature rises. Conversely, if heat production is less than that lost to the external environment, then heat storage will be negative and deep body temperature will fall. We can present these relationships between production and loss mathematically as follows (92):

$$S = M - (+\text{Work}) - E \pm R \pm C \pm K$$

where: S = heat storage, M = metabolic heat production, E = evaporation, R = radiation, C = convection, and K = conduction. Positive numbers indicate heat gain and negative values, heat loss. E, R, C, and K are the heat exchange pathways (107). All units are in $W \cdot m^{-2}$. When environmental temperature and water vapor pressure are lower than these values at the skin, it results in a loss of heat from the body.

Physical exercise can increase whole body metabolism (M) by as much as 15 to 20 times the resting metabolic rate in healthy young males. But since exercise only uses 9% to 20% of this increased metabolism to produce useful work (i.e., 9%-20% efficiency), the balance of the increased metabolism is given off as heat. Exercise M only contributes to heat gain.

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Heat loss occurs through radiation, convection, conduction, and evaporation. Radiation is the movement of heat via electromagnetic waves. In this instance, heat moves down its thermal gradient from high to low. For example, heat is lost from the body to the environment when the skin temperature exceeds air temperature. Convection is the movement of heat down its thermal gradient from an object (body) to a surrounding liquid medium. In this case, liquid may mean air, water or internal body fluids. Conduction is the movement of heat between two objects that are in direct contact with each other, which can become an important contributor to heat loss during cold exposure. Evaporation is the loss of heat through the process of changing sweat from a liquid phase to a gaseous phase. This phase change requires the input of heat from the skin, the "latent heat of vaporization of water."

Although only two primary pathways for heat loss are available in water (convection and conduction), humans cool two to five times more quickly in cold water, compared to air at the same temperature (128). Water can be up to 11°C higher than air and produce an equivalent physiological response (91). Furthermore, thermoneutral temperature in water averages about 35°C with a very narrow range (54, 55); this compares with about 26°C and a broader range for still air (55). Therefore, a naked resting man will be unable to maintain his deep body temperature when immersed in water at a temperature regarded as thermoneutral in air.

The reason for these differences lies in the physical properties of air and water; the thermal conductivity of water is 25 times that of air (247) and water has a specific heat per unit volume that is approximately 4000 times that of air. Thus water, unlike air, provides practically no insulation at the skin-water interface. Therefore, cooling is extremely effective in water and results in rapid dissipation of the heat which is delivered to the skin from the deeper tissues. As a consequence, skin temperature quickly approximates water temperature following cold water immersion. In contrast, the lower cooling capacity of air often allows skin temperature to be adjusted independently of air temperature; therefore, it can assist in the maintenance of heat balance.

The cooling capacity of water is reflected in the values which have been reported for the combined heat transfer coefficient for convection and conduction. In still water, the value is between 44 and 230 $W \cdot m^{-2} \cdot °C^{-1}$ (52, 170, 267), rising to 460 at rest in moving water and 580 during swimming at any speed (170). These figures are difficult to obtain and vary greatly between studies largely because of methodological differences. However, they are at least two orders of magnitude greater than those reported for air.

In a cold-weather environment, although the ambient temperature may be low, clothing insulation can create a relatively warm "micro-environment" in which sweating may become more prevalent and evaporative heat loss an important contributor to maintaining heat balance. In this situation, exposure or exercise in the cold does not increase strain or injury risk, and leads to the concept that "man in the cold is not necessarily a cold man" (9). This concept has important implications for tolerance and performance.

Physiological Responses to Cold

Humans exhibit peripheral vasoconstriction upon exposure to cold. The resulting decrease in peripheral blood flow reduces convective heat transfer between the body's deep body and superficial tissues (skin, subcutaneous fat, and skeletal muscle), effectively increasing the insulation of the body. Since heat is lost from the exposed body surface faster than it is replaced, skin temperature declines. During whole-body exposure to cold, the vasoconstrictor response extends beyond the fingers and spreads throughout the entire body's peripheral circulation. During whole body cooling, vasoconstriction begins when skin temperature falls below about 35°C and becomes maximal when skin temperature is about 31°C or less (253). Thus, the vasoconstrictor response to cold exposure helps retard heat loss and defend deep body temperature, but at the expense of a decline in the temperature of peripheral tissue. In lean individuals, peripheral blood flow is lowest, and maximum insulation is achieved during head-out immersion in water below 33 to 30°C (30, 33, 199). In comparison, air temperatures of 10 to 16°C elicit maximal vasoconstriction and insulation (20, 213). There is further evidence (33, 213, 246) to suggest that fatter individuals do not achieve maximum tissue insulation until they are immersed in much lower water temperatures, as low as 12°C in some cases (33).

Vasoconstriction in a cutaneous region can occur due to direct local cooling or as a result of vasoconstriction evoked due to cooling in other cutaneous regions or the deep body. Norepinephrine (NE) is the primary neurotransmitter that accounts for ~60% of the generalized cold-induced vasoconstriction in skin, while neuropeptide Y is responsible for ~20% to 30% (217, 218). In contrast, early in the local cooling response (with no vasoconstrictor input occurring from other areas), smooth muscle vasoconstriction is mediated by NE and Rho kinase (225). However, as local skin cooling continues down to 24°C, sympathetically mediated adrenergic vasoconstriction appears to be responsible for ~20% of cold-induced vasoconstriction whereas nonadrenergic mechanisms (RhoA/rho kinase) mediate ~60% of the response (137, 225). Local cooling enhances α_2 receptor sensitivity in vascular smooth muscle to NE by translocating α_{2C} receptors from the Golgi apparatus to the plasma membrane (45, 46, 137).

Cold exposure also elicits increased metabolic heat production in humans, which can help offset heat loss. In humans, most cold-induced thermogenesis is attributable to skeletal muscle contractile activity. Humans initiate this thermogenesis by voluntarily modifying behavior, that is, increasing physical activity (e.g., exercise and increased fidgeting), or involuntarily via shivering.

Shivering, which consists of repeated rhythmic muscle contractions during which most of the metabolic energy expended is liberated as heat and little external work is performed (efficiency near zero), may start immediately or after several minutes of cold exposure. It usually begins in torso muscles and then spreads to the limbs (11). The intensity and extent of shivering vary according to the severity of cold stress. As shivering intensity increases and more muscles are recruited to shiver, whole-body metabolic rate increases, typically reaching about 200 to 250 W during resting exposure to cold air but often exceeding 350 W during resting immersion in cold water. Maximal shivering is difficult to quantify, but the highest metabolic rate reported in the literature to date appears to be 763 W, recorded during immersion in 12°C water, and this corresponded to 46% of that test subject's maximal aerobic power (102). Shivering can still occur during exercise but it primarily occurs at low relative exercise intensities. Hong and Nadel (127) showed that the thermoregulatory oxygen consumption and skin surface EMG activity superimposed on exercise diminished as exercise intensity increased from free pedaling to 60 W in 10°C air at any given esophageal temperature. In more severe environmental conditions (5°C air, wind, and wet clothing), Pugh et al. (194) and Weller et al. (259) also observed higher oxygen consumption levels (i.e., shivering) at an exercise intensity of ~30% VO_{2max}, whereas at 60%V_{O2max}, there was no difference in VO_2 in the severe cold conditions versus less severe exposure. Furthermore, the slopes of the VO_2-T_{es} and surface EMG-T_{es} relationship was significantly less as exercise intensity increased.

It has been well established that rodents increase metabolic heat production by brown adipose tissue (BAT) in response to cold exposure, and it was thought for many years that adult humans lacked this mechanism. However a series of papers (203, 250, 254) using positron emission tomography and computed tomography (CT) scans along with the tracer 18F-fluorodeoxyglucose has revealed that adult humans do indeed have active BAT that becomes active upon cold exposure. BAT thermogenesis in humans has recently been reviewed by Blondin et al. (24).

Exposure Tolerance

We have defined cold tolerance in this paper as "the ability to withstand cold stress exposure with no or a minimal change in physiological strain." In cold environments, as with other extreme environments, avoiding any change in physiological strain is close to impossible to achieve; this is broadly evidenced by poorer elite athletic performance and occupational effectiveness in thermal environments that vary too much from thermoneutral. So it is that, depending on the exposure, the impact of cold environments on individuals tends to range from mild degradations in performance to tissue damage (frostbite, nonfreezing cold injury), to life-threatening pathological conditions (hypothermia). This section reviews the impact of short- and long-term cold water and air exposure on physiological responses and the potential harmful effects.

Cold water immersion

Tolerance to cold water immersion is determined by the sequence of body tissues influenced by the cold. First the cold receptors of the skin respond to sudden cooling with the "cold shock" response. Physical performance, initially hand function, is then impaired as the superficial nerves and muscles are cooled; subsequent cooling of deeper muscles impairs whole body physical activity and incapacitates the individual. Finally, deep body tissues begin to cool and the problems associated with cooling become life-threatening due to hypothermia. The speed at which this progression proceeds is determined by many factors including environmental (e.g., water temperature and flow rate), individual (e.g., size, fatness, and fitness) and the level of protection (clothing) worn. In the following sections, the responses to short and prolonged immersion are discussed. Cold incapacitation will be primarily discussed in the section on whether to swim to safety and also in the context of exercise performance in the strength/power section of the manuscript.

Short-term water immersion

Sudden immersion in cold water evokes a group of cardiorespiratory responses which are collectively known as the "cold shock" response and is characterized by an inspiratory gasp, hyperventilation, hypocapnia, increased heart rate, peripheral vasoconstriction, and hypertension (59, 236). This response is initiated by a rapid fall in skin temperature. Interestingly, if the arms and legs are covered, the initial rise in heart rate is lower upon cold water immersion compared to if only the torso is covered (234). No differences exist between arm/leg and torso protection for the respiratory responses.

Respiratory drive is enhanced on immersion in water cooler than 25°C (141). It is inversely related to water temperature, reaching a maximum level in 10 to 15°C water (241). The inspiratory gasp response is between 2 and 3 L (109,241) and is followed by uncontrollable hyperventilation, which can result in a 10-fold increase of minute ventilation (234) and hypocapnia (140). The respiratory drive evoked by cold water immersion can reduce the maximum breath hold times of normally clothed individuals to less than 10 s (242) and significantly increases the chance of aspirating water and drowning during the first few minutes of immersion. The hypocapnia probably accounts for the tetany, disorientation, and clouding of consciousness observed with cold water immersion (5, 53, 104). Furthermore, there is an inspiratory shift in end-expiratory lung volume which can result in the occurrence of tidal breathing within 1 L of total lung capacity (141, 241). This response makes breathing very difficult and probably

contributes to the sensation of dyspnea experienced on initial immersion (141). As well, 30 s of immersion in 0°C water reduces cerebral perfusion by 43% (158). Furthermore, several individuals exhibited presyncopal symptoms when perfusion was reduced >60%.

The precise afferent pathways responsible for the respiratory responses to cold water immersion remain to be elucidated. It has been suggested that thermoafferents from the peripheral cold receptors directly stimulate the respiratory center (109). Keatinge and Nadel (141) concluded that the reflex respiratory responses to cold water in the cat are mediated at midbrain level and that the cerebrum is not essential for the response. The earlier work of Lumsden (149) and St. John et al. (215) sheds light on the neural pathways responsible for gasping. In vagotomized animals, eupnea is transformed to gasping following removal of the pons or during hypoxia (150). Furthermore, destruction of neurons in the rostral medulla with neurotoxins leads to the elimination of gasping but not eupnea (216), indicating that the neural pathways for gasping (an area in adult cats extending from dorsomedial and ventrolateral medulla, termed the pre-Bötzinger complex, to the nucleus ambiguus) may be distinct from those necessary for the generation of eupnea. That the first respiratory response to immersion in cold water is a gasp indicates that the cold thermoreceptor volley elicited by cold immersion excites this area.

Tipton and Harris [cited in Ref. (59)] have identified, using c-*fos* protein immunohistochemistry, the neuronal cell groups activated following the cold shock response evoked by 60-s upright immersion to the diaphragm of rats in 8°C water. The c-*fos*-positive neurons were identified in the nucleus tractus solitarius, area postrema, and dorsal motor nucleus, that is, areas known to process cardiovascular and respiratory afferents. No expression was observed in animals immersed in 39°C. Analogous studies from other groups show the pathway for noxious cold stimuli (hindlimb immersion in 4°C water) causes c-*fos* activation of neurons in lamina I of the spinal dorsal horn (1, 245). Another group showed that this c-*fos* immunoreactivity coreacts with NK-1 receptor staining, indicating that those receptors may mediate some of the noxious cold response (66). Other studies show that facial immersion in 4°C water causes c-*fos* activation of neurons in the dorsal horn of the medulla (67). These neuroanatomic studies corroborate the hypothesis that ascending neurons involved in transmitting the thermoreceptor volley following cold immersion are in the pons (4,125) very close to the neurons involved in the generation of gasping.

The initial cardiovascular responses to immersion include intense vasoconstriction, a 42% to 49% increase in heart rate, and a 59% to 100% increase in cardiac output (123, 140). As a result of these responses arterial and venous pressures are increased. The early cardiovascular responses to immersion in cold water place a significant and sudden strain on the system and are a particular threat to people with coronary heart disease, in whom myocardial ischemia is more likely to develop with sudden increases in cardiac workload. Hypertensive or aneurismal individuals are also at risk from the sudden elevations in blood pressure on immersion.

Cold shock habituation

Cold tolerance can be improved by reducing the initial cold shock response with habituation (106). Tipton et al. (239) showed that repeated immersions in 15°C water reduced respiratory frequency, minute volume, and HR during immersion in 10°C water; a decrease in deep body temperature is not required to develop an attenuated response (243). A follow-on study examined whether habituation reduces the cold shock response through a central or peripheral mechanism (232). Pre- and post-habituation experiments were conducted with only the right side of the body exposed to 10°C water, but the six habituation immersions were done with only the left side exposed. Following the habituation immersions the cold shock response was attenuated, suggesting the adaptation occurs through a central mechanism. Another experiment (235) examined the permanence of habituation. Participants were immersed for 4 straight days and the initial responses were examined at day 5, and 2, 4, 7, and 14 months later. The cold shock response was reduced by 40% after 5 days, and up to 31% 7 months later, even without any intervening cold water exposures. A series of other papers has shown that cold showers (74), psychological training (7, 8), and a combination of multiple cold water immersions plus mental skills training (56) showed a positive habituation response. Barwood et al. (6) found that the normally attenuated HR response observed with habituation was abolished when acute anxiety was present.

Little is known about the pathways involved in habituation of the cold shock response. The frontal areas of the cerebral cortex have been suggested to be needed to develop habituation; data from rats indicate that bilateral frontal lesions prevent the habituation of the heart rate response (100). Data from humans using chlorpromazine (112) also suggest the frontal cortex area is important for habituation. However the mechanism for this habituation is not known since chlorpromazine blocks cholinergic, adrenergic, dopaminergic, and histamine receptors.

Facial cooling, cold shock, and autonomic conflict

Sudden cold water immersion of the face during submersion (head in) is also associated with the diving response. This is characterized by bradycardia, apnea, and vasoconstriction in the trunk and limbs and is evoked by stimulation of the ophthalmic division of the trigeminal nerve (110). Thus, during whole body submersion in cold water, both the cold shock and diving response are elicited with concurrent stimulation of, respectively, the sympathetic and parasympathetic branches of the autonomic nervous system. It has been suggested that simultaneous activation of both branches can very frequently lead to dysrhythmias and arrhythmias which, in the presence of predisposing factors (e.g., cardiac

long *Q-T* interval), result in fatal arrhythmias on immersion and in other situations. This coactivation of sympathetic and parasympathetic activity has been called "autonomic conflict" (212, 240). In most cases, and during head-out immersion, sudden exposure to cold water triggers a primary sympathetic response and resultant tachycardia. However, if the face is also immersed and the diving response is evoked, it is more likely that the diving/parasympathetic response will come into conflict with the sympathetic nervous response and increase the likelihood of cardiac arrhythmias. This is more apparent if the cold shock/sympathetic response is attenuated due to clothing, habituation, aerobic fitness, or postural changes. The arrhythmias that are most apparent are supraventricular and junctional in nature (59, 240), they appear linked to respiration (212), occur between periods of tachycardia and bradycardia, and usually within 10 s after the break in breath holding (59). Figure 1 graphically presents the concept of autonomic conflict.

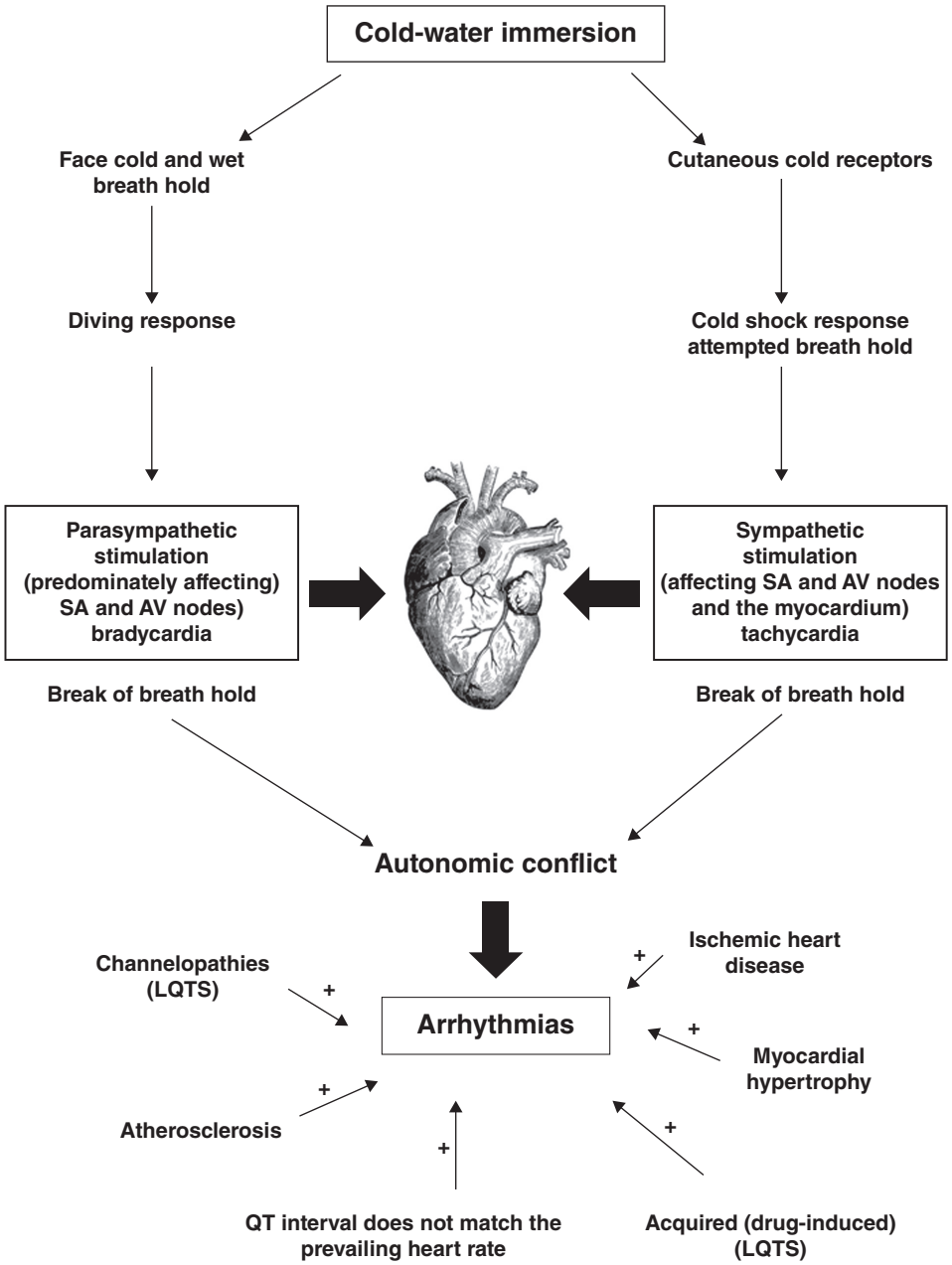

Figure 1 Mechanisms of how cold-water immersion induces autonomic conflict and cardiac arrhythmias. Dual stimulation of a bradycardic and tachycardic cardiac response caused by the diving and cold shock responses, respectively, places the heart in conflict leading to arrhythmias when breath holding ceases. Arrhythmias are more prevalent in individuals with predisposing factors. The magnitude of these responses can vary due to water temperature, clothing, and cold habituation. Adapted, with permission, from Ref. (212).

Prolonged water immersion

Prolonged immersion in water at temperatures below thermoneutrality will inevitably result in hypothermia, that is, a deep body temperature below 35°C. Hypothermia is unlikely to be a problem within 30 min of immersion in water even as low as 5°C, and times to onset of hypothermia will vary between individuals for the reasons given in a later section. As a general rule, however, the deep body temperature of those wearing ordinary clothing will, on average, have fallen to 35°C after 1 h in water at 5°C, after about 2 h in water at 10°C, and after 3 to 6 h in water at 15°C (164). The 50% survival times quoted for prolonged immersion (164) do not directly relate to death from hypothermia, but reveal the time to physical incapacitation due to muscle and peripheral nerve cooling.

Before general hypothermia becomes established, locomotor impairment will produce a deterioration in swimming performance through delays in muscle cell membrane repolarization as a consequence of the cooling of intracellular enzymes. The tone of both protagonist and antagonist muscle groups gradually increases to such a level that swimming becomes virtually impossible; shivering becomes intense and eventually the body tends to assume a semi-rigid, fetal position. Unless extraneous aids to flotation are available, the individual will be unable to maintain the airway clear of the water and drowning will result before deep body temperature falls to a level where cardiac arrest from hypothermia would normally be expected to occur, that is, a myocardial temperature below 28°C, but more usually 24 to 26°C (38).

At cardiac temperatures below 28°C, the conduction velocity of the Purkinje tissue is slowed to approximate that of myocardial fibers. This predisposes the heart to ventricular fibrillation. If a flotation aid such as a life jacket with a splash guard is available, then the airway should remain protected even when unconsciousness from hypothermia occurs, at a deep body temperature of about 30°C (38).

An exception to the general sequence of events outlined above can be found in long-distance outdoor swimmers who are habituated to cold water. Indeed, some swimmers appear to be able to "swim to unconsciousness" probably because they are able to exercise at such high intensities that they end up mixing the superficial and deep tissue/body temperatures. Thus the limbs and deep body cool at about the same rate and unconsciousness occurs (30-33°C) before impaired muscle function (28°C) (237). The ability of these swimmers to withstand prolonged immersion in cold water without apparent ill-effect was attributed to their unique combination of physical fitness and substantial thickness of subcutaneous fat (191). This enabled them to maintain a steady work rate for several hours and to retain much of the heat produced within the body. However, many contemporary outdoor distance swimmers are not as fat as those described by Pugh and Edholm (191). Golden et al. report that the average % body fat in male swimmers before the 1978 Windermere International Race was 18% (103). In an experiment on three long-distance swimmers, the value of cold habituation was clearly demonstrated (103). The two swimmers who routinely trained in cold water swam in 17.4°C with no ill effects. One swimmer completed the crossing of Lake Windermere (16.5 miles) in just under 6 h with an end-swim rectal temperature of 36.1°C, whereas the other swimmer, who was swimming the race the next day, got out voluntarily after 3 h with a rectal temperature of 36.8°C. The swimmer who trained in a heated pool swam for 130 min, became incapacitated, and his deep body temperature was 35.3°C. Conversely, the apparent absence of cold habituation in other published accounts (16,73,118) may explain why some swimmers failed to complete the required distance because of hypothermia, despite relatively high water temperatures (18–19°C) and short swim times (less than 3 h) (16,73). Recent work (19,237) also seems to confirm Golden et al.'s hypothesis that outdoor swimmers demonstrate a form of "insulative" adaptation when swimming in cold water (better able to defend deep body temperature) but a "hypothermic" adaptation (low metabolism, increased comfort, and rapid fall in deep body temperature) when sitting still in cold water. The role of BAT and nonshivering thermogenesis in the insulative response has not been assessed in this group.

Successful outdoor long-distance swimmers would thus appear to be those who are physically fit with a good swimming technique, moderately fat, and habituated to the water temperature at which they are to compete. Lean fast swimmers will be successful in warmer water, but in colder water fatter swimmers are likely to have an advantage. There are no slow, thin cold water swimmers! A negative aspect of the habituation of distance swimmers may be that the incipient onset of hypothermia may go unnoticed in the absence of subjective discomfort and other signs such as shivering (103). This raises the possibility of cold habituated individuals, including those living on the streets and the elderly, becoming hypothermic without knowing or, in the case of swimmers, before swimming is seriously impaired (118). Then, with the cessation of swimming and therefore heat production, heat reserves from the deep body store will continue to be lost to the substantial heat sink in their subcutaneous fat, thereby reducing the interval of useful consciousness from the initial prodromal symptoms to collapse. Encouragingly, recent evidence (243), suggests during resting exposures in those with hypothermic adaptation to cold, an "unhabituated" metabolic response to cold is restored when deep body temperature falls below the levels repeatedly experienced during cold exposure.

An interesting question that has intrigued thermal and exercise physiologists is whether a person who finds themself in cold water should try to swim to safety (147) before their muscles become incapacitated. Advice from 40 to 50 years ago stated that individuals should float in their life jacket or hang on to something to remain above water. Ducharme and Lounsbury (71) recently reviewed the findings of multiple studies and concluded that: (i) individuals can swim 800 to 1500 m in 10 to 14°C water before they reach swim failure; and (ii) swimming duration was 47 min before reaching swim failure, no matter what the swimming ability was. They

concluded that a self-rescue swimming attempt is an option if the swimmer believes they can reach safety within 45 min. The most important factor for prediction of swimming distance while wearing a personal flotation device was triceps skinfold thickness (257). Tipton et al. (233) reported that the reduction in swimming performance in cold water (as low as 10°C), as defined by the change in swimming efficiency, was most closely correlated with sum of the skinfold sites of the upper limbs (triceps, outer forearm, and subscapula, $R^2 = 0.68$). Lounsbury et al. (148) examined whether providing arm insulation would reduce the incidence of swim failure and improve performance times. They did find a significant reduction in deep body temperature cooling rates when swimming in 10°C water when extra insulation was added to the arms.

It follows from the above that the mode of exercise might be critical in determining the thermal responses to immersion. Indeed, the results of a limited number of studies on this topic suggest that the arms are a major source of heat loss during swimming in cold water and leg-only exercise may be preferable in terms of deep body temperature maintenance when compared to whole body swimming and rest (105, 248, 249).

Predicting survival time during cold water immersion has been of keen interest for many decades, primarily for directing search and rescue missions and clothing development (101). Molnar (164) was the first to examine the relationship between ocean temperature and survival time. He found that above ocean temperatures of 16°C, survival time increased quickly. However, below a water temperature of 15°C, survival time was reduced dramatically (~2-3 h in 5°C water, Fig. 2). Since the publication of this first survival curve, others have developed curves to develop decision aids for determining the time to unconsciousness and death (121, 122).

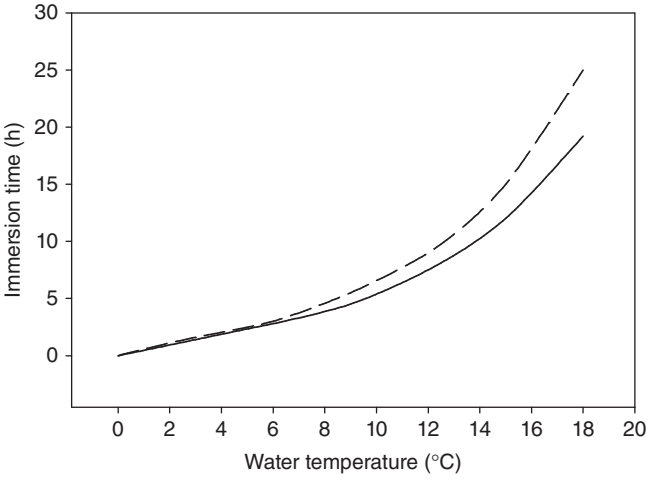

Figure 2 Survival time during immersion as a function of water temperature. Below the solid line, survival is likely; above the dashed line, survival is unlikely. The 50% mortality rate is indicated in the space between the two survival curves. Adapted, with permission, from Ref. (182).

Most of these predictions are very similar to the original times published by Molnar (164). Hayward developed a simple algorithm using water temperature to predict survival time (122). More sophisticated thermal models using biophysical principles and physiological control systems have also been employed to determine multiple end-points including time to hypothermia (35°C), swimming muscle failure, and probability of survival (35,229,230,266,269,272). The Cold Exposure Survival Model and the Probability of Survival Decision Aid are two recent thermoregulatory models used by the search and rescue community (226, 269). Xu and Tikuisis recently reviewed the cold thermoregulatory models (271).

Cold air exposure

With the exception of the cold shock response, the physiological responses to cold air exposure are the same as cold water immersion (increased vasoconstriction, metabolic heat production, and increased sympathetic activity). However, because heat transfer is much less in air versus water, cold tolerance is much greater compared to immersion. As a comparison, the critical temperature, that is, the lowest temperature before there is an increase in metabolic heat production is between 30 and 34°C in water, whereas in air it ranges between 20 and 27°C. Similarly, the water and air temperatures that cause an equivalent resting heat loss (~2× observed in thermoneutral conditions) in a person with a normal skinfold thickness are 29 and 11°C, respectively. As stated earlier, immersion in 5°C water could lead to life-threatening hypothermia in a couple of hours, whereas exposure to 0 to 5°C air for days may cause hypothermia, but not death (181).

The principal danger and threat to tolerance during cold air exposure is an increased risk of peripheral cold injury, primarily frostbite. The skin surface freezes between −3.7 and −4.8°C (58, 165, 262), due to the electrolyte content of cells and fluids, although wet skin freezes at a higher threshold [~−0.6°C, (139)].

The primary determinants of frostbite risk are air temperature, wind speed, and wetness. Most body heat loss during cold exposure occurs through radiation, conduction, and convection, so when ambient temperatures are colder than body temperatures, the thermal gradient favors body heat loss (107). Wind exacerbates heat loss by facilitating convective heat loss (92) and reduces the insulative value of clothing. The Wind Chill Temperature (WCT) Index integrates wind speed and air temperature to provide an estimate of the cooling power of the environment (171, 180). The WCT standardizes the cooling power of the environment to an equivalent air temperature for calm conditions.

WCTs are specific in their correct application, only estimating the danger of cooling for the exposed skin of persons walking at 1.3 $m \cdot s^{-1}$. The WCT presents the relative risk of frostbite and the predicted times to freezing (Fig. 3) of exposed facial skin (69). Facial skin was chosen because this area of the body is typically not protected. Wet skin exposed to the wind will cool even faster and if the skin is wet and exposed to

Wind speed (mph) ↓	Air temperature (°F)																	
	40	35	30	25	20	15	10	5	0	−5	−10	−15	−20	−25	−30	−35	−40	−45
5	36	31	25	19	13	7	1	−5	−11	−16	−22	−28	−34	−40	−46	−52	−57	−63
10	34	27	21	15	9	3	−4	−10	−16	−22	−28	−35	−41	−47	−53	−59	−68	−72
15	32	25	19	13	6	0	−7	−13	−19	−26	−32	−39	−45	−51	−58	−64	−71	−77
20	30	24	17	11	4	−2	−9	−15	−22	−29	−35	−42	−48	−55	−61	−68	−74	−81
25	29	23	16	9	3	−4	−11	−17	−24	−31	−37	−44	−51	−58	−64	−71	−78	−84
30	28	22	15	8	1	−5	−12	−19	−26	−33	−39	−46	−53	−60	−67	−73	−80	−87
35	28	21	14	7	0	−7	−14	−21	−27	−34	−41	−48	−55	−62	−69	−76	−82	−89
40	27	20	13	6	−1	−8	−15	−22	−29	−36	−43	−50	−57	−64	−71	−78	−84	−91
45	26	19	12	5	−2	−9	−16	−23	−30	−37	−44	−51	−58	−65	−72	−79	−86	−93
50	26	19	12	4	−3	−10	−17	−24	−31	−38	−45	−52	−60	−67	−74	−81	−88	−95
55	25	18	11	4	−3	−11	−18	−25	−32	−39	−46	−54	−61	−68	−75	−82	−89	−97
60	25	17	10	3	−4	−11	−19	−26	−33	−40	−48	−55	−62	−69	−76	−84	−91	−91

Frostbite times

Light gray – Frostbite could occur in 30 min
Medium gray – Frostbite occur in 10 min
Dark gray – Frostbite occur in 5 min

Air temperature (°C)

Wind speed (km/h)	5	0	−5	−10	−15	−20	−25	−30	−35	−40	−45	−50
5	4	−2	−7	−13	−19	−24	−30	−36	−41	−47	−53	−58
10	3	−3	−9	−15	−21	−27	−33	−39	−45	−51	−57	−63
15	2	−4	−11	−17	−23	−29	−35	−41	−48	−54	−60	−66
20	1	−5	−12	−18	−24	−30	−37	−43	−49	−56	−62	−68
25	1	−6	−12	−19	−25	−32	−38	−44	−51	−57	−64	−70
30	0	−6	−13	−20	−26	−33	−39	−46	−52	−59	−65	−72
35	0	−7	−14	−20	−27	−33	−40	−47	−53	−60	−66	−73
40	−1	−7	−14	−21	−27	−34	−41	−48	−54	−61	−68	−74
45	−1	−8	−15	−21	−28	−35	−42	−48	−55	−62	−69	−75
50	−1	−8	−15	−22	−29	−35	−42	−49	−56	−63	−69	−76
55	−2	−8	−15	−22	−29	−36	−43	−50	−57	−63	−70	−77
60	−2	−9	−16	−23	−30	−36	−43	−50	−57	−64	−71	−78
65	−2	−9	−16	−23	−30	−37	−44	−51	−58	−65	−72	−79
70	−2	−9	−16	−23	−30	−37	−44	−51	−58	−65	−72	−80
75	−3	−10	−17	−24	−31	−38	−45	−52	−59	−66	−73	−80
80	−3	−10	−17	−24	−31	−38	−45	−52	−60	−67	−74	−81

Frostbite guide

Low risk or frostbite for most people
Increasing risk of frostbite for most people in 10 to 30 min of exposure
High risk for most people in 5 to 10 min of exposure
High risk for most people in 2 to 5 min of exposure
High risk for most people in 2 min of exposure or less

Figure 3 WCT Index in Fahrenheit and Celsius. Frostbite times are for exposed facial skin. Top chart is from the U.S. National Weather Service; bottom chart is from the Meteorological Society of Canada/Environment Canada.

wind, the ambient temperature used for the WCT table should be 10°C lower than the actual ambient temperature (26).

As stated above, WCTs are used to estimate frostbite risk on the cheek. However, fingers will cool more rapidly than the face and frostbite certainly is more prevalent in the fingers compared to the cheek. To this end, Tikuisis, modeled heat loss in the finger and presented modified wind chill charts for uncovered fingers (227). He found that the time to finger freezing was more rapid compared to the cheek, by a factor of ~8. For example, at an air temperature of −50°C and a 10 km·h^{-1} wind speed, the predicted time to finger freezing is 1 to 2 min whereas the time to cheek freezing was >8 min.

Physical activity is an effective countermeasure to increase covered skin temperature when there is no wind. For example, at rest in −10°C air with no wind, the gloved finger temperature is ~18°C. As metabolic heat production increases twofold to fourfold, finger temperature rises to 22 to 27°C (155). However, if conditions are windy, physical

activity does not significantly alter the temperature of exposed or covered fingers. Exposure to a 5 $m \cdot s^{-1}$ wind at an ambient temperature of −10°C when performing light to moderate physical activity only raises the finger temperature in a glove from 10°C at rest to ∼13°C. However, increasing the exercise intensity from 220 to 350 Watts increases nose temperatures from 4.5 to 8.9°C, even in a 5 $m \cdot s^{-1}$ wind (26, 97) and Brajkovic and Ducharme (26) found that nose skin temperature rose from 9.7°C at rest to 18.1°C during exercise.

Physiological model simulations were run for two sedentary cold-air exposures (−10°C, 5 $m \cdot s^{-1}$ wind); in one, the clothing insulation was set at 1.2 clo, equal to that of a typical business suit, with no other clothing worn. In the other, the clothing insulation was 3.0 clo, equal to a multilayer cold weather clothing system, including winter boots, hat, and gloves. The model predicted that clinical hypothermia (35°C deep body temperature) was reached in 11 h while wearing 1.2 clo. For the winter clothing condition, hypothermia was never reached; the predicted deep body temperature was 36.3°C after 72 h of cold exposure. Hand temperatures were also predicted. In the scenario where 1.2 clo was worn, with no handwear, the predicted hand temperature was −3.3°C after 2 h of exposure. In the winter clothing scenario using a glove with a clo value of 1.25, the predicted hand temperature after 72 h of exposure was 6.3°C.

Individual differences and tolerance

Many different factors impact deep body and peripheral cooling rates upon cold exposure. Particularly, one of the difficulties in describing the responses is the wide variation encountered among individuals. Individual differences in body composition and morphology, sex, age, race, and fitness are all sources of variation in thermal responses to cold exposure.

Most of the variability between individuals in their thermoregulatory responses and capability to maintain normal body temperature during cold exposure is attributable to anthropometric differences. In general, persons with a large surface area to mass ratio (i.e., smaller people) have greater declines in body temperature during cold exposure than those with a smaller ratio (92, 247).

All body tissues provide thermal resistance to heat conduction (i.e., insulation). In resting individuals, unperfused muscle tissue provides a significant (up to 75%) contribution to the body's total insulation (72, 183). However, that contribution declines during exercise or other physical activity because increased blood flow through muscles facilitates convective heat transfer from deep body tissues to the body's superficial tissues and thereby reduces insulation (183, 253). Rennie et al. (200) reported that the insulative component of muscle is abolished at exercise intensities of ∼300 Watts or above. At this intensity, body conductance is maximal. The thermal resistivity of fat is equivalent to cork, and greater than that of other tissues per unit mass (247). It has been estimated (160) that each additional percent of body fat equates approximately with a 0.1°C rise in deep body temperature. Consequently, fat persons have smaller body temperature changes and shiver less during cold exposure than lean persons (247), with the fall in deep body temperature during cold water immersion being inversely related to subcutaneous fat thickness. There is no significant difference between the cold shock responses of fat and lean individuals as the cutaneous cold receptors which initiate this response are situated above the subcutaneous fat; however, increased fat thickness is associated with a smaller metabolic and cardiovascular response to prolonged cold water immersion (33, 160). In fatter individuals the metabolic response to cold is primarily stimulated by receptors in the skin, whereas in leaner individuals there is usually also an input from deep body receptors as they cool (33).

Sex-associated differences in thermoregulatory responses and ability to maintain normal thermal balance during cold exposure appear almost entirely attributable to anthropometric characteristics (247). For example, in men and women having equivalent total body masses, surface areas are similar, but the women's greater fat content enhances insulation. However, in women and men of equivalent subcutaneous fat thickness, the women have a greater surface area but smaller total body mass (and lower total body heat content) than men. In addition, data suggest women have a lower thermoregulatory thermosensitivity, that is, women exhibit a smaller increase in metabolism, compared to men, in response to equivalent reductions in deep body temperature of more than 1°C (160). Thus, when insulation is the same, total heat loss during resting cold exposure would be greater in women because they have a larger surface area for convective heat flux, and body temperature would tend to fall more rapidly for any given thermal gradient unless shivering thermogenesis compensated with a more pronounced increment than in men. This compensation may be possible when heat flux is low (mild cold conditions), but a women's smaller lean body mass and metabolic response limits their maximal capacity for a thermogenic response; therefore, a more rapid deep body temperature decline might occur under severely cold conditions than in men of comparable body mass (160, 161). Thus, although women have greater amounts of subcutaneous fat than men, as a consequence of their greater surface area to mass ratio and lower shivering response, it has been calculated that lean women require twice the amount of fat of lean men to show similar changes in deep body temperatures in cold water (160). However, during exercise in cold-water, men and women who have equivalent body fat percentages exhibit similar thermoregulatory responses, due to the women having a more favorable fat distribution over the exercising limbs, compared to the men (162). These morphological differences may be of limited benefit at rest, but an advantage when exercise increases peripheral blood flow and compensate for the higher ratio of surface area to mass in women. The initial respiratory/cold shock response of females to cold water immersion may be smaller than that of males (156), although this has not been confirmed (121, 123).

Peripheral responses to cold also appear to differ between men and women. During exposure of the hand to cold while the rest of the body remains warm, finger temperatures are typically lower in women than men (119, 190, 197). Contact cooling studies suggest that women's fingers cool faster than men, possibly due to hand size (136). Prevalence of peripheral vascular disorders like Raynaud's Phenomenon, a transient, vasospastic disorder that causes blood vessels to constrict to a greater extent than normal when exposed to the cold leading to very low blood flow to the digits (261), is also higher (up to 9×) in women; this may make them more susceptible to peripheral cold injury (113). Individuals with scleroderma, lupus or arthritis are more likely to suffer from Raynaud's, as are individuals who live in cold-weather regions (21).

In general, people who are older than 60 years may be less cold tolerant than younger persons, due to reduced vasoconstriction and heat conservation in comparison to their younger counterparts (29, 81, 130, 144, 214, 256, 276). Older people can also experience a decline in physical fitness. If they are exercising at the same absolute metabolic rates as younger individuals, the older person will be working at a higher $\%VO_{2max}$, will fatigue sooner, and must decrease their absolute heat production if they fatigue, increasing the likelihood of a reduction in deep body temperature. Older individuals also appear to have blunted peripheral sensitivity/feelings. For example, when participants have control of setting a thermostat as the ambient temperature fluctuates, older individuals allow the air temperature to fall to lower levels before readjusting the thermostat (173,220). Furthermore, aging also reduces centrally mediated thermosensitivity. Frank et al. (89) observed that older participants had a lower deep body temperature threshold for initiating cutaneous vasoconstriction and metabolic heat production. The reduced vasoconstrictor response was primarily due to a delayed and blunted NE response to deep body cooling, suggestive of an impairment in alpha-adrenoceptor activity.

Black men and women are two to four times more likely to suffer a cold weather injury than their Caucasian counterparts (64). Early information on frostbite risk due to race was conducted in military populations and observed higher frostbite rates in Blacks (32, 219). A recent epidemiological study (64) controlling for occupational exposure to cold also observed a higher risk for Blacks across many different job descriptions. Physiological and anthropometric reasons suggested for the higher frostbite risk in African-Americans include less-pronounced cold-induced vasodilatation (CIVD), increased sympathetic response to cold exposure, and thinner, longer digits (32).

Overall, exercise training and aerobic fitness appear to have only minor influences on thermoregulatory responses to cold. Most cross-sectional comparisons of aerobically fit and less fit persons find no relationship between maximal aerobic power and temperature regulation in cold, and in studies purportedly demonstrating a relationship, differences in thermoregulation appear more likely attributable to anthropometric differences between the aerobically fit and less fit participants than to an effect of maximal aerobic power *per se* (20). Longitudinal studies have shown interval training to have no measurable effects on thermoregulatory responses to cold (207), and while endurance training was shown to improve the cutaneous vasoconstrictor response during cold water immersion, that effect had little impact on deep body temperature changes during cold exposure (277). There is some evidence (102) to suggest that the maximum shivering intensity is related to maximum oxygen consumption (VO_{2max}). Eyolfson et al. (80) followed this up and demonstrated that the prediction of peak shivering using step-wise regression includes VO_{2max} in the algorithm. Furthermore, on exposure to cold air, rather than water, individuals may show a direct relationship between fitness, metabolic heat production, and skin temperature (2, 20). It has been postulated that fitter individuals have a lower % body fat and thus lose more heat initially, triggering the shivering response (20). Even so, partial correlation coefficient analysis demonstrated that fitness is more important; at the same % body fat, individuals with a higher VO_{2max} had a heightened metabolic response (20). In addition, individuals with high levels of aerobic fitness do appear to have an attenuated cold shock or cardiorespiratory response to cold water immersion (105) and a lower number of arrhythmias/dysrhythmias during helicopter underwater escape (238).

Exercise performance

Physical performance is a function of energy output, neuromuscular function, and psychological factors (13). Exercising in cold conditions impacts each of these to potentially reduce performance through changes in cardiovascular function and delivery of fuel, changes in nerve conduction and skeletal muscle contraction, and perception of effort. Performance can, and has, been defined many ways. Time to exhaustion (TTE), time-trial performance (completing either a fixed amount of work as quickly as possible or completing as much work as possible over a specified time period), VO_{2max}, muscular strength, and muscular endurance have all been used to quantify physical performance. This section first separates aerobic performance into those quantified both in cold air and cold water and then discusses the physiological mechanisms for the observed changes. Following this, the impact of cold on anaerobic performance, primarily muscular strength, is reviewed.

Aerobic exercise performance during cold air and water exposure

Aerobic exercise performance in cold air and water has not been comprehensively documented. Also, one of the problems in this research area has been the lack of standardized methods (e.g., cold exposure temperatures and performance assessment). For experiments conducted in air, the majority of studies have used a TTE test and compared cold air exposure to either relatively warmer ambient temperatures or those defined typically as temperate environments (~20°C). Table 1

Table 1 Summary of Human Studies Examining the Effect of Cold Exposure on Aerobic Exercise Performance

Reference	Sample size	Exercise mode	Intensity	Environments	Findings/results
Patton and Vogel (186)	Eight males	CE 60 RPM	75%-80% VO_{2max} 30 h at each temperature before exercise	20 and −20°C RH 20%-40% AV 0.5 m s^{-1}	TTE decreased 38% from 20 to −20°C
Galloway and Maughan (94)	Eight males	CE 60-70 RPM	70% VO_{2max} to exhaustion	4, 11, 21, and 31°C air RH 70% AV 0.7 m s^{-1}	VO_2 was higher at 4 and 11°C vs. 21°C TTE highest at 11°C (93.5 ± 6.2 min) with 15% reduction at 4°C
Sandsund et al. (205)	Eight males	TM 6% grade	55% VO_{2max} for 10 min 60%-95% VO_{2max} 4 × 5 min 95% VO_{2max} for 10 min (Salbutamol) 85% VO_{2max} for 5 min (Salbutamol)	−15°C air with/without Salbutamol 23°C/RH 52% AV 1.5 m s^{-1}	TTE shorter at −15°C compared to 23°C VO_2 was higher at −15°C Salbutamol increased FEV_1 after exercise
Parkin et al. (184)	Eight males	CE 80RPM	70% VO_{2max} until exhaustion	3, 20, and 40°C RH <50%	TTE highest at 3°C
Carling et al. (34)	Nine males	Professional Soccer Match	Running speeds 0.0-14.3 km/h 14.4-19.7 km/h ≥19.8 km/h	≤5°C, 6-10°C, 11-20°C, and ≥21°C	Shortest distances ran in ≥21°C (118.7 ± 6.9 m) Greater distances covered per minute in ≤5°C (9.1 ± 3.8 m)
Sandsund et al. (204)	Nine males	TM 6% grade	60% VO_{2max} for 10 min then 67%-91% VO_{2max} 4 × 5 min Followed by VO_{2max} and TTE	−14 and −9°C −4 and 1°C 10 and 20°C AV 5 m s^{-1}	TTE highest at −4 and 1°C No significant differences in VO_{2max}
Renberg et al. (198)	Nine women		71% VO_{2max} for 10 min then 76%-89% VO_{2max} 4 × 5 min Followed by VO_{2max} and TTE	−14 and −9°C −4 and 1°C 10 and 20°C AV 5 m s^{-1}	No difference in TTE among different air temperatures

Abbreviations: CE, cycle ergometer, TM, treadmill; TTE, time to exhaustion; RH, relative humidity; VO_{2max}, maximum oxygen uptake; MST, mean skin temperature; FEV_1, forced expiratory volume in 1 s; AV, air velocity.

lists the studies that have examined aerobic performance during cold air exposure. Overall, there is no consensus about whether exercising in cold air, compared to relatively warmer ambient temperatures, impacts aerobic performance. Some studies would suggest that cold ambient temperatures impair TTE (94, 186, 204, 205), whereas others suggest an improvement (34, 184) or no change (43, 198). In the two laboratory studies that systematically used multiple air temperatures, an inverted U pattern emerged. TTE was maximal at some intermediate ambient temperatures (11°C in shorts/t-shirt; −4 and 1°C with cross-country ski clothing) and TTE decreased as ambient temperature either decreased or increased from this optimal temperature. However, this work does contrast with the field study by Carling et al. (34) who found that running distances in soccer players were the same whether the ambient temperatures were in the ranges of <5°C, 6 to 10°C, or 11 to 20°C. Furthermore, the data from Parkin et al. (184) directly contrast that observed by Galloway and Maughan (94); they found that TTE while cycling at 70%VO_{2max} was 42% longer in the cold versus temperate environment, whereas Galloway and Maughan found no difference between these two air temperatures (4 and 21°C). Cheuvront et al. (43) also did not find any differences between 2 and 20°C air during a 30-min time trial. Interestingly, the studies above used men as the volunteers. Recently, Sandsund and colleagues repeated their study in multiple air environments (−14 to 20°C), but in women (198). They found no decrement in TTE as ambient temperature fell, in direct contrast to their study in men. They attributed this sex difference to higher heat losses in men, as they exhibited warmer skin temperatures and lower rectal temperatures. Clearly more systematic data are needed to clarify whether cold air impacts long-duration aerobic performance. What is interesting from the different studies is that optimal aerobic performance is likely not to occur during tests conducted in typical laboratory conditions (20-23°C), but in much cooler environments.

Using VO_{2max} as a surrogate for aerobic performance, cold air was found to reduce performance by 5% in −20°C

(175, 195) and 0°C (195) compared to 20°C. In contrast, no differences were observed between 5 and 18°C air (159).

Cold water effects potentially would impact performance markedly more than cold air due to the much larger heat conduction in water (~25 times greater than air) resulting in greater deep body and tissue temperature changes (247). Cold water exposure is more likely to lead to declines in deep body and muscle temperatures, cause greater thermoregulatory effector responses (shivering and vasoconstriction) and degrade nerve conduction to a greater extent than air.

Only a few studies have examined aerobic performance *per se* during cold water exposure. Holmer and Bergh (126) measured maximal aerobic power (VO_{2max}) during swimming at water temperatures of 18, 26, and 34°C. In three of the five participants (the leanest) VO_{2max} was 6% to 18% lower during maximal swimming in 18°C water. As well, the VO_{2max} during cold-water swimming was compared to running and was found to be 13% lower than the VO_{2max} attained during running. Rennie et al. (200) reported a reduced VO_{2max} during cycle exercise in 20°C water compared to air. Although other water temperatures were studied (25-35°C), data at these other temperatures were not included in the report. Along with a reduced VO_{2max}, they also reported a decline in mechanical efficiency in 20°C water and suggest this is a possible mechanism for the lower VO_{2max} with cooling. However, comparisons between water and air are not meaningful; data should be compared between the 20 and 35°C water temperature trials.

There are multiple putative mechanisms proposed for this reduced aerobic performance in cold environments (Fig. 4). Each of these will be discussed.

Limitations to aerobic exercise performance and maximal oxygen uptake in the cold

Temperature
- a. Lower deep body temperature
- b. Decreased muscle temperature
- c. Reduced skin temperature

Metabolism
- a. Increased lactate
- b. Low glucose levels
- c. Fasting
- d. Increased VO_2/reduced economy

Central/peripheral circulation
- a. Reduced maximal HR
- b. Lower cardiac output
- c. Reduced muscle blood flow

Figure 4 Summary of possible mechanisms that impact maximal oxygen uptake and aerobic performance in a cold environment.

Deep body, muscle, and skin temperatures

It is predicted, through the Q_{10} effect, that lower internal temperatures will reduce metabolism and muscle force generation (63) and thus temperature should be a critical component for physical performance. Bergh and colleagues completed a series of studies in the 1970s examining the effect of lowered deep body temperatures on VO_{2max} (13-15). They used cold water immersion to reduce temperature and then had their participants complete combined leg/arm exercise in the air until reaching exhaustion or VO_{2max}. They also manipulated skin temperatures by changing the air temperature following cold exposure. Figure 5 shows the relationship between deep body temperature and maximal work time/peak oxygen uptake. For

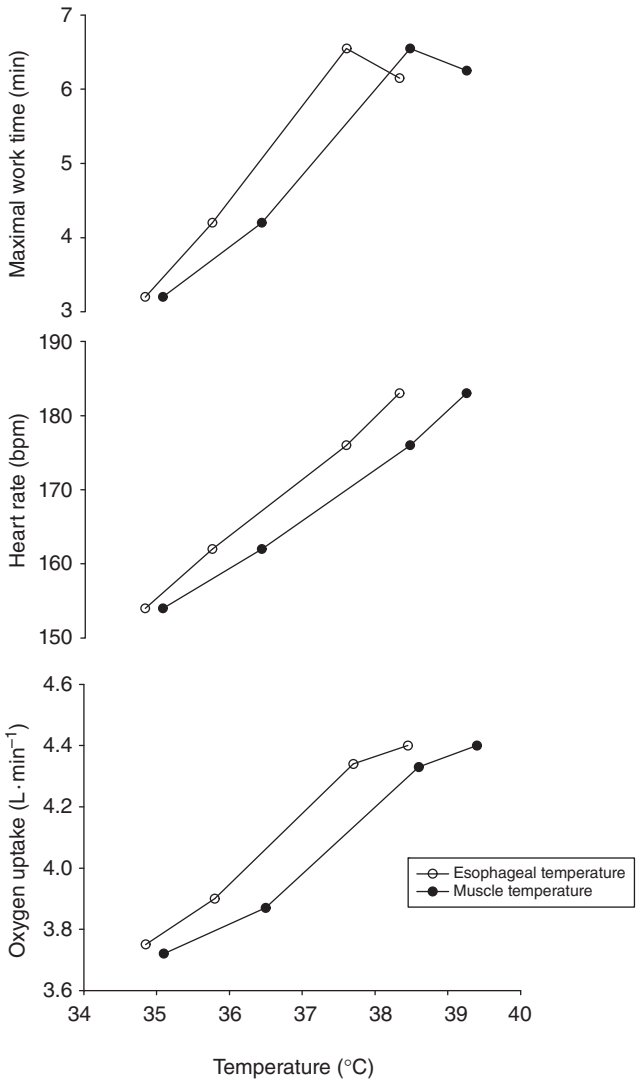

Figure 5 Changes in maximal work time, heart rate, and maximal oxygen uptake during combined arm and bicycling exercise as a function of core temperature (esophageal temperature) and muscle (vastus lateralis) temperature. Subjects either swam in cold water (13-15°C) or pedaled on a cycle ergometer before arm/leg exercise to induce changes in core and muscle temperatures. Adapted, with permission, from Ref. (15).

every °C fall in esophageal temperature (T_{es}), there was a 5% to 6% reduction in maximal aerobic power, which was not affected by skin temperatures of either 27 or 31°C. At T_{es} of 38.4°C, maximal oxygen uptake was 4.4 L·min^{-1}, but at a T_{es} of 34.9°C, VO_{2max} was reduced to 3.75 L·min^{-1}. Similar results have also been reported by Holmer and Bergh (126). Work time to achieve these VO_{2max} values was halved, from 6.2 to 3.1 min. Changes in muscle temperature caused the same response pattern as esophageal temperature, that is, there was a 5% to 6% reduction in VO_{2max} per °C fall in muscle temperature. Along with these temperature-induced reductions in peak aerobic power was a 15% reduction in maximal heart rate (from 183 to 155 beats·min^{-1}). The change in peak aerobic power and heart rate appear to be mediated by significant body heat loss and cooling of deep body tissues and skeletal muscle. However, the idea that thermal balance is the only key to performance is challenged by the performance studies conducted in cold air.

In the recent studies outlined earlier that were completed by Galloway and Maughan (94) and by Sandsund et al. (204), performance decreased at colder air temperatures, but with little effect on thermal balance and rectal temperature. Galloway and Maughan (94) observed a reduction in cycling performance when going from an optimal air temperature of 11°C down to 4°C, and Sandstund et al. (204) found that the optimal temperature for running while wearing 1.3 clo was −4 to 1°C with performance reduced at −14°C air temperatures. In the study of Galloway and Maughan, rectal temperature was 39.2°C during cycling exercise at the ambient temperature of 11°C, compared to 38.8°C at 4°C whereas the mean skin temperature went from 25 to 22.5°C. In Sandsund et al. (204), the rectal temperature was 38.4 to 38.5°C during running at the ambient temperatures of −4 to 1°C, compared to 38.2°C at −14°C whereas the mean skin temperature went from 25 to 27°C at the higher ambient temperatures to 21°C at −14°C. These studies suggest a mean skin temperature threshold of <25°C impacts TTE performance when deep body temperature is elevated. The studies from Bergh et al. (13, 15, 17) suggest skin temperature appears to have no effect when esophageal temperature is severely reduced (near 35°C), although the lowest T_{sk} in those studies was only 27°C.

Cardiovascular limitations

Cold has a profound effect on cardiovascular function during exercise, some of which may impact performance. Significant changes occur in heart rate, stroke volume, sympathetic nervous activity, total peripheral resistance, mean arterial pressure, cardiac work, and myocardial oxygen requirements.

Maximal heart rate is lower by 10 to 30 beats·min^{-1} when the deep body temperature is lowered by 0.5 to 2.0°C (187). This reduction in maximal heart rate is primarily responsible for the reduced VO_{2max} with body cooling (15,61,83,126,170). Figure 5 shows the relationship between esophageal temperature and maximal HR (15). As the deep body temperature declines, HR declines in a linear manner ($r = 0.97$). The reduction in maximal HR accounted for 61% of the variance in the decline in VO_{2max}. Similar to maximal exercise, submaximal exercise heart rate is also lower at any given power output or VO_2 during cold exposure, compared to a temperate environment, when the body temperature is reduced (61, 126). Similarly, McArdle et al. (161) found a 15% reduction in submaximal HR.

Multiple postulates have been put forward for the reduction in maximal HR during exercise-cold stress, including a reduced sympathetic drive and a change in heart mechanics. The reduced sympathetic drive is implied because administering atropine, a parasympathetic inhibitor, has no effect on HR during exercise when hypothermic (deep body temperature = 35°C), suggesting it is a reduction in β-adrenergic stimulation (61). Or perhaps it could be simply due to the fact that, when muscles are cold, one does not reach the same power output during graded exercise, and that is why HR is lower at maximal effort. However, data from Bergh et al. (15) suggest that this is not the case. In one study, participants swam in cold water until their deep body and muscle temperatures decreased and then cycled at 110% of peak power until exhaustion. During the first 2 min, VO_2 and HR were nearly the same, compared to the noncold trial. After these first few minutes, however, HR was lower in the cold trial and began to plateau while in the normal muscle temperature trial, HR continued to rise, leading to a higher maximal VO_2.

In contrast to HR, stroke volume increases during cold exposure as a result of translocation of blood from the peripheral/limb circulation to the central circulation, increasing central venous pressure (161, 196). Pendergast (187) stated that the increase in SV as HR declines demonstrates that the heart is functioning on the flat part of the cardiac pressure-volume curve. Wilson et al. (263) tested this hypothesis. They systematically changed the pulmonary capillary wedge pressure (PCWP) using lower body negative pressure and generated Frank-Starling curves. They found that with skin-surface cooling, the operating point of the PCWP-SV curve was shifted to the right onto the flatter part of the hyperbolic curve. Even when dehydrated up to 4% body mass, cold exposure (8°C air) maintains stroke volume during 30-min of exercise at 72% VO_{2max} (108), leading to no change in cardiac output (Q); this is likely related to the relatively small changes in exercise performance when dehydrated in cool to cold, versus warm, environments (43,142). Cardiac output during exercise in the cold is dependent on exercise intensity. For example, there is a linear relationship between VO_2/workload and Q, with values reaching 20 L·min^{-1} at an external workload of 120 W (161, 187). On a relative basis, Kenefick et al. (143) found that Q was 16 L·min^{-1} at 50% VO_{2max} (4°C air), with Gonzalez-Alonso et al. (108) reporting values of 20 L·min^{-1} at 72% VO_{2max} (8°C air). Maximal Q is reduced during cycle ergometry (200) in 20°C (16.4 L·min^{-1}) versus temperate air (21.8 L·min^{-1}).

Exercise-cold stress, compared to exercise in warm environments, increases sympathetic nervous activity, total peripheral resistance, mean arterial pressure, cardiac work,

and myocardial oxygen requirements during rest or exercise (65,78,129). For example, mean arterial pressure increases by ~17 mmHg (18%) and rate pressure product (systolic pressure × heart rate) increases by 10% (28). Facial cooling by wind, alone, lowers the heart rate by ~10 beats.min^{-1} during low-intensity exercise (<35%VO_{2max}) but also causes mean arterial blood pressure and rate pressure product to rise, secondary to an increase in peripheral vasoconstriction and systemic vascular resistance (146). However, these central and peripheral changes in cardiovascular function probably have no impact on exercise performance.

The one cardiovascular change resulting from cold likely to cause a decrease in exercise performance is a reduction in muscle blood flow. Several studies suggest that resting cold exposure lowers muscle blood flow. Gregson et al. (111) observed a 30% to 40% reduction in femoral artery blood flow and conductance after immersion in 8 and 22°C water. Wray et al. (268) found that cold-water immersion of the foot also reduced femoral artery blood flow. Brachial artery blood flow is also reduced during skin-surface cooling at rest (264). It should be noted that the above studies used duplex ultrasound and Doppler to measure arterial blood flow, which does not reflect/isolate only muscle blood flow, but also includes skin blood flow.

During exercise-cold exposure, two studies provide evidence for a reduction in blood flow to working muscle. Both Ferretti et al. (84) and Ishii et al. (132) had their participants sit in 19 to 20°C water (abdominal level) for 1 to 2 h, reducing leg muscle temperatures by ~7.5°C. Following this cooling period, vastus lateralis blood flow was assessed during leg exercise via ^{133}Xe clearance. Thigh blood flow was reduced with muscle cooling, compared to no cooling, by 36% during leg exercise at 75 W, 26% at 125 W, and by 12% at 260 W (which was the maximal exercise intensity in the cold condition). Rennie et al. (200) systematically examined the effect of water temperature and exercise intensity on thigh muscle blood flow using Xe clearance. Figure 6 shows the decline in leg muscle blood flow as a function of lower water temperature at all three levels of metabolic activity. The cold-induced muscle vasoconstriction thus appears to supersede the ability to perfuse active muscle, which could limit exercise performance. In addition to a decrease in muscle blood flow, cold will likely cause a decrease in muscle unloading of oxygen from hemoglobin, due to a leftward shift in the oxygen-hemoglobin dissociation curve (279).

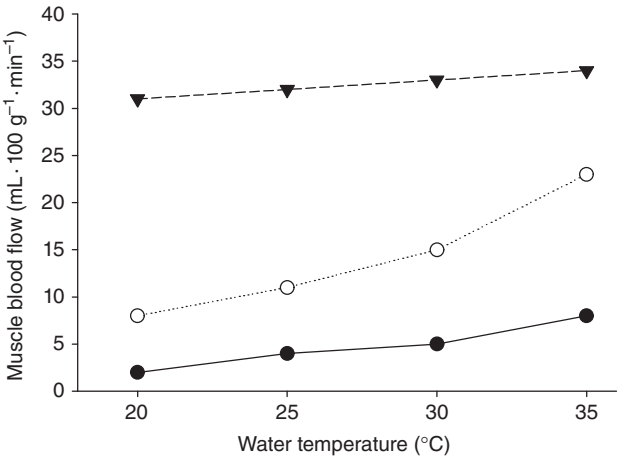

Figure 6 Muscle blood flow as a function of water temperature during three levels of activity (rest, oxygen uptakes of 1 and 3 L·min^{-1}). Exercise was performed on a bike ergometer. Redrawn, with permission, from Ref. (200).

Metabolism

Exercise performance may be impacted during cold exposure through a shift from aerobic to anaerobic metabolism. Blood and muscle lactate levels increase during exercise to a greater extent in cold compared to temperate environments (22, 23, 83, 126, 132, 162, 170, 222, 258) suggesting a greater reliance on anaerobic metabolism. Blomstrand et al. (22, 23) found during very high-intensity exercise (350-370 W) with subnormal muscle temperatures (28-29°C), a 40% decrease in TTE concomitant with higher glucose-6-phosphate and muscle lactate levels in both Type 1 and Type 2 fibers. It was not clear whether this increase in muscle lactate was due to increased glycolysis or less efflux of lactate from the muscle. In another study (133) at a high relative exercise intensity (70% VO_{2max}), lactate was significantly higher when running in a cold rain in 5°C versus no rain, also suggesting a switch from aerobic to anaerobic metabolism. This switch from aerobic to anaerobic glycolysis potentially would lead to earlier fatigue during exercise. In fish, there is evidence that cold exposure causes recruitment of Type 2 muscle fibers (202). There is no research in humans that muscles preferentially switch over to fast twitch fibers as they become colder, but this could provide another mechanism for a decrease in exercise performance as these fibers fatigue sooner than Type 1 fibers. An important point to make is that fuel utilization between exercise and shivering are not analogous. Tipton et al. (244) observed that at comparable levels of metabolic heat production over a 2-h period, substrate utilization was not similar between exercise and shivering, as hepatic glucose output and utilization were higher during exercise and fat and insulin levels were higher with shivering.

Exercise economy is defined as the oxygen uptake at a given running speed and exercise efficiency is the ratio of work to energy expenditure on a cycle ergometer. These variables have been considered one of the major determinants of endurance performance success (10, 138). A reduction in exercise economy/efficiency may be partly responsible for a decline in aerobic performance in cold environments. Multiple studies have shown that running at the same speed or cycling at the same external workload elicits a higher VO_2 in the cold, compared to temperate or hot environments (44, 47, 94, 117, 175, 176, 186, 205), although this is not universal (135, 198, 204). In the studies cited earlier that showed differences, this reduction in economy/efficiency occurred even though deep body temperature, well known to increase

VO_2, was not lower among the different ambient temperatures studied. Some of the reasons postulated for the reduced efficiency besides shivering, which is unlikely in conditions where deep body temperature is not affected, include nonshivering thermogenesis and an increase in the number of motor units recruited, since cooled muscles develop less force (133). In studies where local leg temperatures were reduced with cold water immersion, no effect was observed on running economy (87) or mechanical efficiency during cycle exercise (265). However, cold muscle temperatures did cause an increase in stride frequency and decrease in stride length which were attributed to a change in muscle tendon elasticity. Interestingly, cold acclimatization has been shown to reduce the VO_2 at any given power output during cycle ergometry (167), although the use of independent groups makes a definitive conclusion tentative.

Fuel and fluid intake

It is well established that fuel and fluid intake are important for exercise performed for greater than 60 min. Many reviews discuss performance and fuel intake as well as fluid intake and are treated elsewhere. The majority of these studies were conducted in temperate or hot environments; the impact of replacing fuel/fluid during exercise in the cold is less well documented.

Replacing food and fluid is still important in cold environments, as reserves can be depleted. Jacobs et al. (135) found that during light-intensity exercise (30% VO_{2max}) glycogen depletion was greater in 9°C versus 21°C air, although this was not observed at 60% VO_{2max}. Weller et al. (259) found that in cold versus more temperate conditions, there were increased carbohydrate oxidation rates during low-intensity exercise (~30% VO_{2max}), but not during high-intensity exercise (~60% VO_{2max}). Parkin et al. (184) have observed lower glycogenolytic rates during exercise in 3°C air, versus 20°C, during a ride to exhaustion at 70% VO_{2max}. In fasted conditions, the importance of fuel replacement during low-intensity exercise is demonstrated by the fact that plasma glucose levels fall to 3.5 $mmol \cdot L^{-1}$, levels traditionally commensurate with a reduction in exercise capacity (51, 65). Weller et al. (260) tested volunteers during low-intensity walking (30% VO_{2max}) in a cold, wet environment following a 36-h fast. Exercise time was 16% less when fasted (349 min nonfasted vs. 294 min fasted) and lower rectal temperatures were observed in the fasted condition.

Since exercise in the cold can lower muscle glycogen, consuming exogenous carbohydrates during exercise in the cold may be beneficial for performance. Table 2 presents the experiments that have been conducted. The conclusion from these studies is that carbohydrate supplementation prolongs

Table 2 The Effect of Carbohydrate Feeding on Exercise Performance During Cold Exposure

Reference	Method	Environment	Procedure	Significant findings
Febbraio et al. (82)	Five males; one female cycle to exhaustion at 70% VO_{2peak} trials >5 days apart random order	5°C w/50% RH	Consume prior to and at 15 min intervals 250 mL of one of three drinks a. 0% CHO b. 7% CHO c. 14% CHO cycle until unable to maintain 70 $rev \cdot min^{-1}$ for 20 s	Mean TTE (min) a. ~130 b. ~200 c. ~175 7% solution was most effective in aiding performance
Galloway and Maughan (95)	Six males cycle to exhaustion at 80% VO_{2max} 1-2 weeks apart crossover random order	10°C, 72% RH	Three trials—different CHO composition a. no drink (ND) b. 2% CHO c. 15% CHO cycle until unable to maintain >60 rpm	Median TTE (min) a. 90.6 b. 102.0 c. 97.7 4.4% improvement in TTE with 2% CHO drink
Pitsiladis and Maughan (189)	Six subjects cycle to exhaustion at 70% VO_{2max} 8 day trial random order	10°C w/ 70% RH	Day 1 deplete muscle glycogen stores, afterward begin consuming 10% CHO diet until day 4 performance trial Day 5 deplete muscle glycogen stores, afterward begin consuming 80% CHO diet until day 8 performance trial	Performance trial time (min) a. 10% CHO diet, 89.2 min b. 80% CHO diet, 158.2 min
Galloway et al. (96)	Six males cycle to exhaustion at 80% VO_{2max} 1-2 weeks apart crossover random order	10°C w/ 70% RH	Four trials—consume different CHO beverages a. 0% b. 2% c. 6% d. 12% cycle until unable to maintain >60 rpm	Median exercise time (min) a. 83.5 b. 103.2 c. 100.4 d. 94.8 No statistically significant effect of CHO drink on exercise capacity, but exercise time was increased by 24% and 20% with 2% and 6% CHO, respectively

exercise time during cold exposure, as has been observed in other environmental extremes (201, 251).

Dehydration can be a serious problem in the cold due to high sweat losses, conscious underdrinking to avoid urination, cold-induced diuresis, high respiratory fluid losses, loss of thirst, and poor water availability (90). However, few studies have examined hydration effects on performance in the cold. Cheuvront et al. (43) examined the impact of 3% hypohydration during a 30-min time trial in 2 and 20°C air. In 20°C, hypohydration caused an 8% decline in time-trial performance, whereas only a 3% decline in performance was observed during the time trial in 2°C. Furthermore, there was no difference in performance when hypohydrated in the cold compared to the cold-euhydrated condition. Kenefick et al. (142) found that a 15-min time trial performance was not different in 10°C air between euhydrated and hypohydrated (4% body mass loss) conditions. In both of these studies, cold skin temperatures likely maintained central blood volume and the resulting increase in venous return, maintained cardiac output (108).

One of the limiting factors in generalizing the impact of cold on aerobic performance is that in the few studies that have examined this, cold has been examined in isolation. However in reality, cold occurs in combination with other environmental stressors, for example, cold and altitude; cold nights and hot days in the desert. More attention should be given to understanding how multiple environmental stressors affect human physiology and performance (231). Indeed, there is some evidence that adaptation to one environment (cold) impacts the physiological responses to hypoxia (151). As research moves forward to understanding how cold exposure affects physical performance, future efforts should utilize research designs that not only enable the independent effects of cold exposure to be understood, but also the impact of other environmental and exercise stressors that exist in combination with cold in natural environments.

Strength/power/balance

Cold has a significant effect on strength, primarily on dynamic or isotonic strength. Table 3 presents the studies examining the impact of cold on muscular strength. Bergh and Ekblom (14) were able to quantify the relationship between the fall in deep body temperature and power. They found a 4.2% decrease per °C fall in esophageal temperature. Davies and Young (60) found that during voluntary jumping, lower heights as well as lower impulse and power were achieved. On a force bicycle, they observed a decline in peak (31%) and average (44%) power as muscle temperature was reduced by 8.8°C. They also observed a shift to the left in the force-velocity curve in cold muscles compared to control conditions (Fig. 7). Their findings show that cold muscles have a direct impact on the mechanical and contractile properties of muscle and thus impair maximal power. Sargeant (1987) measured peak anaerobic power and force during 20-s maximal sprint efforts on a cycle ergometer at four different muscle temperatures. He found that peak force at muscle temperatures of 32 and 29°C was 12% and 21% lower, respectively, compared to a muscle temperature of 36.6°C. This reduction in peak force was lower throughout the 20-s period. Interestingly, cold exposure does not have to be severe to impact performance. Oksa et al. (177) subjected their volunteers to 60 min of exposure to 27, 20, 15, and 10°C air and then measured their ability to perform a rebound jump (plyometric or stretch-shortening movement where the subject jumps off a box, lands, and then jumps again—the measured value is the time in the air between the two landing phases). For a fairly low level of cooling, there was a significant impact on performance. A 15°C air exposure caused calf muscle temperature to decrease by 2°C and a 10°C exposure reduced calf temperature by 3.4°C, resulting in a 24% decline in flight time. Other effects of cold exposure include an increase in EMG amplitude (67, 68), that is, more muscle fibers must be recruited to perform a given work output, the power frequency distribution shifts to the left, similar to that observed with fatigue (67), a slowing of twitch and tetanic contractions (68), an increased level of coactivation of agonist and antagonistic muscle pairs and an increase in the number of motor units recruited (174). Twitch force is reduced with a 5°C fall in muscle temperature (18). The nerve firing rate is the same with cold muscle but contractile slowing still occurs. Also there is an increase in the propagation time of muscle action potentials with a loss of synchrony among motor units (18). The stretch reflex is also impaired with cooling, likely causing a decline in muscle spindle activity (12, 75, 172, 178, 210). As well, nerve cooling would alter neuromuscular function. If nerve temperatures decline from 36°C down to 20°C, nerve conduction velocity falls from 49.6 to 7.2 $m.s^{-1}$ or at a linear decrease of 1-5 to 1.8 $m.s^{-1}$ per °C (62, 252). No nerve conduction occurs below temperatures of 10°C (41, 120). It is likely that nerve conduction during exercise in air is not contributing to a decrease in neuromuscular performance, but more so during exercise in very cold water. Other reasons have also been postulated for the decline in the rate in force development as muscle temperatures are lowered (Q_{10} values of 1.6-3). These include a decrease in metabolic rate, ATP hydrolysis rate, calcium release from the sarcoplasmic reticulum, and calcium sensitivity (67).

Several studies have tried to separate the influence of central (lower deep body temperature/lower mean skin temperature) versus peripheral (lower local temperature) effects on muscle function. Oksa et al. (174) reported that systemic cooling (colder overall skin temperatures) caused a bigger effect than just local cooling of the forearm. They found higher EMG amplitudes with systemic cooling even though muscle temps in the forearm were the same. As well, lower skin temperature caused a greater afferent input to the spinal cord and thus a greater/higher recruitment of motor units. Cahill and Giesbrecht (31) found that whole body hypothermia ($T_{deep\ body}$ = 34.8°C) reduced peak voluntary torque by 15%, decreased the amplitude of the superimposed twitch and muscle twitch tension, slowed twitch dynamics, and lowered maximal resting twitch by 43%. These findings are

Table 3 The Effect of Cold Exposure on Measures of Strength and Power

References	Methods	Environmental conditions	Procedure	Results/discussion
Bergh and Ekblom (14)	Four males Two experiments per condition with varied order Testing ≥2 days apart *Measurements:* T_m (left leg vastus lateralis), T_{es}	T_{es} and T_m A. Low, 30-36°C, cold-water immersion (legs) B. High, 36-39°C, bicycle ergometer exercise	Left leg knee extension (max muscle strength and power output) Vertical jumping Sprinting (bicycle ergometer)	Peak torque ($180°\cdot s^{-1}$) decreased at rate of 4.9% for every degree fall in T_m Vertical jump decreased at rate of $4.2\%\cdot°C^{-1}$ change in T_m Sprinting performance decreased at rate of $4.4\%\cdot°C^{-1}$ change in T_m
Davies and Young (60)	Five males Exposed to both conditions Procedure repeated on five separate occasions *Measurements:* T_m (gastrocnemius), MVC, TPT, $^1/_2$ RT	Water immersion (legs) A. 0°C (cold) reduced T_m by 8.4°C B. 46°C (hot)	Electrical twitch and tetanic stimulation of the triceps surae Vertical jumping Cycling (bicycle ergometer)	T_m cooling increased TPT by 38% and $^1/_2$ RT by 93% T_m cooling reduces power output by 31% and MVC by 19%
Bigland-Ritchie et al. (18)	Three males and two females Exposure to both conditions Several experiments on each subject on different days *Measurements:* T_m (first dorsal interosseous-hand), T_{es}, EMG, CT, $^1/_2$ RT, MVC	T_m A. 30°C (control) B. 25°C (water immersion of the arm not the hand)	FDI contractions and supramaximal nerve shocks to the ulnar nerve at the wrist after: 1. fatiguing 60 s MVC at T_m of 30°C 2. lowered T_m −25°C	Muscle cooling caused: CT increased by 50% $^1/_2$ RT increased 65%
Giesbrecht et al. (99)	Six volunteers Order of conditions followed a balanced design four trials, 4-5 min to complete 1 trial *Measurements:* T_m (biceps), T_{es}, timed tasks	Water immersion A. Cold body-cold arm B. Cold body-warm arm C. Warm body-cold arm Cold water = 8°C Warm water = 29-38°C	Dominant hand motor tasks 1. Speed of finger (fine) 2. Finger dexterity (fine) 3. Handgrip strength (gross) 4. Nut and bolt test (fine) 5. Peg and ring (gross) 6. Speed of arm (gross) (1-6 = 1 trial) Trials First preimmersion Second to fourth immersion, 15, 45, and 70 min, respectively	T_m accounted for 85%-98% of the decrease in performance Decrease in performance due almost entirely due to local effects of cooling Decrement in performance was greater in fine motor tasks
Meigal et al. (163)	14 males Random exposure to both conditions *Measurements:* T_m (biceps brachii), T_{sk}, T_{re}, EMG, MVC	Air A. 10°C (cold) B. 27°C (control) Decreased T_m from 35 to 31°C	Elbow flexion with a 6-8 kg mass corresponding to 30% of MVC	MVC decreased 12.3 in 10°C vs. 27°C air
Oksa et al. (174)	Eight males exposed to all three conditions in random order ≥4 days between exposures *Measurements:* T_m (wrist joint flexor), T_{re}, T_{sk}, EMG, MVC, TPT, $^1/_2$ RT	Air A. 5°C (systemic cooling) B. 5°C (local cooling) C. 25°C (control)	Wrist flexion-extension at 10% MVC six 20-min work bouts in each condition (120 min)	Repetitive wrist flexion-extension exercise in the cold caused higher EMG activity and fatigue than repetitive work alone
Cahill et al. (31)	Six males and one female exposed to both conditions in randomized and counterbalanced order Neural stimulation (biceps brachii, brachial plexus, cortical) applied during MVCs *Measurements:* T_{es}, EMG, MVC, TPT, $^1/_2$ RT	A. 8°C water immersion (hypothermia) B. 22°C air (control)	Dominant arm elbow flexion A. *Pretest:* Three series of intermittent 3-s MVCs B. *Intervention:* hypothermia or control C. *Posttest:* 2 series of intermittent 3s MVCs D. Sustained MVC (120 s) E. Recovery MVCs (3 s)	Rate of fatigue was greater during control conditions during sustained MVC Hypothermia Decreased MVC (16%) and MRT (43%) with an increased TPT (41%) and $^1/_2$ RT (41%). Decreased fatigue rate during sustained contractions by reducing central fatigue Attenuated the decrease in voluntary activation seen with cortical stimulation

Abbreviations: T_m, muscle temperature; T_{re}, rectal temperature; TPT, time to peak tension; FI, fatigue index; CT, twitch contraction time; T_{es} core temperature; EMG, electromyography; $^1/_2$ RT, half relaxation time; FDI, first dorsal interosseous; MRT, maximal resting twitch amplitude; T_{sk}, skin temperature; MVC, maximal voluntary contraction; MVIC, maximal voluntary isometric contraction; MAHF, maximal active hip flexion.

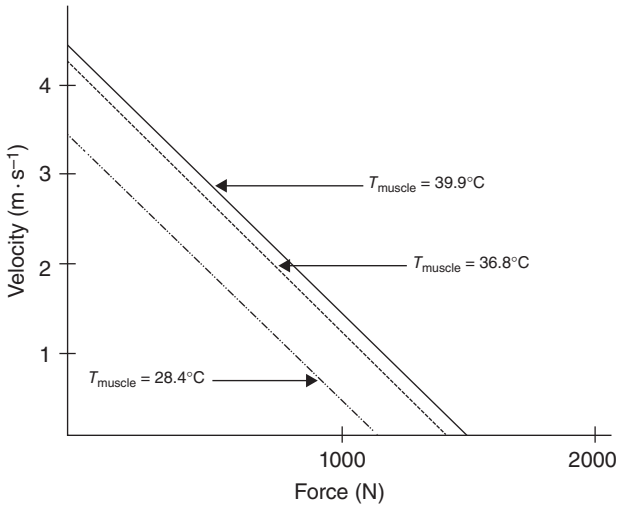

Figure 7 The effect of cold muscle on the force-velocity relationship during cycling exercise. Muscle temperature was changed passive exposure to 0 and 46°C water for 30 to 45 min. The leftward shift in the force-velocity curve in the cold muscle demonstrates that at any given force output, the velocity generated during cycle ergometry is less. Redrawn, with permission, from Ref. (60).

suggestive of a peripheral effect within the cold muscle (i.e., decreased myosin ATPase activity; decreased ATP utilization, slowed calcium release—delayed cross-bridge formation; decreased excitability of nerve membrane; recruit fewer fibers—decreased firing rate and increased antagonistic muscle coactivation). Because the arm was also immersed, it is difficult to tease out a central effect caused by hypothermia versus a peripheral/cold muscle only effect. Giesbrecht et al. (99) demonstrated that local muscle cooling accounts for most of the impact on hand and arm performance, while whole body hypothermia ($T_{deep\ body} = 35.6°C$), coupled with a warm arm/hand, had very little effect.

Cold exposure also degrades balance. Cooling protocols, using either 2 h of cold air (10°C), or 10 to 60 min of cold water (12-15°C) exposure to the hip, caused both static (154) and dynamic balance (166, 188) to decrease. Static balance requires trying to remain motionless while standing on an unstable surface, whereas dynamic balance involves movement in eight different planes with one leg, while maintaining balance with the stance leg. Others have also shown that foot cooling alone is sufficient to impact balance (153).

Manual dexterity

Cold, for the majority of people who are exposed, has little effect on deep body and muscle temperature cooling, as the exposure durations are relatively short in most cases. However, cold exposure for many people does impact hand and finger temperatures and subsequently, manual dexterity. Informal queries of soldiers and workers who are exposed to the cold list the loss of manual dexterity as the number one performance problem in the cold.

Multiple studies through the years have demonstrated that cold exposure reduces manual dexterity (42,48,49,77,98,120, 124, 221). Skin temperature is the primary correlate of manual dexterity. As the skin temperature of the fingers decreases below 15°C, manual dexterity begins to decline more rapidly (124). A series of studies examined the impact of body heat storage and changes in body heat content on manual dexterity (25, 27, 70, 86). This work found that the best predictor of finger temperature and maintaining dexterity throughout cold exposure was a change in body heat content. A change in body heat content of −300 kJ was associated with a finger temperature of <16°C and degraded dexterity (27). One caveat to this is that if the change in body heat content falls below this value, dexterity can still be maintained if the finger skin temperature can be kept above 30°C (25). Figure 8 depicts the skin and tissue temperatures associated with performance degradation. Cold is experienced at relatively mild temperatures; however, pain begins at temperatures about 5°C warmer than the decline in dexterity (76). When the skin temperatures go below 8°C, numbness and a loss of sensitivity are present (252). Other mechanisms responsible for a decline in dexterity include a reduction in efferent nerve conduction velocity (62), synovial fluid viscosity (131), and hand muscle temperatures (50, 120).

External factors not intrinsic to the hand and/or fingers that influence peripheral skin temperatures and dexterity have also been studied. Exercise has also been shown to improve dexterity. Muller et al. (168) reported that working at 50% VO_{2peak} for 30 min performed in the middle of a 5°C cold-air exposure increased dexterity by ~25%, although it did not approach the baseline levels that were measured in a temperate environment. In contrast, cold habituation, either through natural exposure or laboratory cold-water immersions, has not been shown to improve manual dexterity (114, 169).

Prediction of dexterity has received considerable interest in recent years and several studies have been published. Daanen (57) reported the combination of wind-chill temperatures and durations that cause finger skin temperatures to reach 14°C and thus reduce dexterity. Using regression analysis, he computed the equations for a decrease in finger dexterity, manual dexterity, and grip force. Interestingly, he normalized dexterity performance to the scores obtained in a 0°C, low wind environment. Another model was developed to predict hand manual performance (HMP) impairment during cold exposure (270). HMP is defined as the performance (tasks completed per unit time) normalized relative to performance at a finger/hand skin temperature (FST) of approximately 33°C. An empirical algorithm describing the relationship between HMP and FST was developed from published data and predicts the critical FST thresholds for reduced HMP.

Thermoregulation following exercise

Traditionally, hypothermia experienced by participants in winter sports and recreational activities was simply attributed

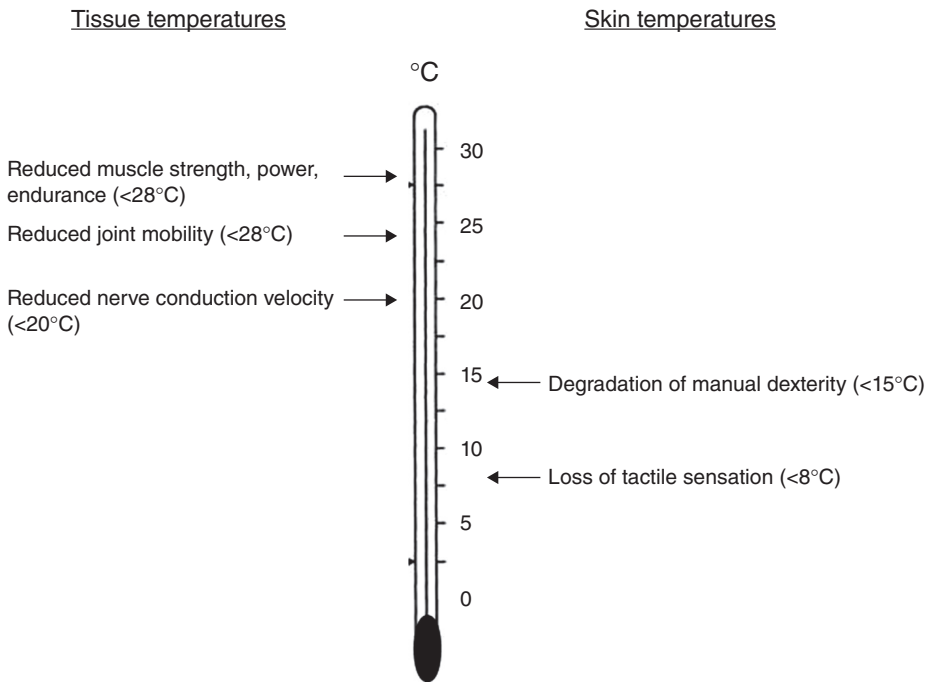

Figure 8 Tissue and skin temperatures associated with decrements in manual dexterity.

to effects of sustained cold exposure combined with inadequate clothing. However, degraded thermoregulatory effector responses (shivering and vasoconstriction) during/following physical exertion may potentially increase the risk of hypothermia. This degraded effector response was termed "thermoregulatory fatigue" (39). Thermoregulatory fatigue factors clearly identified include prior physical exercise and physical exhaustion, while evidence suggests that sleep loss and caloric deficit, with and without hypoglycemia, may be additional factors.

Pugh first suggested that physical exertion might increase the risk of hypothermia (193). He analyzed reports of 23 separate occasions that had led to numerous cases of hypothermia and 25 deaths. On one occasion, 240 individuals began walking at 0600 h in a drizzle and light wind, vying to be the fastest participants in the Four Inns Walking Competition (192), a combination race/hike traversing 72 km over the moors of Derbyshire, England (time to complete usually ranged from 9.5 to 22 h). During this particular contest, the weather became progressively worse with heavy rain and high winds (25 knots). Only 22 competitors completed the race that year (compared to 66%-85% typically observed) and three died from hypothermia. In his analysis of these incidences, Pugh identified physical exhaustion as a contributing factor for hypothermia, although he was unable to provide conclusive evidence. More recently exhaustion and hypothermia were again linked as causal factors for a large number of serious hypothermia casualties and four deaths during cold exposure following a grueling 60-day military training course in which soldiers are underfed, sleep deprived, and physically exhausted (274). These cases suggested the possibility that physical exhaustion was linked to hypothermia, but again the scientific evidence for a cause and effect association between physical exertion and susceptibility to hypothermia was lacking.

The first experimental observation suggesting that a thermoregulatory response, namely, shivering, could fatigue during exercise was reported by Thompson and Hayward (224). Those authors reported that during a 5-hour walk at a constant pace in a controlled experimental environment that simulated hiking in cold, rainy conditions, one participant who, having maintained stable metabolic rate and deep body temperature for the first 3 h of exposure, exhibited a progressive decline in metabolic rate and deep body temperature over the final 2 h, despite the fact that walking pace remained unchanged throughout (224). The authors concluded that the decline in metabolic rate exhibited by this individual reflected a decrease in shivering, since they reported that the participant maintained the same walking pace; they concluded that this decrease in shivering was evidence for a fatigue or exhaustion of shivering. However, since this effect was only observable in one of the five participants, and decreases in muscular activity besides shivering might account for a decline in overall metabolic rate without a change in walking pace, the author's conclusions remained speculative. More quantitative evidence for shivering fatigue has been reported by Bell et al. (11). They found that over a 2-h period during resting exposure in 10°C air, the central frequency of the EMG recording in the pectoralis major decreased with time, suggesting fatigue of this muscle group.

Young et al. (274) investigated how physiological responses to cold were affected by fatigue associated with

more prolonged physical exertion, controlling for possible confounders. Responses to cold were measured in eight men who had completed an arduous 9-week military training course, throughout which participants perform very strenuous physical activity and daily sleep was limited to about 4 h (274). The participants in this study completed a standardized experimental cold air exposure within 2 h of finishing this regimen (no rest), again following a short (48 h) recovery period for rest and refeeding, and again a third time following 16 weeks of recovery. The experiments demonstrated that cold tolerance (ability to maintain a deep body temperature above 35.5°C) was compromised during the trial performed without rest and remained compromised even after 48 h of recovery. Alterations in the normal shivering responses to cold may have contributed to the impaired maintenance of thermal balance. Shivering responses were delayed during the cold-exposure trial performed immediately after completing the exhaustive training course, compared to trials completed after rest and recovery (274).

Subsequently, a controlled, laboratory-based simulation to expose participants to the multistressor environment (albeit shorter) replicated Young et al.'s observations that exertional fatigue, negative energy balance and sleep loss, in combination, impair cold-induced shivering (36). Volunteers were exposed to 84 h of negative energy balance, sleep restriction, and high levels of physical activity. The principal finding in that study was that deep body temperature fell to a greater extent during cold-air exposure following the 84-h period. Figure 9 shows the mean body temperature-metabolic heat production response before and after the 84-h multistress period. As in the Young et al. study, there was a delayed onset of shivering (shift to the left in the metabolic heat production-mean body temperature relationship, suggesting a central attenuation in this thermoregulatory effector response)

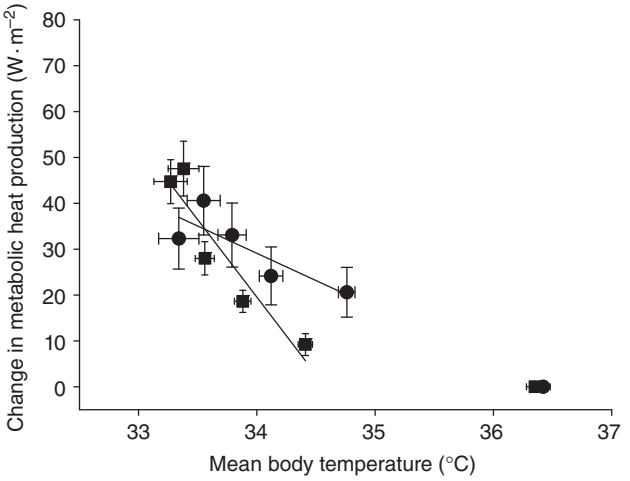

Figure 9 The change in metabolic heat production versus mean body temperature before (circles) and after (triangles) 84 h of sleep restriction and energy deficit during 180-min cold air exposure (10°C). A shift to the left in this relationship suggests a central attenuation of the shivering response due to the multiple stressors. From (36).

following the multistressor exposure, compared to rested conditions. However, the short-term multistressor experiment avoided the confounding effects of large changes in body fat and tissue insulation that were observed following 61 days of US Army Ranger training (274).

Three primary stressors were present during the 61-day and 84-h studies that may impact shivering thermogenesis. They are sleep deprivation, negative energy balance, and exertional fatigue caused by previous exercise. Sleep deprivation studies (85) (145) (206) have generally observed no effect on deep body temperature responses to cold exposure, but the methodologies (e.g., using exercise to keep people awake vs. sedentary exposure) and study protocols preclude definitive conclusions. Negative energy balance also impacts shivering thermogenesis. Macdonald and colleagues found a reduced gain (sensitivity) in the metabolic rate-deep body temperature relationship after 2 days of fasting in men (152). Similarly, a decline in the metabolic rate response to cold after fasting was observed in women following 48 h of food deprivation (157).

Exercise could increase the risk of hypothermia during subsequent cold exposure for several reasons. First, exercise might mediate "thermoregulatory fatigue" which would blunt shivering responses and reduce vasoconstriction during subsequent cold exposure. Second, cold exposure immediately after performing exercise might result in accentuated heat loss from an inability to immediately switch from heat dissipation responses caused by exercise in temperate conditions (209) to heat conserving responses needed during cold exposure. Third, exercise might mediate greater heat loss during subsequent cold exposure due to "heat redistribution" to active limbs that develops during exercise. During exercise, perfusion of active skeletal muscle increases and perfusion can remain elevated for extended durations (223) facilitating regional heat loss over these active limbs during exercise (208).

Castellani et al. (39) tested these three hypotheses by exposing 10 men to cold air (5°C) following cycle ergometer exercise that increased deep body temperature by 1°C and comparing responses to those observed when cold exposure was preceded by passively heating volunteers until they achieved the same preimmersion rectal temperature. The experimental data appear to confirm the suggestion that exertional fatigue is a primary factor that can impair vasoconstrictor responses to cold. Skin surface heat flow was greater, skin temperatures tended to be higher, and rectal temperatures fell more and faster following exercise compared to passive exposure. Tikuisis et al. (228) also demonstrated that 5 h of acute exercise increased peripheral heat loss during subsequent cold-wet exposure. In contrast, Scott et al. (211) observed that passive heat exposure results in faster deep body cooling rates versus exercise, most likely due to a greater skin to environmental temperature gradient than Castellani et al. (39) observed. Since the experimental conditions employed allowed sleep deprivation and energy substrate availability to be ruled out as significant influences, those findings were

interpreted as evidence for an impairment of cold-induced vasoconstrictor response induced by exertional fatigue and elevated sympathetic nervous system activity (39). Cold-induced vasoconstriction is sympathetically mediated, and the NE response to cold, considered reflective of sympathetic nervous activation (79), was the same whether cold exposure was preceded by exercise or passive heating. On the other hand, sensitivity of peripheral arterioles to NE released in response to cold might be diminished following exercise (134).

There was no effect of exercise on shivering thermogenesis which suggests that this response to cold is not easily fatigable (33). Scott et al. observed a similar response (211). Castellani et al. observed no difference in the mean body temperature versus change in metabolic heat production relationship between trials suggesting that the differences in deep body temperature were not due to a change in central control of shivering thermogenesis. Perhaps exercise intensity and duration were not sufficient to fatigue the shivering mechanism, which is a relatively low intensity activity (275), at least compared to exercise. In Pugh's case report of the Four Inns Walk (192), the participants were exercising up to 20 h in cold-wet conditions. Likewise, the volunteer in Thompson and Hayward's study (224) who developed shivering fatigue was exercising for 4 h in severe cold-wet conditions. Another possibility is that shivering impairments observed in these earlier studies may not reflect fatigue, but rather hypoglycemia, which is known to impair shivering (93,185). Plasma glucose levels were not measured in those previous studies (192,224). In the studies that demonstrated no effect of acute exercise on shivering (37, 39), plasma glucose concentrations remained normal throughout cold exposure.

Because exercise-induced hyperemia may have persisted during cold exposure following acute exercise and accounted for the greater heat loss rather than impaired vasoconstriction, Castellani et al. conducted a follow-on study (37). In this experiment volunteers walked for up to 6 h at 1.34 $m \cdot s^{-1}$ in 5°C air with a 5.4 $m \cdot s^{-1}$ wind while completely wet. This experimental design utilized exercise at a fixed intensity so any effects of exercise-induced hyperemia should have been similar for all trials (exercise fatigue or no fatigue). A group of ten men performed this "wet-walk" on days 0, after 3 days (D3) of fatiguing exercise, and following 7 days (D7) of exercise. Three control participants performed the "wet-walk" on these days also, but did not perform the exhaustive exercise regimen. Also, experimental controls were again in place to obviate sleep deprivation and energy substrate levels as influential factors.

A similar impairment of the vasoconstrictor response to cold, as observed in the acute exercise studies, was observed following 3 and 7 days of prolonged physical exertion for 4 h each day compared to cold exposures completed when physically rested (37). Shivering thermogenesis was not affected by chronic exercise. Since the exercise was at a fixed intensity in the cold, the effects of exercise-induced hyperemia ("heat redistribution") should have been similar for all trials and thus that mechanism could be ruled out. These observations indicate that fatigue induced by exhaustive exercise may indeed blunt the vasoconstrictor response during cold exposure. Furthermore, the development of cold habituation could also be ruled out as a potential confounder in this study because the design included a control group who completed the repeated cold exposure experiments without participating in the intervening exercise regimen, and demonstrated no between condition differences in thermoregulatory and body temperature responses. Thus, it seems reasonable to conclude that some other mechanism related to exertional fatigue acts to impair the vasoconstrictor response to cold.

The blunting of the vasoconstrictor response to cold subsequent to severe physical exertion may be related to concomitant elevations in basal circulating NE levels that we observed after 3 and 7 days (37). Opstad (179) observed higher circulating NE levels in soldiers following multiple days of exhaustive exercise coupled with sleep deprivation, and Young et al. (274) reported similar effects in soldiers following Ranger school. In this chronic exercise study, we observed that basal NE levels were elevated in our participants after three and seven consecutive days of exercise. Despite the elevation of basal NE concentrations, cold exposure elicited similar sympathetic activation during all three cold exposures, as evidenced by the increment in NE concentrations over preexposure levels observed by the end of each of the cold exposures, the magnitude of which did not differ among trials. Stimulation of adrenergic receptors is thought the primary mechanism which mediates cold-induced vasoconstriction (88). Since the increment in NE, relative to preexposure levels, was similar during all three cold exposure trials, a blunted sympathetic nervous stimulus does not appear to account for the less-pronounced vasoconstrictor response. However, a diminished sensitivity of the adrenergic receptors remains as a viable mechanism to explain the blunting of cold-induced vasoconstriction observed. Chronically elevated NE levels have been shown to decrease adrenergic receptor sensitivity in animal models (255), and similar effects have been suggested to develop in humans in whom circulating NE levels remain chronically elevated (179).

The specific fatigue-related physiological mechanisms by which thermoregulatory responses to cold become impaired following prolonged exercise remain unidentified. One possibility is that exercise-induced depletion of energy substrates impairs thermoregulation. Ainslie et al. (3) observed that men completing a 21-km, self-pace walk in mountainous terrain sustained lower deep body temperatures when energy intake was severely restricted (616 kcal, 2.6 mJ) compared to trials in which energy intake was significantly higher (3019 kcal, 12.6 mJ). However, a direct effect of dietary energy restriction impairing shivering and metabolic heat production during cold exposure seems unlikely. Research (115, 116, 273, 278) has generally shown that exercise-associated depletion of muscle glycogen is an unlikely explanation for the impaired shivering responses that we have reported. Further, while severe depletion of circulating energy substrate (i.e., blood

glucose) has been shown to impair shivering (93, 185), this too has been ruled out as a factor in the experimental observations of shivering fatigue that we have reported (36, 40, 274). Ainslie et al. (3) speculated that lower blood glucose during the restricted energy intake trials of their hill-walking study, might have caused an increase in peripheral blood flow, facilitating heat loss, compared to trials when energy intake was high, but presented no data to support this speculation. However, in the studies that found an increase in heat flow and skin temperature (32, 34), blood glucose levels did not decline and glucose levels appear unlikely to be mediating this response.

Therefore, a reasonable working hypothesis to explain "themoregulatory fatigue" and the impairment of thermoregulatory responses to cold with sustained overexertion is that with chronic sympathetic nervous activation, the capacity for further activation in response to an added cold stimulus may be limited. Perhaps adrenergic receptors involved in modulating cold-induced vasoconstriction become "downregulated" (reduced receptor density and affinity for ligand) by chronic sympathetic activation, associated with chronic exertion.

Conclusion

Cold tolerance and performance are impacted by many different factors. Cold tolerance is defined as the ability to adjust to cold stress so that physiological strain is minimized. Cold tolerance includes the habituation of the initial responses to cold, known as the cold-shock response, as well as the physiological adjustments that occur over long-term cold exposure. Many different factors impact upon cold tolerance including body composition, sex, age, race, fatigue, and multiple stressors. Cold habituation can attenuate many of these physiological responses. Exercise performance in the cold has not been as well studied as it has in other environmental extremes (e.g., heat and altitude). Aerobic and strength/power performance are both degraded during cold exposure, with the magnitude of decline related to the fall in muscle temperature. Other important factors that appear to impact physical performance include changes in peripheral blood flow and central cardiovascular limitations. Understanding the physiological mechanisms underlying cold's effect on performance is an area requiring further research.

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