AMERICAN COLLEGE of SPORTS MEDICINE®

POSITION STAND

Prevention of Cold Injuries during Exercise

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SUMMARY

It is the position of the American College of Sports Medicine that exercise can be performed safely in most cold-weather environments without incurring cold-weather injuries. The key to prevention is use of a comprehensive risk management strategy that: a) identifies/assesses the cold hazard; b) identifies/assesses contributing factors for cold-weather injuries; c) develops controls to mitigate cold stress/strain; d) implements controls into formal plans; and e) utilizes administrative oversight to ensure controls are enforced or modified. The American College of Sports Medicine recommends that: 1) coaches/athletes/medical personnel know the signs/symptoms and risk factors for hypothermia, frostbite, and nonfreezing cold injuries, identify individuals susceptible to cold injuries, and have the latest up-to-date information about current and future weather conditions before conducting training sessions or competitions; 2) cold-weather clothing be chosen based on each individual's requirements and that standardized clothing ensembles not be mandated for entire groups; 3) the wind-chill temperature index be used to estimate the relative risk of frostbite and that heightened surveillance of exercisers be used at wind-chill temperatures below −27°C (−18°F); and 4) individuals with asthma and cardiovascular disease can exercise in cold environments, but should be monitored closely.

INTRODUCTION

People exercise and work in many cold-weather environments (low temperature, high winds, low solar radiation, rain/water exposure). For the most part, cold-weather is not a barrier to performing physical activity. Successful and safe exploration to the North and South Poles, and swimming for hours across the English Channel are clearly indicative that human beings can perform in extreme cold. Many factors, including the environment, clothing, anthropometric factors, health status, age, and exercise intensity, interact to determine if exercising in the cold elicits additional physiological strain and injury risk beyond that associated with the same exercise done under temperate conditions. In many cases, exercise in the cold does not increase strain or injury risk, and David Bass, the noted environmental physiologist, once stated that "man in the cold is not necessarily a cold man." (6). However, there are scenarios (immersion, rain, low ambient temperature with wind) where whole-body or local thermal balance cannot be maintained during exercise-cold stress, contributing to cold-weather injuries and diminished exercise capability and performance. Furthermore, exercise-cold stress can increase the risk of morbidity and mortality in certain susceptible populations (45,84,98).

This position statement provides guidance to enable people exercising in the cold to avoid cold-weather injuries. Objectives of the Position Stand are to: 1) define the most common cold-weather injuries expected during exercise-cold stress, 2) present factors that increase the risk of sustaining a cold-weather injury, and 3) provide appropriate guidance to prevent or lower susceptibility to cold-weather injuries. Cold-weather outcomes or cold-related injuries include hypothermia, frostbite, cold urticaria, and nonfreezing cold injuries, and also outcomes secondary to being in the cold including cold-induced asthma and acute cardiovascular events such as myocardial infarction. Cold stress refers to environmental and/or personal conditions that tend to remove body heat and decrease body temperature. Cold strain refers to physiological and/or psychological consequences of cold stress. This position statement is applicable to all athletic activities in the cold including those that are short-term (jogging, running, skiing, biathlon, speed skating, outdoor hockey), medium term (adventure racing, triathlon, marathons, long-distance swimming), and long-term (mountaineering and expeditions).

EVIDENCE CLASSIFICATION

This Position Stand presents an evidence-based review based on a criteria scale proposed by the American Academy of Family Physicians. This scale is called the Strength of Recommendation Taxonomy (SORT). Table 1 presents the SORT. Recommendations are given a grade of

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TABLE 1. Strength of recommendation taxonomy.

Strength of Recommendation	Definition
A	Recommendation based on consistent and good quality patient oriented evidence (morbidity, mortality, symptom improvement, cost reduction, and quality of life).
B	Recommendation based on inconsistent or limited-quality patient-oriented evidence.
C	Recommendation based on consensus, usual practice, opinion, disease-oriented evidence (measures of intermediate, physiologic, or surrogate end-points that may or may not reflect improvements in patient outcomes), or case series for studies of diagnosis, treatment, prevention, or screening.

A, B, or C based on patient- or disease-oriented outcomes. The SORT can be accessed from the *American Family Physician* Web site, www.aafp.org. The reader should be cognizant that classifying evidence using this taxonomy heavily emphasizes outcome-based treatments. However, outcome-based research on medical effects of exposure to cold is limited by the ethical constraints that preclude the design of studies that use frostbite or severe hypothermia as an outcome in human volunteer subjects.

PHYSIOLOGICAL RESPONSES TO COLD

Acute cold exposure. Humans exhibit peripheral vasoconstriction upon cold exposure. The resulting decrease in peripheral blood flow reduces convective heat transfer between the body's core and shell (skin, subcutaneous fat and skeletal muscle), effectively increasing insulation by the body's shell (39,168,178). Heat will then be lost from the exposed body surface faster than it is replaced, so skin and underlying tissue temperatures decline (168). During whole-body cold exposure, the vasoconstrictor response occurs throughout the entire body's peripheral shell and the limbs effectively become part of the shell. Vasoconstriction begins when mean weighted skin temperature falls below 34–35°C (159), and becomes maximal when mean skin temperature is about 31°C or less during whole-body water immersion (185) or 26–28°C during localized cooling (22). Thus, the vasoconstrictor response to cold exposure helps retard heat loss and defend core temperature, but at the expense of a decline in skin and muscle temperatures.

The vasoconstriction-induced blood flow reduction and fall in skin temperature probably contribute to the etiology of peripheral cold injuries. Cold-induced vasoconstriction has pronounced effects in the hands and fingers making them particularly susceptible to cold injury and a loss of manual dexterity (13). In these areas, another vasomotor response, cold-induced vasodilation (CIVD), modulates the effects of vasoconstriction (114,133). Periodic oscillations of skin temperature follow the initial decline during cold exposure, resulting from transient increases in blood flow to the cooled finger. A similar CIVD also occurs in the forearm, likely reflecting the CIVD of the extremities (40). It is believed that CIVD plays a substantial role in reducing the risk of local cold injuries (86) and may be beneficial for improving dexterity and tactile sensitivity during exposure to cold (25). CIVD responses are more pronounced when the body core and skin temperatures are warm (hyperthermic state) and suppressed when they are cold (hypothermic state), when compared to normothermia (26,27,135).

Cold exposure also elicits an increased metabolic heat production in humans, which can help offset heat loss. In humans, cold-induced thermogenesis is attributable to skeletal muscle contractile activity (168). Humans initiate this thermogenesis through involuntary shivering or by voluntarily modifying behavior, i.e., increasing physical activity (exercise, increased "fidgeting," etc). While certain animals exhibit an increased metabolic heat production by noncontracting tissue (brown adipose tissue) in response to cold exposure, i.e., nonshivering thermogenesis, experimental evidence does not support a large role for brown-fat mediated thermogenesis in adult humans (4,18).

Shivering, which consists of involuntary, repeated, rhythmic muscle contractions, may start immediately, or after several minutes of cold exposure, usually beginning in torso muscles, then spreading to the limbs (8). The intensity and extent of shivering varies according to the severity of cold stress. As shivering intensity increases and more muscles are recruited to shiver, whole body oxygen uptake increases, typically reaching about 600–700 $mL \cdot min^{-1}$ during resting exposure to cold air, but often exceeding 1000 $mL \cdot min^{-1}$ during resting immersion in cold water (178). Maximal shivering is difficult to quantify, but the highest oxygen uptake reported in the literature to date appears to be 2.2 $L \cdot min^{-1}$, recorded during cold-water immersion, and this was ~ 6 times the resting metabolic rate (50% $\dot{V}O_{2max}$) for that subject (47).

Patterns of human cold acclimatization. Athletes exposed to cold weather may acclimatize but the physiologic adjustments are very modest and depend on the severity of the exposures. Cold acclimatization in persons repeatedly or chronically exposed to cold manifests in three different patterns of thermoregulatory adjustments: habituation, metabolic acclimatization, and insulative acclimatization (193).

The most commonly observed acclimatization pattern exhibited is habituation, in which physiological responses to cold become less pronounced than in the unacclimatized state. Blunting of both shivering and cold-induced vasoconstriction are the hallmarks of habituation (193). Cold-habituated persons with blunted shivering and vasoconstrictor responses to cold, sometimes, but not always, also exhibit a more pronounced decline in core temperature during cold exposure than nonacclimatized persons. Thus, this pattern of cold acclimatization is sometimes referred to as hypothermic habituation, or hypothermic acclimatization. Findings from different cold acclimation studies, when viewed collectively (see (193) for a detailed review), suggest that short intense cold exposures (e.g., less than 1 h), a few times per week will produce habituation, but that longer exposures (e.g., more than 8 h) to more moderate cold conditions, on consecutive days over a fairly long period

(e.g., more than 2 wk) are required to induce the hypothermic form of habituation. Habituation also occurs locally (i.e., hands), leading to warmer skin temperatures and decreased discomfort (1,108,161).

Chronic cold exposure can induce two other distinct patterns of acclimatization. A more pronounced thermogenic response to cold characterizes the metabolic acclimatization pattern (193). An exaggerated shivering response has been reported to develop because of chronic cold exposure, and the possibility that humans develop a nonshivering thermogenesis continues to be argued. However, the evidence purporting to document the existence of this pattern does not definitively demonstrate whether this enhanced thermogenic response to cold represents an adjustment to chronic cold, or confounding effects of differences in diet or body composition among experimental and control subjects.

The third major pattern of cold acclimatization, referred to as insulative cold acclimatization, is characterized by enhanced heat conservation mechanisms (193). With insulative acclimatization, cold-exposure elicits a more rapid and more pronounced decline in skin temperature and lower thermal conductance at the skin than in the unacclimatized state, mediated by a more pronounced vasoconstrictor response to cold, possibly due to enhanced sympathetic nervous response to cold. In addition, some data suggest that insulative cold acclimatization may also involve development of enhanced circulatory countercurrent heat exchange mechanisms to limit convective heat loss, as evidenced by the observation that before wet-suits came into common usage, Korean diving women immersed in cool water exhibited lower forearm heat loss than control subjects, despite the fact that forearm blood flow remained higher in the diving women (83). After wet-suit use became widespread, Korean diving women no longer exhibited any thermoregulatory adjustments compared to control subjects, suggesting that the previous differences truly reflected adjustments to frequent exposure to cold while diving (for review see (193)). Compared to the effects of heat acclimatization, physiological adjustments to chronic cold exposure are less pronounced, slower to develop, less reproducible, and less practical in terms of relieving thermal strain, defending normal body temperature, and preventing thermal injury.

HYPOTHERMIA

Clinically, hypothermia is defined as a core temperature below 35°C (95°F), which represents ~ 2°C (3.5°F) fall from normal body temperature (143), while physiologically, hypothermia is a core temperature below the value observed typically observed during active phases (< 36.8°C). Hypothermia develops when heat losses exceed heat production causing the body heat content to decrease. Declines in core temperature may eventually impact exercise performance.

Hypothermia is characterized as mild, moderate, or severe (143). Table 2 lists the core temperatures and physiological changes associated with these low body core temperatures. The symptoms of hypothermia are quite variable from person to person even at the same core temperature. Early symptoms of hypothermia include feeling cold, shivering, and exhibiting signs of apathy and social withdrawal. Coaches and athletes should be aware of these early symptoms so that proper preventative measures can be taken at this time. More pronounced hypothermia manifests as confusion or sleepiness, slurred speech, and a change in behavior or appearance (158). Severe hypothermia is associated with changes in cardiac rhythms requiring immediate treatment to rewarm and restore normal temperature. Resuscitation has been successful even with core temperatures as low as 13.7°C (60). At these temperatures, life signs are almost impossible to discern and no one should be pronounced dead until they have been rewarmed. Hence the use of the adage, "a person is not dead until they are warm and dead."

Table 3 presents a list of factors thought to predispose individuals to hypothermia. Important risk factors commonly encountered by exercisers are considered in detail below. The majority of factors known to influence the onset of hypothermia have been identified in experiments that used whole-body cold water immersion, but the risk factors are likely to have similar influence, if less pronounced, on hypothermia onset in cold-air exposure.

TABLE 2. Core temperature and associated physiological changes that occur as core temperature falls. Individuals respond differently at each level of core temperature.

Stage	Core Temperature °F	Core Temperature °C	Physiological Changes
Normothermia	98.6	37.0	
Mild	95.0	35.0	Maximal shivering, increased blood pressure
	93.2	34.0	Amnesia; dysarthria; poor judgment; behavior change
	91.4	33.0	Ataxia; apathy
Moderate Hypothermia	89.6	32.0	Stupor
	87.8	31.0	Shivering ceases; pupils dilate
	85.2	30.0	Cardiac arrhythmias; decreased cardiac output
	85.2	29.0	Unconsciousness
Severe Hypothermia	82.4	28.0	Ventricular fibrillation likely; hypoventilation
	80.6	27.0	Loss of reflexes and voluntary motion
	78.8	26.0	Acid-base disturbances; no response to pain
	77.0	25.0	Reduced cerebral blood flow
	75.2	24.0	Hypotension; bradycardia; pulmonary edema
	73.4	23.0	No corneal reflexes; areflexia
	66.2	19.0	Electroencephalographic silence
	64.4	18.0	Asystole
	59.2	15.2	Lowest infant survival from accidental hypothermia
	56.7	13.7	Lowest adult survival from accidental hypothermia

TABLE 3. Predisposing factors for hypothermia based on cause. Table drawn from data presented in (31,143).

Decreased Heat Production	Increased Heat Loss	Impaired Thermoregulation	Miscellaneous Clinical States
Training factors	*Training factors*	*Peripheral failure*	
• Inactivity	• Immersion	• Trauma	Infection
• Fatigue	• Rain	• Neuropathies	Renal failure
• Energy depletion	• Wet clothing from sweat	• Acute spinal cord transection	
• Lack of sleep	• Wind	*Central failure*	
Endocrine	• Fatigue	• Central nervous system (CNS) lesions and trauma	
• Hypopituitarism	• Low body fat	• Stroke	
• Hypoadrenalism	*Age (young & old)*	• Subarachnoid hemorrhage	
• Hypothyroidism	*Erythrodermas*	• Hypothalamic dysfunction	
• Hypoglycemia	• Burns	• Parkinson's disease	
• Diabetes	• Psoriasis	• Multiple sclerosis	
Age (young & old)	• Icthyosis	• Pharmacologic	
	• Exfoliative dermatitis	• Toxicologic	
	• Sunburn	• Drug and alcohol abuse	
	Iatrogenic		
	• Emergency birth		
	• Cold infusions		
	• Heat illness treatment		
	• Open wound		

Immersion, rain and wind. Water has a much higher thermal capacity than air, with the convective heat transfer coefficient being 70 times greater compared to air (54). Therefore, swimmers and athletes exercising in rainy weather can experience considerable body heat loss even in relatively mild water or air temperatures.

Thermal balance during exercise-cold water immersion and cold-wet air exposure depends on a complex interaction among metabolic heat generated, exercise mode, anthropomorphic and clothing factors that insulate, and magnitude of cooling caused by water temperature, rain, and wind. Individuals vary with respect to the water temperature that can be tolerated without experiencing a dangerous decline in core temperature during exercise (178). A decrease in water temperature increases the thermal gradient between the person and the environment and leads to significantly greater heat loss via convection and conduction. The more of a person's surface area is immersed, the greater the effective heat exchange area is between the person and the water. As surface area immersed increases, core temperature will decrease more rapidly (109).

The maintenance of a normal core temperature also depends on the ability to generate enough heat to offset heat lost to the environment. Exercise in cold water may either increase or decrease core temperatures compared to rest in cold water (157,178,180), depending on whether exercise was performed with the legs-only or if a combination of arms and legs were used. Arm-leg exercise (e.g., swimming) increases the circulation to the extremities where peripheral heat loss is optimized due to the small diameter of the extremities and short conductive pathway for heat transfer from the limb core to the skin surface (19,181). However if the exercise intensity is high enough (~75% $\dot{V}O_{2max}$ or 2.75 $L \cdot min^{-1}$), core temperature will increase (49), even though combined arm-leg exercise is performed. Muscle provides an important insulator at rest during cold-water immersion (39,185), but during exercise this insulation is reduced, as blood flow increases to support metabolism (178,185). Thus there may be some benefit from adding clothing insulation to cover active muscle areas during prolonged, active immersions as in long-distance cold water swimming (186).

As with cold water immersion, many factors interact to determine if core temperature can be maintained during exercise in cold, wet, and windy conditions. At an air temperature of 5°C, heat loss in wet clothes may be double that in dry conditions (97). Furthermore, wind increases convective heat loss. Exercise performed at intensities greater than 60% $\dot{V}O_{2max}$ can maintain core temperatures at or above 37°C (146,188,189) when the ambient temperature is 5°C, clothes are completely wet, and the wind is 5 $m \cdot s^{-1}$. However, when only light exercise is performed (< 30% $\dot{V}O_{2max}$), heat losses exceed heat production leading to declines in core temperature (20,145,146,188). Also exercise performed before cold-water immersion (163) or exercise in the rain (20) leads to more rapid declines in core temperature compared to not exercising. **Evidence Statement**. Exercising in water and rain significantly increases the risk for developing hypothermia. *Category A*

Anthropometry and % fat. Subcutaneous body fat provides a relatively high thermal resistance (178) and persons who have a high % fat tend to maintain core temperature better than lean people (64,78,99,124,145,146,178), although this has not always been observed (62). There is also some evidence that individuals with fat greater than 25% have a higher threshold for vasoconstriction and this enables them to limit heat loss (96). Unperfused muscle also provides insulation during resting cold exposure (148) and can contribute as much as 85% of the limb insulation (39). During exercise, perfused muscle loses its insulatory potential and skin and subcutaneous fat provide most of the insulation (179). Some studies suggest that people with large surface area-to-mass ratios have a more rapid fall in core temperature (16,165), although in two studies where subjects were matched for body fat but differed in body mass and surface area-to-mass ratio, core temperature was the same at rest and during exercise (63,179). **Evidence Statement**. Individuals with high combined values of subcutaneous fat thickness, % fat, and muscle mass can maintain core temperature better than individuals with less fat and muscle. *Category B*

Sex. Sex differences in thermoregulatory responses during cold water exposure are primarily attributable to the woman's generally greater body fat content, thicker subcutaneous fat layer, less muscle mass, and higher surface area-to-mass ratio than men of comparable age and weight (176). However, in women and men of equivalent subcutaneous fat thickness, the women have a greater surface area and smaller total body mass and musculature (and lower total body heat content) than men. Thus, total heat loss is greater in the women, versus men, due to the larger surface area for heat loss and less insulation provided by muscle, and body temperature falls

more rapidly during resting cold water immersion (124). Interestingly, during exercise in cold water, men and women of equivalent percent body fat exhibit the same decline in core temperature, perhaps due to loss of insulation in perfused muscle in men and a favorable distribution of subcutaneous fat over active musculature in women (125). Other data also suggest that men begin shivering sooner and at higher mean body temperatures than women, i.e., men are more sensitive to a change in body temperature (69), although Glickman-Weiss et al. did not find a sex effect on thermosensitivity between men and women (61). Cyclic changes in female reproductive hormones also may impact thermoregulatory responses to cold. Data suggest that the onset of shivering occurs sooner in the luteal phase (80) when estrogen and progesterone levels peak, although this finding has been challenged (68) and there are no data to suggest that differences in the absolute starting core during cold exposure places a women at higher risk for hypothermia in the follicular vs. luteal phase. Amenorrheic women cannot maintain their core temperature during exercise in cold air as well as their eumenorrheic counterparts, even if they have a similar body composition profile (69). **Evidence Statement**. Core temperature responses to cold exposure between average men and women are primarily attributable to differences in body composition and anthropometry. *Category C*

Age. People who are older than 60 yr may be less cold tolerant than younger persons, due to reduced vasoconstriction and heat conservation in comparison to their younger counterparts (48,102,166,194). Older people 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 $\%\dot{V}O_{2max}$, will fatigue sooner, and must decrease their absolute heat production if they fatigue, increasing the likelihood of a reduction in core temperature. Older individuals also appear to have a blunted thermal sensitivity to cold. For example, in studies where subjects have control of setting a thermostat as the ambient temperature fluctuates, older individuals will allow the air temperature to fall to lower levels before readjusting the thermostat (137,173).

Children, in comparison to adults, typically have a higher body surface area-to-mass ratio and lower subcutaneous fat amounts and this leads to substantial falls in core temperature when swimming in cold (20°C) water (165). Interestingly, in 11–12-yr-old boys who had similar amounts of subcutaneous fat as men (19–34 yr), core temperature was the same at rest and during exercise in 5°C air between the groups, but the mechanism for achieving this was different with the boys exhibiting a more pronounced vasoconstrictor and metabolic response compared to the men (167). Premenarcheal girls do not defend core temperature as well as eumenorrheic girls during exercise-cold stress, due to a diminished vasoconstrictor response (105). **Evidence Statement**. Older individuals (> 60 yr) are at an increased risk of hypothermia due to blunted physiological and behavioral responses to cold. Children are at a greater risk of hypothermia than adults due to differences in body composition and anthropometry. *Category B*

Hypoglycemia and fasting. Recent findings show that shivering, like low intensity exercise, relies on lipid as the predominant metabolic substrate in well-fed individuals, but that blood glucose, muscle glycogen and even some protein are also metabolized (72,73). Underfeeding can lead to hypoglycemia, and acute hypoglycemia impairs shivering through a central nervous system mediated effect (55,140). Also, declining peripheral carbohydrate stores probably contribute to an inability to sustain exercise thermogenesis during cold exposure (141). Glycogen depletion, itself, has been shown, during cold-water immersion, to either impair initial shivering rates (121) or to have no effect on shivering thermogenesis (196). Muscle glycogen depletion has been observed to be more pronounced during low intensity (e.g., below 25% maximal oxygen uptake) exercise-cold stress compared to temperate conditions, but differences between environments are not seen when bouts of higher intensity exercise are compared (162). Shivering bursts also affect muscle glycogen levels, with more bursts leading to greater glycogen utilization (71). Complete food restriction for 48 h, even in the absence of hypoglycemia, impairs shivering and causes core body temperatures to decline more rapidly (116,120). **Evidence Statement**. Hypoglycemia impairs shivering and increases the risk for hypothermia. *Category B*

Physical fitness and training. Overall, physical training and level of fitness appear to have only minor influences on thermoregulatory responses to cold (48). Cross-sectional comparisons of aerobically fit and less fit persons find relationships between maximal aerobic power and temperature regulation in the cold (9,90), but in those studies, differences in thermoregulation appear more likely attributable to anthropometric differences between the aerobically fit and less fit subjects, rather than an effect of fitness state, training, or the level of maximal aerobic power, per se (9). In a recent study comparing novice and expert swimmers (115), the expert swimmers could swim further, but not longer, before becoming incapacitated in cold water compared to the novice swimmers. This is likely because arm fatigue due to muscle cooling was the primary cause of swim failure. Longitudinal studies have shown interval training has no measurable effects on thermoregulatory response to cold (160), and that while endurance training appears to strengthen cutaneous vasoconstrictor responses to cold, that effect has little impact on core temperature changes experienced during cold exposure (195). The effects of resistance training programs on thermoregulatory responses to cold have not been documented, but it seems likely that any such effects would be primarily attributable to training-related changes in body composition. The primary thermoregulatory advantage provided by the increased strength and aerobic power resulting from physical training is that the fitter individual can sustain voluntary activity at a higher intensity, and thus sustain higher rates of metabolic heat production than less

fit persons during cold-exposure. **Evidence Statement.** Physical fitness and training, per se, do not improve thermoregulatory responses to cold. Physical fitness does allow someone to exercise for a longer period at a higher metabolic rate, and may contribute to maintenance of normal core temperatures. *Category C*

Prevention Strategies for Hypothermia

Risk management. Hypothermia is best prevented by first assessing how cold it is by monitoring the temperature, wind, solar load, rain, immersion depth, and altitude (34). Then the hazard of exercising in the cold is assessed by analyzing the exercise regimen to be performed, the clothing available, and identifying those who are at higher risk of getting hypothermia. Specific factors that can be evaluated include the exercise intensity, duration, experience of the athlete, condition of the athlete (fit and rested or fatigued), general health, and nutritional status.

Risk management is the process of identifying potential hazards before performing in cold weather and taking the steps necessary to control these hazards (34), because hypothermia can occur during athletic events (93,149). Figure 1 outlines a cold strain risk management process for preventing cold injuries. An important aspect of this is recognizing changes in weather conditions so that people can be alerted to potential modifications that may be necessary to reduce exposure and susceptibility to cold injuries. Therefore, the risk management process must continually be reevaluated as input changes. Planning ahead could mean bringing additional clothing, cutting short the duration of an event, changing venues, offering warming facilities, or possibly even canceling an event. The greatest occurrence of hypothermia happens when people are not prepared for it, i.e., when people are not expecting it (rainy weather in spring/summer/fall; ocean/lake swimming on a hot day in spring and early summer). As stated above, cold, wet, and windy weather poses the

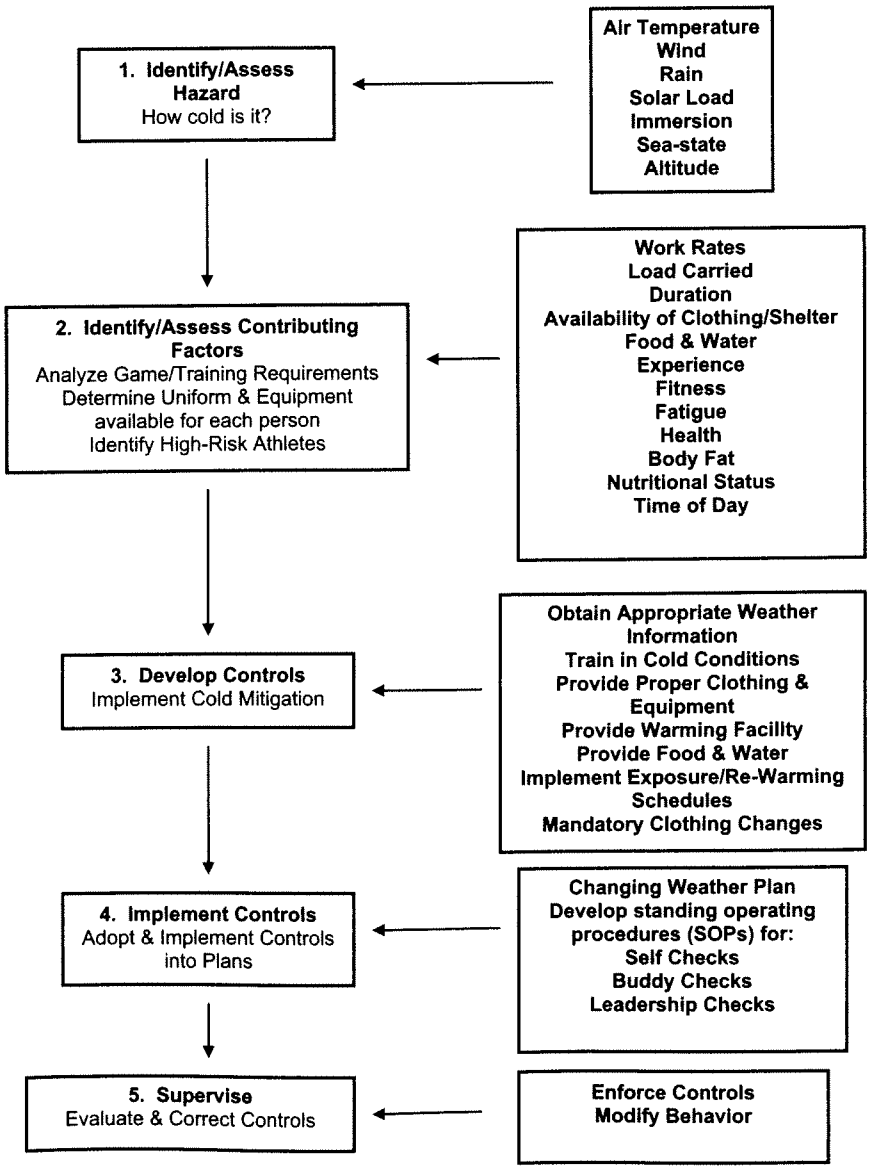

FIGURE 1—Risk management process for evaluating cold stress and strain.

greatest risk for developing hypothermia. Heat loss is much greater in these conditions and if the exercise intensity is not high enough to match heat loss (123,146,189) due to fatigue or if fatigue occurs before cold exposure (20,21), an individual may be more susceptible to hypothermia.

Clothing. Cold weather clothing protects against hypothermia and peripheral cold injuries by reducing heat loss through the insulation provided by the clothing and the trapped air within and between clothing layers (7). Typical cold-weather clothing consists of three layers: an inner layer (lightweight polyester or polypropylene) which is in direct contact with the skin and does not readily absorb moisture, but wicks moisture to the outer layers where it can evaporate, a middle layer (polyester fleece or wool) which provides the primary insulation, and an outer layer, which is designed to allow moisture transfer to the air, while repelling wind and rain. Sweating can easily exceed the vapor transfer rate of the outer shell layer, causing moisture to accumulate on the inside, even if the outer layer has substantial venting (e.g., zippers in armpits) to allow moisture to escape. The outer layer should typically not be worn during exercise (unless it is rainy or very windy), but should be donned during subsequent rest periods.

Clothing insulation needs during physical activity can vary with changes in the ambient temperature and exercise intensity. Figure 2 depicts the insulation needed to maintain thermal balance at different ambient temperatures and exercise intensities (7,66,82,88). Table 4 presents the approximate insulation of various clothing articles and ensembles (89). As the exercise intensity increases (jogging, skiing—consult (2) for a comprehensive list of activities and metabolic requirements), the amount of clothing insulation needed to maintain body heat content and thermal balance decreases at any given air temperature. Imposing a single standard clothing ensemble for an entire group could result in overheating and sweating during exercise in some, while others would not be kept warm, therefore people should adjust clothing according to their own needs. A common problem is that people begin exercising while still wearing clothing layers appropriate for resting conditions, and thus, are "overdressed" after initiating exercise. If the combination of environmental conditions, work intensity, and available clothing suggest that body heat content cannot be maintained (e.g., low exercise intensity in rainy conditions), then supervision of the exerciser or use of the buddy system should be encouraged. All exercisers need to be aware that the risk of hypothermia increases if the weather is wet and wet-weather clothing is not available and exercise intensity is low (e.g., stop running and begin walking). Remaining dry, especially for those exercising in remote regions, is extremely important and dictates that carrying extra clothing that is waterproof and dry clothing to change into is vital.

Wet suit use is becoming more widespread, especially during triathlon competitions. They are primarily used in recreational diving and commercial fishing to maintain body core temperatures and increase survival time during immersion (177). The international swimming association (FINA) has adopted guidelines to allow wet suit use during

TABLE 4. Approximate clothing insulation of various representative clothing ensembles that may be worn in cold weather (89). Values are tabulated from measurements on copper manikins (89). Insulation values provided in units of clo and SI units.

Clothing Ensemble	Clothing Insulation (I_{cl}) clo	$m^2 \cdot °C \cdot W^{-1}$
Shirt, light-weight trousers, socks, shoes, underwear briefs	0.6	0.095
Shirt, trousers, jacket, socks, shoes, underwear briefs	1	0.155
Windproof, waterproof jogging suit (shell jacket and pants), T-shirt, briefs, running shorts, athletic socks, athletic shoes	1.03	0.160
Fleece long-sleeve shirt, fleece pants, briefs athletic socks, athletic shoes	1.19	0.184
Lightweight jacket, thermal long underwear top and bottoms, briefs, shell pants, athletic socks, athletic shoes	1.24	0.192
Lightweight jacket, long sleeve fleece shirt, fleece pants, underwear briefs, shell pants, athletic socks, athletic shoes	1.67	0.259
Ski jacket with detachable fiberfill liner, thermal long underwear bottoms, knit turtleneck, sweater, fiberfill ski pants, knit hat, goggles, mitten shell with fleece glove inserts, thin knee-length ski socks, insulated waterproof boots	2.3	0.357
Extreme cold weather down-filled parka with hood, shell pants, fiberfill pants liner, thermal long underwear top and bottoms, sweat shirt, mitten shell with inner fleece gloves, thick socks, insulated waterproof boots	3.28	0.508
Extreme cold weather expedition suit with hood (down—filled, one—piece suit), thermal long underwear top and bottoms, sweat shirt, mittens with fleece liners, thick socks, insulated waterproof boots	3.67	0.569

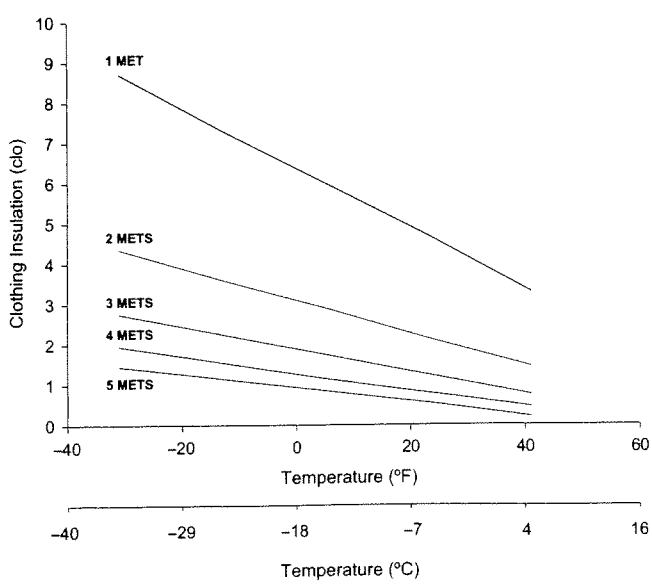

FIGURE 2—Approximate amount of clothing insulation needed at different air temperatures and physical activity levels. Wind speed is assumed to be less than 5 mph (2.2 m·s^{-1}). One MET refers to energy expenditure at rest. One clo of insulation is the clothing necessary to allow a resting person to be comfortable when the air temperature is 21°C (70°F) (7). Refer to Table 4 for a list of typical clothing ensembles and their respective clo values.

triathlons to aid in thermal protection (www.fina.org). This guidance is based on athletic status (elite or not), swim length, and water temperature. For example, an elite triathlete swimming a course between 1500 and 3000 m cannot use a wet suit if the water temperature is above 23°C, but must wear one if the water temperatures is below 15°C. Studies have shown that wet suits reduce drag (182), increase buoyancy and lower oxygen consumption at any given swimming speed (183), and thus, their use in swimming competitions is primarily as a performance enhancer. For this reason, their use has been banned in open-water swimming competitions (e.g., English Channel). Core temperature goes up slightly when swimming with a wet suit at 20°C (184) and wet suits have no negative impact on further triathlon performance (cycling, running) after swimming in 25°C water (103). At lower water temperatures, wet suits with arm protection may provide the best thermal protection during swimming (132) since arm exercise causes a greater cooling rate than leg only or combined arm-leg exercise (181).

Heat losses from the head have been measured up to 50% of the total resting heat production in a person sitting in −4°C (25°F) air while wearing winter clothing (53). Knit caps and balaclavas can decrease this heat loss substantially. Headbands can be used to cover the ears, but allow for heat loss through the head. Socks should not fit tight and constrict blood flow. Shoes can be to one size larger with thick socks. Feet perspire even in the cold, particularly in heavy winter boots. This necessitates changing socks at least 2 times per day, but perhaps even more if activities levels are high. **Evidence Statement.** Clothing insulation requirements during exercise are a function of metabolic rate and ambient temperature. Layering provides the most flexibility to adjust insulation to prevent sweating, overheating, underdressing, and remaining dry in wet conditions. *Category C*

Food and fluid intake. Athletes can expend more energy during cold weather (by 10–40%, (52)) due to a combination of heavy clothing and equipment and the increased effort required for working or walking in snow or mud (139). The body may also expend more energy keeping warm through shivering when the weather is cold, but this depends on how well the individual has protected herself through proper clothing choices. If the core temperature remains above resting values during exercise, cold exposure does not increase oxygen uptake or caloric requirements above normal (188,189). In most cases, people do not need to change from their normal diet to meet their caloric needs in cold weather since they are not continually exercising for days and weeks (e.g., mountaineering). If caloric requirements are indeed higher, the 10–40% extra calories needed per day can be obtained by eating a "normal" breakfast, lunch, and dinner, and then supplementing with frequent snacks throughout the day. For the majority of exercisers who do not experience a decline in core or muscle temperature, fatigue is related to carbohydrate availability rather than thermoregulatory limitations (56,141) and exercise can be sustained by ingesting carbohydrate beverages of 6–12% (56,57). Furthermore, since carbohydrate availability appears limiting, carbohydrate loading to maximize muscle glycogen stores before exercise in the cold is beneficial (141). Thus, as with exercise in temperate environments, the majority of people who exercise for very long durations in cold weather (cross-country skiers, marathoners) will maintain performance by eating carbohydrate-rich foods such as crackers, potatoes, cereals, bread, and pasta.

Fluid balance may be affected by cold-weather exercise. Exercise can increase sweat loss in the cold just as in temperate climates by increasing core temperature and initiating thermoregulatory sweating (52). Sweat losses occur if activities are performed at a high intensity, while wearing heavy cold-weather clothing systems, and traversing in snow resulting in high metabolic rates (139). In these conditions a person could become dehydrated if fluid intake is substantially lower than fluid loss. In addition, if skin temperatures fall significantly, thirst is less noticeable in cold compared to hot weather (101). Exposure to cold air or immersion in cold water may also increase urine flow rate, the so-called cold-induced diuresis (CID). This response is likely caused by a redistribution of body fluids from the periphery to the central circulation as peripheral vasoconstriction occurs (52). CID is self-limiting because the response diminishes as body water content falls. CID is also prevented by moderate-intensity exercise (112).

Moderate fluid loss may not be as important for exercise performance in the cold as it is for temperate and hot environments. Recent data (23) show that if the skin temperatures are low, 4% dehydration has no effect on cycling performance in the cold. But if cold strain is minimized by clothing, thereby maintaining skin and core temperatures near that observed in temperate or even hot environments, dehydration will likely degrade performance (52). Dehydration does not alter heat conservation, heat production, or CIVD responses (134,136) and thus does not appear to increase the likelihood of cold injuries.

Simple solutions can be instituted to ensure adequate hydration before and during exercise. Before exercise, athletes can monitor hydration status by noting the color and volume of their urine and their body weight. Dark, low volume and infrequent urination indicates that fluid consumption should be increased. Likewise, frequent and large volumes of clear urine indicate that fluid replacement is adequate. Body weight can be assessed daily. People usually drink most of their water with meals, and eating food improves fluid consumption (87,170). During mealtime individuals can drink a variety of fluids (milk, juice, tea, sports drink, coffee), as each will be equally effective in replacing body water (87). In addition, meals provide the salt intake necessary to retain body water. Sodium-containing beverages, compared to pure water, have been shown to aid in fluid retention (~ 1 kg more fluid retained with Na^+) over several days of a cold survival scenario (150), but little information is available on their effectiveness during short-duration exercise bouts in cold-weather. Snow, in most cases, should be avoided because it can

potentially lower body temperature, contains dirt and other pollution, and provides relatively little water per volume of snow to counteract dehydration. However in a person with a normal or high body temperature, snow is not contraindicated if it is the only source of water. During exercise, frequent fluid intake can be an effective strategy for maintaining hydration. **Evidence Statement**. Cold environments can increase energy expenditures and may cause fluid losses; dehydration does not impair vasoconstriction or shivering, thus dehydration does not increase susceptibility to cold injuries. *Category C*

FROSTBITE

Frostbite occurs when tissue temperatures fall below 0°C. The freezing point of skin is slightly below the freezing point of water due to the electrolyte content of the cells and extracellular fluid, with the skin surface reportedly freezing from −3.7 to −4.8°C (30,129,192). Wet skin will cool faster (129), will reach a lower temperature (12), and will freeze at a higher threshold (~−0.6°C, (100)). Frostbite is most common in exposed skin (nose, ears, cheeks, exposed wrists), but also occurs in the hands and feet because peripheral vasoconstriction significantly lowers tissue temperatures (33). Instantaneous frostbite can occur when the skin comes in contact with supercooled liquids, such as petroleum products, oil, fuel, antifreeze, and alcohol, all of which remain liquid at temperatures of −40°C. Contact frostbite can occur by touching cold objects with bare skin (particularly highly conductive metal or stone), which causes rapid heat loss.

Usually, the first sign of frostbite is numbness. In the periphery, the initial sense of cooling begins at skin temperatures of 28°C (~82°F; (81) and pain appears at ~ 20°C (68°F; (42,81), but as skin temperature falls below 10°C (50°F) these sensations are replaced by numbness (144). Individuals often report feeling a "wooden" sensation in the injured area. After rewarming, pain is significant. The initial sensations are an uncomfortable sense of cold, which may include tingling, burning, aching, sharp pain, and decreased sensation (128). The skin color may initially appear red; it then becomes waxy white. Note that peripheral temperatures (hands, feet) may be indicative of a generalized whole body cooling that may ultimately result in hypothermia. Body heat content has been found to be directly related to the skin temperature of fingers and toes (14).

Table 5 presents the predisposing factors for frostbite. Risk factors are separated by cause including environmental, mechanical, physiological, and psychological. For the relatively healthy athlete, the most relevant are the environmental and mechanical factors since these can be prevented through recognition and employing appropriate countermeasures. Important risk factors that cannot be changed include sex and race. Peripheral responses to cold 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 (76,142,147). Contact cooling studies suggest that women's fingers cool faster than men, possibly due to hand size (91). Prevalence of peripheral vascular disorders like Raynaud's phenomenon is also higher in women, which may also make them more susceptible to peripheral cold injury (70). Raynaud's phenomenon (RP) is 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 (190). The affected area first turns white, blue if the area becomes cyanotic and finally red upon rewarming. RP is associated with tingling, swelling, or painful throbbing. Individuals with scleroderma, lupus or arthritis are more likely to suffer from RP as are individuals who live in cold-weather regions (11). Black men and women are 2–4 times more likely to suffer a cold weather injury than their Caucasian counterparts (33). Early information on frostbite risk due to race was conducted in military populations and observed higher frostbite rates in blacks (17,172) and a recent epidemiological study (33) 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 CIVD, increased sympathetic response to cold exposure, and thinner, longer digits (17).

In most cases, sojourning to high altitude is synonymous with cold exposure. The air temperature decreases 2°C with every 310 m (1000 ft) above the site at which the temperature was measured. Also, the wind chill temperature will be lower at higher altitudes due to the combination of lower air temperatures and higher wind speeds caused by less tree cover. Epidemiological evidence suggests that the risk of frostbite significantly increases above 5182 m (77). The combination of the known cognitive deficits elicited by hypoxia (5) and preliminary

TABLE 5. Predisposing factors for frostbite and peripheral cold injury. Table drawn from data presented in references (24,32,33,44,94,106).

Environmental	Mechanical	Physiological	Psychological
• Temperature • Wetness • Exposure duration • Wind chill • Altitude • POLs (petroleum, oil, lubricants) • Contact with metals	• Constrictive clothing • Inadequate clothing & shelter • Tight boots • Cramped positioning	• Hypothermia • Race • Sex • Wet skin • Prior peripheral cold injury • Trauma • Erythrodermas • Hyperhidrosis • Hypoxia • Smoking • Energy depletion • Poor physical conditioning • Hypotension • Atherosclerosis • Raynaud's syndrome • Vasospastic disorders • Diabetes • Shock • Vasoconstrictor drugs	• Severe mental stress

data suggesting that cutaneous sensitivity to cold is blunted in the toes (65) can potentially lead to poor behavioral choices at high altitude and increase individual susceptibility to cold injury. Physiologically, CIVD responses appear to be blunted by altitude exposures (> 4350 m) in nonaltitude acclimatized subjects (28,122,171), with possibly some restoration of responses after altitude acclimatization of at least 21–45 d (28,122). Altitude exposure (> 8000 ft) also decreases the shivering and vasoconstrictor response to cold exposure (10,92).

Prevention Strategies for Frostbite

Wind chill. The principal cold-stress determinants during outdoor activities in cold weather 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 (67). Wind exacerbates heat loss by facilitating convective heat loss (54) and reduces the insulative value of clothing. The wind chill temperature (WCT) index (Fig. 3) integrates wind speed and air temperature to provide an estimate of the cooling power of the environment (130,138). 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 $m \cdot s^{-1}$ (3 mph). Wind does not cause an exposed object to become cooler than the ambient temperature, but instead wind causes exposed objects to cool toward ambient temperature more rapidly than without wind. Wind speeds obtained from weather reports do not take into account man-made wind. For example, running and skiing produce wind across the body at the same rate as the body is moving. The WCT presents the relative risk of frostbite and the predicted times to freezing (Fig. 3) of exposed facial skin (38). Facial skin was chosen

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	−66	−72
15	32	25	19	13	6	0	−7	−13	−19	−26	−32	−39	−45	−51	−58	−64		
20	30	24	17	11	4	−2	−9	−15	−22	−29	−35	−42	−48	−55	−61			
25	29	23	16	9	3	−4	−11	−17	−24	−31	−37	−44	−51	−58				
30	28	22	15	8	1	−5	−12	−19	−26	−33	−39	−46	−53					
35	28	21	14	7	0	−7	−14	−21	−27	−34	−41	−48						
40	27	20	13	6	−1	−8	−15	−22	−29	−36	−43	−50						
45	26	19	12	5	−2	−9	−16	−23	−30	−37	−44							
50	26	19	12	4	−3	−10	−17	−24	−31	−38	−45							
55	25	18	11	4	−3	−11	−18	−25	−32	−39	−46							
60	25	17	10	3	−4	−11	−19	−26	−33	−40								

Frostbite Times

Light Gray – Frostbite could occur in 30 minutes
Medium Gray – Frostbite could occur in 10 minutes
Dark Gray – Frostbite could occur in 5 minutes

Wind Speed (km/h)	Air Temperature (°C) 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 of frostbite for most people
Increasing risk of frostbite for most people in 10 to 30 minutes of exposure
High risk for most people in 5 to 10 minutes of exposure
High risk for most people in 2 to 5 minutes of exposure
High risk for most people in 2 minutes of exposure or less

FIGURE 3—Wind chill temperature 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.

because this area of the body is typically not protected. Frostbite cannot occur if the air temperature is above 0°C (32°F). Wet skin exposed to the wind will cool even faster and if the skin is wet and exposed to wind, the ambient temperature used for the WCT table should be 10°C lower than the actual ambient temperature (12). Also, the local weather may vary greatly depending on the local topography. Wind speeds are also measured at ~ 10 m and the actual exposure of someone varies with trees, buildings and the direction you are facing. Compared to lower elevations, wind speeds are generally greater at high altitudes, where there is little tree cover. **Evidence Statement**. The risk of frostbite is less than 5% when the ambient temperature is above −15°C (5°F), but increased safety surveillance of exercisers is warranted when the WCT falls below −27°C (−18°F) since, in those conditions, frostbite can occur in 30 min or less in exposed skin. *Category C*

Exercise. Physical activity is an effective countermeasure to increase finger 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 2–4 fold, finger temperature rises to 22–27°C (118). However, if conditions are windy, physical activity does not significantly alter the temperature of exposed or covered fingers. Exposure to a 5 $m \cdot s^{-1}$ (11 mph) 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 W (2.2–3.5 METs) increases nose temperatures from 4.5°C to 8.9°C, even in a 5 $m \cdot s^{-1}$ wind (12,58) and Brajkovic and Ducharme (10) found that nose skin temperature rose from 9.7°C at rest to 18.1°C during exercise.

Clothing. The same clothing principles of layering and staying dry are also used for gloves/mittens, socks, and hats. Gloves and hats can be used to regulate heat loss for each individual by adding or removing particular items based on individual subjective thermal sensations. Gloves and mittens should be donned before the hands become cold. Then as the work intensity increases and the hands become warm, gloves can be removed so that sweat does not accumulate in the fabric. Using mittens, compared to gloves, will provide greater protection from cold injuries. However, this protection must be weighed against the significant decline in manual dexterity that occurs with mitten use. Liner gloves can be used to keep moisture away from skin, allow for dexterity with protection, and add a layer of insulation. Individuals should not blow warm breath into mittens or gloves because it can cause the hands to become even colder due to the vapor from the breath adding moisture to the glove that may freeze and contribute to further cooling.

Spreading petroleum jelly or other emollients onto the skin does not lower the risk of frostbite (111,175); indeed the use of these products may increase the relative risk of frostbite on the head (110). Using white petroleum jelly may increase risk by giving a false sense of security because subjects perceive the skin to be warmer, compared to using no petroleum jelly, when the face is exposed to the cold (111). These products should not be used in freezing weather. Straps on gloves and other equipment should not be pulled too tight and shoelaces should not be tied too tightly. Backpack straps may decrease blood flow to the arms and hands, so dropping the load every few hours may be needed to allow increased circulation. Buddy checks can be implemented at this time.

NONFREEZING COLD INJURIES

The most common nonfreezing cold injuries (NFCI) are trenchfoot and chilblains. Trenchfoot typically occurs when tissues are exposed to temperatures between 0 and 15°C (32–60°F) for prolonged periods of time (75,174), whereas chilblains, a more superficial injury, can occur after just a few hours of exposure to bare skin (75). These injuries may occur due to actual immersion or by the creation of a damp environment inside boots caused by sweat soaked socks. Diagnosing NFCI involves observation of clinical symptoms over time as different, distinct stages emerge days to months after the initial injury (174).

Trenchfoot initially appears as a swollen, edematous foot with a feeling of numbness. The initial color is red but soon becomes pale and cyanotic if the injury is more severe. Peripheral pulses are hard to detect. Trenchfoot is accompanied by aches, increased pain sensitivity, and infections (75). The exposure time needed to develop trenchfoot is quite variable, with estimates ranging from > 12 h to 3–4 d in cold-wet environments (74,174). Most commonly, trenchfoot develops when wet socks and shoes are worn continuously over many days. The likelihood of trenchfoot in most sporting activities is low, except in winter hiking, camping, and expeditions.

Chilblain (also known as pernio or kibe) is a superficial cold injury typically occurring after 1–5 h in cold wet conditions (75), at temperatures below 16°C (60°F). Small erythematous papules appear on the skin, most often on the dorsal surface of the fingers, but the ears, face and exposed shins are also common (75). The lesions are swollen, tender, itchy and painful. Upon rewarming, the skin becomes inflamed, red and hot to the touch, swollen, with an itching or burning sensation that may continue for several hours after exposure. There are no lasting effects from chilblain.

Prevention of trenchfoot can be achieved by encouraging individuals to remain active and increase blood flow to the feet and keeping feet dry by continually changing socks. Changing socks 2–3 times throughout the day is mandatory in cold-wet environments during long-term exposure. Prophylactic treatment with antiperspirants containing aluminum hydroxide may also decrease sweating in the foot. Vapor barrier boots (some hiking boots, ski boots) and liners do not allow sweat from the foot to evaporate and sock changing is important. These boots and liners should be taken off each day, wiped out, and allowed to dry (75,174). If regular boots are worn, these

boots need time to dry to avoid getting moisture in the insulation.

COLD-RELATED INJURIES

Cold urticaria. Cold urticaria is a disorder characterized by the rapid onset of itching, redness, and swelling of the skin within minutes after exposure to a cold stimulus (75). It is probably the most common form of urticaria (hives). In extreme cases, anaphylactic shock may occur. This condition can begin at any age, affects both men and women, and is most prevalent in young adults (18–25 yr old). There are two forms of the disorder: essential (acquired) cold urticaria, and familial (hereditary) cold urticaria (75). The symptoms of the acquired form become obvious in two to five minutes after exposure to the triggering substance or situation, while it takes 24 to 48 h for symptoms of familial cold urticaria to appear. Also, symptoms tend to last longer with the familial form, typically about 24 h although they may remain for as long as 48 h. With the acquired form, symptoms tend to last for one to two hours. Diagnosis of cold urticaria is made by placing an ice cube or ice water on the skin. Management of cold urticaria occurs through patient education, avoiding cold exposure, and giving patients epinephrine pens.

Cold-induced bronchoconstriction. Exercise-induced bronchoconstriction (EIB) is defined as a transient narrowing of the airways that is caused by exercise (46,187) and clinically is demonstrated by a > 10% decrease in the forced expiratory volume in 1 s (FEV_1). EIB has an incidence rate of ~ 4–20% in the general population and 11–50% in elite athletes (46,154). In people with asthma, exercise causes EIB in ~ 80% of these individuals (127). Cold exposure has been implicated as a trigger for bronchoconstriction and asthma-like symptoms. Cold-weather athletes have an increased prevalence of EIB (23% of Olympic winter athletes) with cross-country skiers reportedly having an incidence rate of 33–50% (107,191). Additionally, women are reported to have higher rates of EIB than men (187,191). Two mechanisms have been postulated for EIB. One theory (the osmotic theory) suggests that airway drying caused by hyperventilation causes surface airway cells to become hyperosmotic and thus draw fluid from adjoining cells. This leads to a cascade of vasoconstrictor mediators to be secreted (3,46). The second theory suggests that cooling of airways (exacerbated by cold air and higher ventilations) and subsequent rewarming causes high blood flow, engorgement of blood vessels, and edema formation in the airway vasculature leading to airway obstruction (46,156). Evans et al. (46) tested this hypothesis and found that cold air per se did not lead to EIB, but that dry air associated with cold exposure is the likely cause of EIB, suggesting hyperosmolality as a trigger for airway narrowing. Thus the use of bottled dry air during eucapnic voluntary hyperventilation is the recommended test for identifying EIB in athletes (153). Other studies also report that facial or torso cooling alone can cause FEV_1 to be lower in people with asthma and normal controls (126,197). Thus EIB caused by cold exposure is likely caused by a combination of breathing dry air along with a reflex response due to skin or facial cooling, leading to high amounts of inflammation, especially in athletes who have high ventilation rates (95,169). Persons who experience EIB when breathing cold air during heavy exercise exhibit a reduced FEV_1 (79,156) which can limit maximal ventilation, thus maximal performance. Lastly, even healthy persons can experience an increase in respiratory passage secretions and decreased mucociliary clearance when breathing very cold air during exercise, and any associated airway congestion may impair pulmonary mechanics and ventilation during exercise, also impacting on performance (59). One possible countermeasure for decreasing the occurrence of EIB in cold-weather is to use a mouth-borne heat and moisture exchanger (41,59) or even a scarf, although the increased resistance when ventilation rates are high may preclude the use of a moisture exchanger for most competitive athletes.

EIB is more prevalent in indoor ice rink athletes compared to warm-weather athletes (119,154,155,191). Data from several investigators implicate air quality in the rink to the higher incidence (113,152). Ice rink resurfacing machines produce high levels of carbon monoxide, nitrogen dioxide, and ultrafine and fine particulate matter with observed levels as much as 20-times higher than the outside air (152). Particulate matter increases allergic sensitization and airway hyperresponsiveness (35) and exercise increases deposition of ultrafine particulate matter (29), which is related to the resting FEV_1 (104). Thus indoor ice rink athletes (figure skaters, ice-hockey players, speedskaters) and their health-care providers need to be aware that exercise in this environment may be a causative factor for EIB. **Evidence Statement**. Winter athletes, especially those exercising at high intensities and ventilation rates and in indoor ice rinks, have a higher incidence of EIB than the general population. Breathing dry air and skin/facial cooling act in synergy to trigger exercise-induced bronchospasm during winter activities. Indoor pollutants are also a trigger for EIB. *Category C*

Mortality/morbidity in winter. Mortality rates are higher in winter (45,98) compared to summer months; however hypothermia only accounts for a very minor percentage of these excess deaths. Instead, there are significant increases in death due to ischemic heart disease, stroke, and respiratory disease (45,84). Mortality increases to a greater extent in regions with relatively warm winters that have cold snaps and in people who are less active outdoors.

Cardiac. 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 (36,43,85). For example, mean arterial pressure increases by ~ 17 mm Hg (18%) and rate pressure product (systolic pressure × heart rate) increases by 10% (15). Facial cooling by wind, alone, lowers the heart rate

by ~ 10 bpm during low intensity exercise (< 35%$\dot{V}O_{2max}$) but also causes mean arterial blood pressure and rate pressure product to rise, secondary to an increase in peripheral vasoconstriction and systemic vascular resistance (108). Therefore whole-body and facial cooling can theoretically lower the threshold for the onset of angina during aerobic exercise and many studies support this view (15,43,51,131,151).

The type and intensity of exercise-cold stress also modifies the risk for the cardiac patient. Activities that involve the upper body or increase metabolism potentially increase risk. Snow shoveling has an isometric component which raises systolic blood pressures above that observed with arm ergometry alone and shoveling has been shown to raise the heart rate to 97% of maximal heart rate and systolic blood pressure to increase to 200 mm Hg (50). The data are limited on how cold exposure affects these responses. Dougherty et al. (37) found that mean arterial pressure was higher during static-dynamic shoveling in the cold (−8°C vs. 27°C), but there were no adverse changes to the electrocardiogram. Other studies suggest that patients with coronary artery disease (CAD) self-select exercise intensities below their angina threshold during snow shoveling (164). Walking in snow (either packed or soft) significantly increases energy requirements (139) and increases myocardial oxygen demands, so patients with CAD may have to slow their walking pace. Swimming in water below 25°C (77°F) can be a threat to patients with CAD because they may not be able to recognize angina symptoms and therefore may place themselves at greater risk (117). Twenty five percent of patients reported angina while swimming in 25°C water and 13% while swimming in 18°C water, but ST-segment depression was observed in 75% of the patients tested (117). **Evidence Statement**. Patients with CAD must use caution when exercising/working in the cold and should be knowledgeable of angina symptoms. Swimming in cold water may not be a good choice because it can potentially mask symptoms of angina. *Category C*

CONCLUSIONS

Exercise is primarily pursued outside in a variety of environmental extremes, including exercising in cold air and water. Because this topic affects many different people, the ACSM presents an evidence-based review of the state of knowledge on exercising in the cold.

A summary of the evidence statements and their respective evidence category grades are presented in Table 6. Since outcomes-based (development of hypothermia, frostbite, nonfreezing cold injury) research in the cold is limited due to ethical constraints, the majority of the evidence categories are graded based on physiologic end-points. However, this collection of information still enables a recommendation to be made that will aid in preventing cold injuries during exercise.

It is the position of the American College of Sports Medicine that exercise can be safely performed in cold-weather if coaches, athletes, medical personnel, and officials follow a risk management strategy. Successful implementation of this strategy includes asking the following questions: a) how cold is it?; b) what clothing

TABLE 6. American College of Sports Medicine prevention of cold injuries during exercise Position Stand evidence statements.

Section Heading	Evidence Statement	Evidence Category
Hypothermia		
	Exercising in water and rain significantly increases the risk for developing hypothermia.	A
	Individuals with high combined values of subcutaneous fat thickness, % fat, and muscle mass can maintain core temperature better than individuals with less fat and muscle.	B
	Core temperature responses to cold exposure between average men and women are primarily attributable to differences in body composition and anthropometry.	C
	Older individuals (< 60 yr) are at an increased risk of hypothermia due to blunted physiological and behavioral responses to cold. Children are at a greater risk of hypothermia than adults due to differences in body composition and anthropometry.	B
	Hypoglycemia impairs shivering and increases the risk for hypothermia.	B
	Physical fitness and training, per se, do not improve thermoregulatory responses to cold. Physical fitness does allow someone to exercise for a longer period at a higher metabolic rate, and may contribute to maintenance of normal core temperatures.	C
Prevention strategies for hypothermia		
	Clothing insulation requirements during exercise are a function of metabolic rate and ambient temperature. Layering provides the most flexibility to adjust insulation to prevent sweating, over-heating, under-dressing, and remaining dry in wet conditions.	C
	Cold environments can increase energy expenditures and may cause fluid losses; dehydration does not impair vasoconstriction or shivering, thus dehydration does not increase susceptibility to cold injuries.	C
Prevention strategies for frostbite		
	The risk of frostbite is less than 5% when the ambient temperature is above −15°C (5°F), but increased safety surveillance of exercisers is warranted when the WCT falls below −27°C (−18°F) since, in those conditions, frostbite can occur in 30 minutes or less in exposed skin.	C
Cold-related injuries		
	Winter athletes, especially those exercising at high intensities and ventilation rates and in indoor ice rinks, have a higher incidence of EIB than the general population. Breathing dry air and skin/facial cooling act in synergy to trigger exercise-induced bronchospasm during winter activities. Indoor pollutants are also a trigger for EIB.	C
	Patients with CAD must use caution when exercising/working in the cold and should be knowledgeable of angina symptoms. Swimming in cold water may not be a good choice because it can potentially mask symptoms of angina.	C

protection is available?, c) who is at risk for a cold-weather injury?, d) what is the health condition of the exerciser?, e) what effective strategies do I have available to mitigate the cold stress and injury risk?, and f) is there a contingency plan in place to deal with changing conditions? Training in cold weather is very important as athletes and coaches can learn strategies to aid in making good decisions. Training for shorter durations and near definitive care and rewarming facilities will aid athletes when the weather is worse than normal. Cold environmental conditions, in most cases, should not be a limiting factor for successfully exercising in athletic competitions, recreational pursuits, leisure activities, and occupational work.

This pronouncement was reviewed for the American College of Sports Medicine by the Pronouncements Committee and by Ira Jacobs, Ph.D., FACSM; Joel B. Mitchell, Ph.D., FACSM; Timothy D. Noakes, M.D., FACSM; Kent B. Pandolf, Ph.D., FACSM; Kenneth W. Rundell, Ph.D., FACSM; and Susan M. Shirreffs, Ph.D., FACSM.

REFERENCES

1. Adams, T., and R. E. Smith. Effect of chronic local cold exposure on finger temperature responses. *J. Appl. Physiol.* 17:317–322, 1962.
2. Ainsworth, B. E., W. L. Haskell, A. S. Leon, et al. Compendium of physical activities: classification of energy costs of human physical activities. *Med. Sci. Sports Exerc.* 25:71–80, 1993.
3. Anderson, S. D., and E. Daviskas. Pathophysiology of exercise-induced asthma: the role of respiratory water loss. In: *Allergic and Respiratory Disease in Sports Medicine*, J. M. Weiler (Ed.). New York, NY: Marcel Dekker, pp. 87–114, 1997.
4. Astrup, A. Thermogenesis in human brown adipose tissue and skeletal muscle induced by sympathomimetic stimulation. *Acta Endocrinol. Suppl. (Copenh.)* 278:1–32, 1986.
5. Banderet, L. E., and B. Shukitt-Hale. Cognitive performance, mood, and neurological status at high terrestrial elevations. In: *Medical Aspects of Harsh Environments*, K. B. Pandolf and R. E. Burr (Eds.). Falls Church, VA: Office of the Surgeon General, United States Army, pp. 729–763, 2002.
6. Bass, D. E. Metabolic and energy balances of men in a cold environment. In: *Cold Injury*, S. M. Horvath (Ed.). Montpelier, VT: Capital City Press, pp. 317–338, 1958.
7. Belding, H. S. Protection against dry cold. In: *Physiology of Heat Regulation and the Science of Clothing*, L. H. Newburgh (Ed.). Philadelphia: W. B. Saunders, pp. 351–366, 1949.
8. Bell, D. G., P. Tikuisis, and I. Jacobs. Relative intensity of muscular contraction during shivering. *J. Appl. Physiol.* 72:2336–2342, 1992.
9. Bittel, J. H. M., C. Nonott-Varly, G. H. Livecchi-Gonnot, G. Savourey, and A. M. Hanniquet. Physical fitness and thermoregulatory reactions in a cold environment in men. *J. Appl. Physiol.* 65:1984–1989, 1988.
10. Blatteis, C. M., and L. O. Lutherer. Effect of altitude exposure on thermoregulatory response of man to cold. *J. Appl. Physiol.* 41:848–858, 1976.
11. Block, J. A., and W. Sequeira. Raynaud's phenomenon. *Lancet* 357:2042–2048, 2001.
12. Brajkovic, D., and M. B. Ducharme. Facial cold-induced vasodilatation and skin temperature during exposure to cold wind. *Eur. J. Appl. Physiol.* 96:711–721, 2006.
13. Brajkovic, D., and M. B. Ducharme. Finger dexterity, skin temperature, and blood flow during auxiliary heating in the cold. *J. Appl. Physiol.* 95:758–770, 2003.
14. Brajkovic, D., M. B. Ducharme, and J. Frim. Relationship between body heat content and finger temperature during cold exposure. *J. Appl. Physiol.* 90:2445–2452, 2001.
15. Brown, C. F., and N. B. Oldridge. Exercise-induced angina in the cold. *Med. Sci. Sports Exerc.* 17:607–612, 1985.
16. Buskirk, E. R., R. H. Thompson, and G. D. Whedon. Metabolic response to cold air in men and women in relation to total body fat content. *J. Appl. Physiol.* 18:603–612, 1963.
17. Candler, W. H., and H. Ivey. Cold weather injuries among U. S. soldiers in Alaska: a five-year review. *Mil. Med.* 162:788–791, 1997.
18. Cannon, B., and J. Nedergaard. Brown adipose tissue: function and physiological significance. *Physiol. Rev.* 84:277–359, 2004.
19. Cannon, P., and W. R. Keatinge. The metabolic rate and heat loss of fat and thin men in heat balance in cold and warm water. *J. Physiol.* 154:329–344, 1960.
20. Castellani, J. W., A. J. Young, D. W. DeGroot, et al. Thermoregulation during cold exposure after several days of exhaustive exercise. *J. Appl. Physiol.* 90:939–946, 2001.
21. Castellani, J. W., A. J. Young, J. E. Kain, A. Rouse, and M. N. Sawka. Thermoregulation during cold exposure: effects of prior exercise. *J. Appl. Physiol.* 87:247–252, 1999.
22. Charkoudian, N., D. P. Stephens, K. C. Pirkle, W. A. Kosiba, and J. M. Johnson. Influence of female reproductive hormones on local thermal control of skin blood flow. *J. Appl. Physiol.* 87:1719–1723, 1999.
23. Cheuvront, S. N., R. Carter, J. W. Castellani, and M. N. Sawka. Hypohydration impairs endurance exercise performance in temperate but not cold air. *J. Appl. Physiol.* 99:1972–1976, 2005.
24. Cleophas, T. J. M., J. F. M. Fennis, and A. Van't Laar. Finger temperature after a finger-cooling test: influence of air temperature and smoking. *J. Appl. Physiol.* 52:1167–1171, 1982.
25. Daanen, H. A. M. Finger cold-induced vasodilation. *Eur. J. Appl. Physiol.* 89:411–426, 2003.
26. Daanen, H. A. M., and M. B. Ducharme. Finger cold-induced vasodilation during mild hypothermia, hyperthemia and at thermoneutrality. *Aviat. Space Environ. Med.* 70:1206–1210, 1999.
27. Daanen, H. A. M., F. J. G. Van de Linde, T. T. Romet, and M. B. Ducharme. The effect of body temperature on the hunting response of the middle finger skin temperature. *Eur. J. Appl. Physiol.* 76:538–543, 1997.
28. Daanen, H. A. M., and H. J. A. Van Ruiten. Cold-induced peripheral vasodilation at high altitudes - a field study. *High Alt. Med. Biol.* 1:323–329, 2000.
29. Daigle, C. C., D. C. Chalupa, F. R. Gibb, et al. Ultrafine particle deposition in humans during rest and exercise. *Inhal. Toxicol.* 15:539–552, 2003.
30. Danielsson, U. Windchill and the risk of tissue freezing. *J. Appl. Physiol.* 81:2666–2673, 1996.
31. Danzl, D. F. Accidental hypothermia. In: *Rosen's Emergency Medicine*, J. A. Marx (Ed.). St Louis, MO: Mosby, pp. 1979–1996, 2002.
32. Danzl, D. F. Frostbite. In: *Rosen's Emergency Medicine*, J. A. Marx (Ed.). St Louis, MO: Mosby, pp. 1972–1979, 2002.
33. DeGroot, D. W., J. W. Castellani, J. O. Williams, and P. J. Amoroso. Epidemiology of U. S. Army cold weather injuries, 1980–1999. *Aviat. Space Environ. Med.* 74:564–570, 2003.
34. Department of the Army. In: *Prevention and management of cold-weather injuries*, Washington, D.C.: Department of the Army, Technical Bulletin Medicine, p. 508, 2005.
35. Dockery, D. W., C. A. Pope, X. Xu, et al. An association between air pollution and mortality in six U. S. cities. *N. Engl. J. Med.* 329:1753–1759, 1993.
36. Doubt, T. J. Physiology of exercise in the cold. *Sports Med.* 11:367–381, 1991.
37. Dougherty, S. M., L. M. Sheldahl, N. A. Wilke, S. G. Levandoski, M. D. Hoffman, and F. E. Tristani. Physiologic

responses to shoveling and thermal stress in men with cardiac disease. *Med. Sci. Sports Exerc.* 25:790–795, 1993.
38. Ducharme, M. B. and D. Brajkovic. Guidelines on the risk and time to frostbite during exposure to cold wind. In Proceedings of the RTO NATO Factors and Medicine Panel Specialist Meeting on Prevention of Cold Injuries. Amsterdam: NATO, pp. 2–1–2–9, 2005.
39. Ducharme, M. B., and P. Tikuisis. In vivo thermal conductivity of the human forearm tissue. *J. Appl. Physiol.* 70:2682–2690, 1991.
40. Ducharme, M. B., W. P. VanHelder, and M. W. Radomski. Cyclic intramuscular temperature fluctuations in the human forearm during cold-water immersion. *Eur. J. Appl. Physiol.* 63:188–193, 1991.
41. Eiken, O., P. Kaiser, I. Holmer, and R. Baer. Physiological effects of a mouth-borne heat exchanger during heavy exercise in a cold environment. *Ergonomics* 32:645–653, 1989.
42. Enander, A. Perception of hand cooling during local cold air exposure at three different temperatures. *Ergonomics* 25:351–361, 1982.
43. Epstein, S. E., M. Stampfer, D. Beiser, R. E. Goldstein, and E. Braunwald. Effects of a reduction in environmental temperature on the circulatory response to exercise in man: implications concerning angina pectoris. *New Engl. J. Med.* 280:7–11, 1969.
44. Ervasti, O., K. Juopperi, P. Kettunen, et al. The occurrence of frostbite and its risk factors in young men. *Int. J. Circum. Health* 63:71–80, 2004.
45. Eurowinter Group. Cold exposure and winter mortality from ischaemic heart disease, cerebrovascular disease, respiratory disease, and all causes in warm and cold regions of Europe. *Lancet* 349:1341–1346, 1997.
46. Evans, T. M., K. W. Rundell, K. C. Beck, A. M. Levine, and J. M. Baumann. Cold air inhalation does not affect the severity of EIB after exercise or eucapnic voluntary hyperventilation. *Med. Sci. Sports Exerc.* 37:544–549, 2005.
47. Eyolfson, D. A., P. Tikuisis, X. Xu, G. Weseen, and G. G. Giesbrecht. Measurement and prediction of peak shivering intensity in humans. *Eur. J. Appl. Physiol.* 84:100–106, 2001.
48. Falk, B., O. Bar-Or, J. Smolander, and G. Frost. Response to rest and exercise in the cold: effects of age and aerobic fitness. *J. Appl. Physiol.* 76:72–78, 1994.
49. Ferretti, G., A. Veicsteinas, and D. W. Rennie. Conductive and convective heat flows of exercising humans in cold water. *J. Appl. Physiol.* 67:2473–2480, 1989.
50. Franklin, B. A., P. Hogan, K. Bonzheim, et al. Cardiac demands of heavy snow shoveling. *JAMA* 273:880–882, 1995.
51. Freedberg, A. S., E. D. Spiegl, and J. E. F. Riseman. Effect of external heat and cold on patients with angina pectoris: evidence for the existence of a reflex factor. *Am. Heart J.* 27:611–622, 1944.
52. Freund, B. J., and M. N. Sawka. Influence of cold stress on human fluid balance. In: *Nutritional Needs in Cold and in High-Altitude Environments*, B. M. Marriott and S. J. Carlson (Eds.). Washington, D.C.: National Academy Press, pp. 161–179, 1996.
53. Froese, G., and A. C. Burton. Heat losses from the human head. *J. Appl. Physiol.* 10:235–241, 1957.
54. Gagge, A. P., and R. R. Gonzalez. Mechanisms of heat exchange: biophysics and physiology. In: *Handbook of Physiology: Environmental Physiology*, M. J. Fregly and C. M. Blatteis (Eds.). Bethesda, MD: American Physiological Society, pp. 45–84, 1996.
55. Gale, E. A. M., T. Bennett, J. H. Green, and I. A. Macdonald. Hypoglycaemia, hypothermia and shivering in man. *Clin. Sci. (Colch.)* 61:463–469, 1981.
56. Galloway, S. D. R., and R. J. Maughan. The effects of substrate and fluid provision on thermoregulatory, cardiorespiratory and metabolic responses to prolonged exercise in a cold environment in man. *Exp. Physiol.* 83:419–430, 1998.
57. Galloway, S. D. R., S. A. Wootton, J. L. Murphy, and R. J. Maughan. Exogenous carbohydrate oxidation from drinks ingested during prolonged exercise in a cold environment in humans. *J. Appl. Physiol.* 91:654–660, 2001.
58. Gavhed, D., T. Mäkinen, I. Holmér, and H. Rintamäki. Face cooling by cold wind in walking subjects. *Int. J. Biometeorol.* 47:148–155, 2003.
59. Giesbrecht, G. G. The respiratory system in a cold environment. *Aviat. Space Environ. Med.* 66:890–902, 1995.
60. Gilbert, M., R. Busund, A. Skagseth, P. A. Nilsen, and J. P. Solbo. Resuscitation from accidental hypothermia of 13.7 degrees C with circulatory arrest. *Lancet* 355:375–376, 2000.
61. Glickman-Weiss, E. L., C. Cheatham, N. Caine, M. Blegen, J. Marcinkiewicz, and K. D. Mittleman. The influence of gender and menstrual phase on thermosensitivity during cold water immersion. *Aviat. Space Environ. Med.* 71:715–722, 2000.
62. Glickman-Weiss, E. L., F. L. Goss, R. J. Robertson, K. F. Metz, and D. A. Cassinelli. Physiological and thermal responses of males with varying body compositions during immersion in moderately cold water. *Aviat. Space Environ. Med.* 62:1063–1067, 1991.
63. Glickman-Weiss, E. L., A. G. Nelson, C. M. Hearon, F. L. Goss, R. J. Robertson, and D. A. Cassinelli. Effects of body morphology and mass on thermal responses to cold water: revisited. *Eur. J. Appl. Physiol.* 66:299–303, 1993.
64. Glickman-Weiss, E. L., A. G. Nelson, C. M. Hearon, R. Prisby, and N. Caine. Thermal and metabolic responses of women with high fat versus low fat body composition during exposure to 5 and 27°C for 120 min. *Aviat. Space Environ. Med.* 70:284–288, 1999.
65. Golja, P., A. Kacin, M. J. Tipton, O. Eiken, and I. B. Mekjavic. Hypoxia increases the cutaneous threshold for the sensation of cold. *Eur. J. Appl. Physiol.* 92:62–68, 2004.
66. Gonzalez, R. R. Biophysical and physiological integration of proper clothing for exercise. *Exerc. Sport Sci. Rev.* 15:261–295, 1987.
67. Gonzalez, R. R., and M. N. Sawka. Biophysics of heat transfer and clothing considerations. In: *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*, K. B. Pandolf, M. N. Sawka, and R. R. Gonzalez (Eds.). Indianapolis, IN: Benchmark, pp. 45–95, 1988.
68. Gonzalez, R. R., and L. A. Blanchard. Thermoregulatory responses to cold transients: effects of menstrual cycle in resting women. *J. Appl. Physiol.* 85:543–553, 1998.
69. Graham, T. E., M. Viswanathan, J. P. V. Dijk, A. Bonen, and J. C. George. Thermal and metabolic responses to cold by men and by eumenorrheic and amenorrheic women. *J. Appl. Physiol.* 67:282–290, 1989.
70. Grisanti, J. M. Raynaud's phenomenon. *Am. Fam. Physician* 41:134–142, 1990.
71. Haman, F. Shivering in the cold: from mechanisms of fuel selection to survival. *J. Appl. Physiol.* 100:1702–1708, 2006.
72. Haman, F., F. Peronnet, G. P. Kenny, et al. Effects of carbohydrate availability on sustained shivering I. Oxidation of plasma glucose, muscle glycogen, and proteins. *J. Appl. Physiol.* 96:32–40, 2004.
73. Haman, F., F. Peronnet, G. P. Kenny, et al. Effect of cold exposure on fuel utilization in humans: plasma glucose, muscle glycogen, and lipids. *J. Appl. Physiol.* 93:77–84, 2002.
74. Hamlet, M. P. Human cold injuries. In: *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*, K. B. Pandolf, M. N. Sawka, and R. R. Gonzalez (Eds.). Indianapolis, IN: Benchmark, pp. 435–466, 1988.
75. Hamlet, M. P. Nonfreezing cold injuries. In: *Textbook of Wilderness Medicine*, P. S. Auerbach (Ed.). St. Louis, MO: Mosby, pp. 129–134, 2001.
76. Hartelink, M. L., H. Wollersheim, E. Leesman, T. de Boo, and T. Thien. A standardized finger cooling test for Raynaud's phenomenon: diagnostic value and sex differences. *Eur. Heart J.* 14:614–622, 1993.

77. Hashmi, M. A., M. Rashid, A. Haleem, S. S. Bokhari, and T. Hussain. Frostbite: epidemiology at high altitude in the Karakoram mountains. *Ann. R. Coll. Surg. Engl.* 80:91–95, 1998.
78. Hayward, M. G., and W. R. Keatinge. Roles of subcutaneous fat and thermoregulatory reflexes in determining ability to stabilize body temperature in water. *J. Physiol. (Lond.)* 320:229–251, 1981.
79. Helenius, I. J., H. O. Tikkanen, and T. Haahtela. Exercise-induced bronchospasm at low temperatures in elite runners. *Thorax* 51:628–629, 1996.
80. Hessemer, V., and K. Brück. Influence of menstrual cycle on shivering, skin blood flow, and sweating responses measured at night. *J. Appl. Physiol.* 59:1902–1910, 1985.
81. Heus, R., H. A. M. Daanen, and G. Havenith. Physiological criteria for functioning of hands in the cold. *Appl. Ergonomics* 26:5–13, 1995.
82. Holmer, I. Assessment of cold stress in terms of required clothing insulation-IREQ. *Int. J. Indust. Ergon.* 3:159–166, 1988.
83. Hong, S. K., D. W. Rennie, and Y. S. Park. Humans can acclimatize to cold: a lesson from Korean women divers. *N. I. P. S.* 2:79–82, 1987.
84. Hong, Y. C., J. H. Rha, J. T. Lee, E. H. Ha, H. J. Kwon, and H. Kim. Ischemic stroke associated with decrease in temperature. *Epidemiology* 14:473–478, 2003.
85. Horvath, S. M. Exercise in a cold environment. *Exerc. Sport Sci. Rev.* 9:221–263, 1981.
86. Iida, T. Studies concerning vascular reaction to cold (Part I), Physiological significance of vascular reactions to cold [In Japanese]. *J. Physiol. Soc. Japan* 11:73–78, 1949.
87. Institute of Medicine. *Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate*, Washington, D.C.: The National Academies Press, 2005.
88. ISO. *Evaluation of cold environments - Determination of required clothing insulation (IREQ)*, Geneva: International Organization for Standardization, 1993. TR 11079.
89. ISO. *Ergonomics of the thermal environment- Estimation of the thermal insulation and evaporative resistance of a clothing ensemble*, Geneva: International Organization for Standardization, 2005. ISO 9920.
90. Jacobs, I., T. Romet, J. Frim, and A. Hynes. Effects of endurance fitness on responses to cold water immersion. *Aviat. Space Environ. Med.* 55:715–720, 1984.
91. Jay, O., and G. Havenith. Finger skin cooling on contact with cold materials: a comparison between male and female responses during short-term exposures. *Eur. J. Appl. Physiol.* 91:373–381, 2004.
92. Johnston, C. E., M. D. White, W. Mingpu, G. K. Bristow, and G. G. Giesbrecht. Eucapnic hypoxia lowers human cold thermoregulatory response thresholds and accelerates core cooling. *J. Appl. Physiol.* 80:422–429, 1996.
93. Jones, B. H., P. B. Rock, L. S. Smith, et al. Medical complaints after a marathon run in cool weather. *Physician Sportsmed.* 13:103–110, 1985.
94. Juopperi, K., J. Hassi, O. Ervasti, A. Drebs, and S. Näyhä. Incidence of frostbite and ambient temperature in Finland, 1986–1995. *Int. J. Circum. Health* 61:352–362, 2002.
95. Karjalainen, E., A. Laitinen, M. Sue-Chu, A. Altraja, L. Bjermer, and L. Laitinen. Evidence of airway inflammation and remodeling in ski athletes with and without bronchial hyperresponsiveness to methacholine. *Am. J. Respir. Crit. Care Med.* 161:2086–2091, 2000.
96. Kasai, T., M. Hirose, T. Matsukawa, A. Takamata, and Y. Tanaka. The vasoconstriction threshold is increased in obese patients during general anaesthesia. *Acta Anaesthesiol. Scand.* 47:588–592, 2003.
97. Kaufman, W. C., and D. J. Bothe. Wind chill reconsidered, Siple revisited. *Aviat. Space Environ. Med.* 57:23–26, 1986.
98. Keatinge, W. Medical problems of cold weather. *J. R. Coll. Physicians Lond.* 20:283–287, 1986.
99. Keatinge, W. R. The effects of subcutaneous fat and of previous exposure to cold on the body temperature, peripheral blood flow and metabolic rate of men in cold water. *J. Physiol. (Lond.)* 153:166–178, 1960.
100. Keatinge, W. R., and P. Cannon. Freezing-point of human skin. *Lancet* 1:11–14, 1960.
101. Kenefick, R. W., M. P. Hazzard, N. V. Mahood, and J. W. Castellani. Thirst sensations and AVP responses are attenuated when hypohydrated at rest and during exercise-cold exposure. *Med. Sci. Sports Exerc.* 36:1528–1534, 2004.
102. Kenney, W. L., and C. G. Armstrong. Reflex peripheral vasoconstriction is diminished in older men. *J. Appl. Physiol.* 80:512–515, 1996.
103. Kerr, C. G., T. A. Trappe, R. D. Starling, and S. W. Trappe. Hyperthermia during Olympic triathlon: influence of body heat storage during the swimming stage. *Med. Sci. Sports Exerc.* 30:99–104, 1998.
104. Kim, C. S., and T. C. Kang. Comparative measurement of lung deposition of inhaled fine particles in normal subjects and patients with obstructive airway disease. *Am. J. Crit. Care Med.* 155:899–905, 1997.
105. Klentrou, P., M. Cunliffe, J. Slack, et al. Temperature regulation during rest and exercise in the cold in premenarcheal and menarcheal girls. *J. Appl. Physiol.* 96:1393–1398, 2004.
106. Koljonen, V., K. Andersson, K. Mikkonen, and J. Vuola. Frostbite injuries treated in the Helsinki area from 1995 to 2002. *J. Trauma* 57:1315–1320, 2004.
107. Larsson, K., P. Ohlsen, L. Larsson, P. Malmberg, P. O. Rydstrom, and H. Ulriksen. High prevalence of asthma in cross country skiers. *BMJ* 307:1326–1329, 1993.
108. LeBlanc, J. *Man in the Cold*, Springfield, Ill: Charles C. Thomas, 1975.
109. Lee, D. T., M. M. Toner, W. D. McArdle, J. S. Vrabas, and K. B. Pandolf. Thermal and metabolic responses to cold-water immersion at knee, hip and shoulder levels. *J. Appl. Physiol.* 82:1523–1530, 1997.
110. Lehmuskallio, E. Emollients in the prevention of frostbite. *Int. J. Circumpolar Health* 59:122–130, 2000.
111. Lehmuskallio, E., H. Rintamaki, and H. Anttonen. Thermal effects of emollients on facial skin in the cold. *Acta Derm. Venereol.* 80:203–207, 2000.
112. Lennquist, H., P. Granberg, and B. Wedin. Fluid balance and physical work capacity in humans exposed to cold. *Arch. Environ. Health* 29:241–249, 1974.
113. Levy, J. I., K. Lee, Y. Yanagisawa, P. Hutchinson, and J. D. Spengler. Determinants of nitrogen dioxide concentrations in indoor ice skating rinks. *Am. J. Public Health.* 88:1781–1786, 1998.
114. Lewis, T. Observations upon the reactions of the vessels of the human skin to cold. *Heart* 15:177–208, 1930.
115. Lounsbury, D. S. and M. B. Ducharme. Self-rescue strategies during accidental cold water immersion: performance and thermal considerations. In Proceedings of the 11th International Conference on Environmental Ergonomics. Ystad, Sweden: Lund University, pp. 553–556, 2005.
116. Macdonald, I. A., T. Bennett, and R. Sainsbury. The effect of a 48 h fast on the thermoregulatory responses to graded cooling in man. *Clin. Sci.* 67:445–452, 1984.
117. Magder, S., D. Linnarsson, and L. Gullstrand. The effect of swimming on patients with ischemic heart disease. *Circulation* 63:979–986, 1981.
118. Mäkinen, T. T., D. Gavhed, I. Holmér, and H. Rintamäki. Effects of metabolic rate on thermal responses at different air velocities in −10°C. *Comp. Biochem. Physiol. Part A* 128:759–768, 2001.
119. Mannix, E. T., F. Manfredi, and M. O. Farber. A comparison of two challenge tests for identifying exercise-induced bronchospasm in figure skaters. *Chest* 115:649–653, 1999.

120. Mansell, P. I., and I. A. Macdonald. Effects of underfeeding and of starvation on thermoregulatory responses to cooling in women. *Clin. Sci.* 77:245–252, 1989.
121. Martineau, L., and I. Jacobs. Muscle glycogen availability and temperature regulation in humans. *J. Appl. Physiol.* 66:72–78, 1989.
122. Mathew, L., S. S. Purkayastha, W. Selvamurthy, and M. S. Malhotra. Cold-induced vasodilation and peripheral blood flow under local cold stress in man at altitude. *Aviat. Space Environ. Med.* 48:497–500, 1977.
123. Maughan, R. J. Thermoregulation in marathon competition at low ambient temperature. *Int. J. Sports Med.* 6:15–19, 1985.
124. McArdle, W. D., J. R. Magel, T. J. Gergley, R. J. Spina, and M. M. Toner. Thermal adjustment to cold-water exposure in resting men and women. *J. Appl. Physiol.* 56:1565–1571, 1984.
125. McArdle, W. D., J. R. Magel, R. J. Spina, T. J. Gergley, and M. M. Toner. Thermal adjustment to cold-water exposure in exercising men and women. *J. Appl. Physiol.* 56:1572–1577, 1984.
126. McDonald, J. S., J. Nelson, K. A. Lenner, M. L. McLane, and E. R. McFadden. Effects of the combination of skin cooling and hyperpnea of frigid air in asthmatic and normal subjects. *J. Appl. Physiol.* 82:453–459, 1997.
127. Mellion, M. B., and R. H. Kobayashi. Exercise-induced asthma. *Am. Fam. Physician* 45:2671–2677, 1992.
128. Mills, W. J. Clinical aspects of freezing cold injury. In: *Textbooks of Military Medicine: Medical Aspects of Harsh Environments, Volume 1*, K. B. Pandolf and R. E. Burr (Eds.). Falls Church, VA: Office of the Surgeon General, U. S. Army, pp. 429–466, 2002.
129. Molnar, G. W., A. L. Hughes, O. Wilson, and R. F. Goldman. Effect of skin wetting on finger cooling and freezing. *J. Appl. Physiol.* 35:205–207, 1973.
130. National Weather Service. Windchill Temperature Index. Office of Climate, Water, and Weather Services, Washington, D.C., National Oceanic and Atmospheric Administration 2001.
131. Neill, W. A., D. A. Duncan, F. Kloster, and D. J. Mahler. Response of coronary circulation to cutaneous cold. *Am. J. Med.* 56:471–476, 1974.
132. Noakes, T. D. Exercise and the cold. *Ergonomics* 43:1461–1479, 2000.
133. O'Brien, C. Reproducibility of the cold-induced vasodilation response in the human finger. *J. Appl. Physiol.* 90:254–259, 2005.
134. O'Brien, C., and S. J. Montain. Hypohydration effect on finger skin temperature and blood flow during cold-water finger immersion. *J. Appl. Physiol.* 94:598–603, 2003.
135. O'Brien, C., A. J. Young, D. T. Lee, A. Shitzer, M. N. Sawka, and K. B. Pandolf. Role of core temperature as a stimulus for cold acclimation during repeated immersion in 20°C water. *J. Appl. Physiol.* 89:242–250, 2000.
136. O'Brien, C., A. J. Young, and M. N. Sawka. Hypohydration and thermoregulation in cold air. *J. Appl. Physiol.* 84:185–189, 1998.
137. Ohnaka, T., Y. Tochihara, K. Tsuzuki, Y. Nagai, T. Tokuda, and Y. Kawashima. Preferred temperature of the elderly after cold and heat exposures determined by individual self-selection of air temperature. *J. Therm. Biol.* 18:349–353, 1993.
138. Osczevski, R. J., and M. Bluestein. The new wind chill equivalent temperature chart. *Bulletin Amer. Meteor. Soc.* 86:1453–1458, 2005.
139. Pandolf, K. B., M. F. Haisman, and R. F. Goldman. Metabolic energy expenditure and terrain coefficients for walking on snow. *Ergonomics* 19:683–690, 1976.
140. Passias, T. C., G. S. Meneilly, and I. B. Mekjavic. Effect of hypoglycemia on thermoregulatory responses. *J. Appl. Physiol.* 80:1021–1032, 1996.
141. Pitsiladis, Y. P., and R. J. Maughan. The effects of exercise and diet manipulation on the capacity to perform prolonged exercise in the heat and in the cold in trained humans. *J. Physiol. (Lond.)* 517:919–930, 1999.
142. Pollock, F. E., L. A. Koman, B. P. Smith, M. Holden, G. B. Russell, and G. G. Poehling. Measurement of hand microvascular blood flow with isolated cold stress testing and laser doppler fluxmetry. *J. Hand Surg.* 18A:143–150, 1993.
143. Pozos, R. S., and D. F. Danzl. Human physiological responses to cold stress and hypothermia. In: *Textbooks of Military Medicine: Medical Aspects of Harsh Environments, Volume 1*, K. B. Pandolf and R. E. Burr (Eds.). Falls Church, VA: Office of the Surgeon General, U. S. Army, pp. 351–382, 2002.
144. Provins, K. A., and R. Morton. Tactile discrimination and skin temperature. *J. Appl. Physiol.* 15:155–160, 1960.
145. Pugh, L. G. C. E. Deaths from exposure on Four Inns walking competition, March 14–15, 1964. *Lancet* 1:1210–1212, 1964.
146. Pugh, L. G. C. E. Cold stress and muscular exercise, with special reference to accidential hypothermia. *Br. Med. J.* 2:333–337, 1967.
147. Reading, J. E., D. E. Roberts, and W. K. Prusaczyk. Gender differences in finger temperatures during cold air exposure. San Diego, CA: Naval Health Reseach Center, Technical Report 97–37, 1997.
148. Rennie, D. W. Tissue heat transfer in water: lessons from the Korean divers. *Med. Sci. Sports Exerc.* 20(Supplement): S177–S184, 1988.
149. Roberts, W. O. A 12-yr profile of medical injury and illness for the Twin Cities Marathon. *Med. Sci. Sports Exerc.* 32:1549–1555, 2000.
150. Rogers, T. A., and J. A. Setliff. Value of fluid and electrolyte supplements in subarctic survival situations. *J. Appl. Physiol.* 19:580–582, 1964.
151. Rosengren, A., B. Wennerblom, T. Bjuro, L. Wilhelmsen, and B. Bake. Effects of cold on ST amplitudes and blood pressure during exercise in angina pectoris. *Eur. Heart J.* 9:1074–1080, 1988.
152. Rundell, K. W. High levels of airborne ultrafine and fine particulate matter in indoor ice arenas. *Inhal. Toxicol.* 15:237–250, 2002.
153. Rundell, K. W., S. D. Anderson, B. A. Spiering, and D. A. Judelson. Field exercise vs laboratory eucapnic voluntary hyperventilation to identify airway hyperresponsiveness in elite cold weather athletes. *Chest* 125:909–915, 2004.
154. Rundell, K. W., and D. M. Jenkinson. Exercise-induced bronchospasm in the elite athlete. *Sports Med.* 32:583–600, 2002.
155. Rundell, K. W., B. A. Spiering, T. M. Evans, and J. M. Baumann. Baseline lung function, exercise-induced bronchconstriction, and asthma-like symptoms in elite women ice hockey players. *Med. Sci. Sports Exerc.* 36:405–410, 2004.
156. Rundell, K. W., B. A. Spiering, D. A. Judelson, and M. H. Wilson. Bronchoconstriction during cross-country skiing: is there really a refractory period? Med. *Sci. Sports Exerc.* 35:18–26, 2003.
157. Sagawa, S., K. Shiraki, M. K. Yousef, and N. Konda. Water temperature and intensity of exercise in maintenance of thermal equilibrium. *J. Appl. Physiol.* 65:2413–2419, 1988.
158. Sallis, R., and M. C. Chassay. Recognizing and treating common cold-induced injury in outdoor sports. *Med. Sci. Sports Exerc.* 31:1367–1373, 1999.
159. Savage, M. V., and G. L. Brengelmann. Control of skin blood flow in the neutral zone of human body temperature regulation. *J. Appl. Physiol.* 80:1249–1257, 1996.
160. Savourey, G., and J. Bittel. Thermoregulatory changes in the cold induced by physical training in humans. *Eur. J. Appl. Physiol.* 78:379–384, 1998.
161. Savourey, G., L. Clerc, A. L. Vallerand, G. Leftheriotis, H. Mehier, and J. H. M. Bittel. Blood flow and muscle bioenergetics by 31p-NMR after local cold acclimation. *Eur. J. Appl. Physiol.* 64:127–133, 1992.
162. Sawka, M. N., and A. J. Young. Physical exercise in hot and cold climates. In: *Exercise and Sport Science*, W. E. Garrett and D. T. Kirkendall (Eds.). Philadelphia: Lippincott Williams & Wilkins, pp. 385–400, 2000.

163. Scott, C. G., M. B. Ducharme, F. Haman, and G. P. Kenny. Warming by immersion or exercise affects initial cooling rate during subsequent cold water immersion. *Aviat. Space Environ. Med.* 75:956–963, 2004.
164. Sheldahl, L. M., N. A. Wilke, S. M. Dougherty, and F. E. Tristani. Snow blowing and shoveling in normal and asymptomatic coronary artery diseased men. *Int. J. Cardiol.* 43:233–238, 1994.
165. Sloan, R. E. G., and W. R. Keatinge. Cooling rates of young people swimming in cold water. *J. Appl. Physiol.* 35:371–375, 1973.
166. Smolander, J. Effect of cold exposure on older humans. *Int. J. Sports Med.* 23:86–92, 2002.
167. Smolander, J., O. Bar-Or, O. Korhonen, and J. Ilmarinen. Thermoregulation during rest and exercise in the cold in pre- and early pubescent boys and in young men. *J. Appl. Physiol.* 72:1589–1594, 1992.
168. Stocks, J. M., N. A. Taylor, M. J. Tipton, and J. E. Greenleaf. Human physiological responses to cold exposure. *Aviat. Space Environ. Med.* 75:444–457, 2004.
169. Sue-Chu, M., L. Larsson, T. Moen, S. I. Rennard, and L. Bjermer. Bronchoscopy and bronchoalveolar lavage findings in cross-country skiers with and without "ski asthma". *Eur. Respir. J.* 13:626–632, 1999.
170. Szlyk, P. C., I. V. Sils, R. P. Francesconi, and R. W. Hubbard. Patterns of human drinking: effects of exercise, water temperature, and food consumption. *Aviat. Space Environ. Med.* 61:43–48, 1990.
171. Takeoka, A., Y. Yanagidaira, A. Sakai, et al. Effects of high altitudes on finger cooling test in Japanese and Tibetans at Quighai plateau. *Int. J. Biometeor.* 37:27–31, 1993.
172. Taylor, M. S. Cold weather injuries during peacetime military training. *Mil. Med.* 157:602–604, 1992.
173. Taylor, N. A., N. K. Allsopp, and D. G. Parkes. Preferred room temperature of young vs. aged males: the influence of thermal sensation, thermal comfort, and affect. *J. Gerontol. A Biol. Sci. Med. Sci.* 50:216–221, 1995.
174. Thomas, J. R., and E. H. N. Oakley. Nonfreezing cold injury. In: *Textbooks of Military Medicine: Medical Aspects of Harsh Environments, Volume 1*, K. B. Pandolf and R. E. Burr (Eds.). Falls Church, VA: Office of the Surgeon General, U. S. Army, pp. 467–490, 2002.
175. Thorleifsson, A., and H. C. Wulf. Emollients and the response of facial skin to a cold environment. *Br. J. Dermatol.* 148:1149–1152, 2003.
176. Tikuisis, P., I. Jacobs, D. Moroz, A. L. Vallerand, and L. Martineau. Comparison of thermoregulatory responses between men and women immersed in cold water. *J. Appl. Physiol.* 89:1403–1411, 2000.
177. Tipton, M. J., and F. S. C. Golden. Immersion in cold water: effects on performance and safety. In: *Oxford Textbook of Sports Medicine*, M. Harries, C. Williams, W. D. Stanish, and L. J. Micheli (Eds.). Oxford: Oxford University Press, pp. 241–254, 1998.
178. Toner, M. M., and W. D. McArdle. Human thermoregulatory responses to acute cold stress with special reference to water immersion. In: *Handbook of Physiology: Environmental Physiology*, M. J. Fregley and C. M. Blatteis (Eds.). Bethesda, MD: American Physiological Society, pp. 379–418, 1996.
179. Toner, M. M., M. N. Sawka, M. E. Foley, and K. B. Pandolf. Effects of body mass and morphology on thermal responses in water. *J. Appl. Physiol.* 60:521–525, 1986.
180. Toner, M. M., M. N. Sawka, W. L. Holder, and K. B. Pandolf. Comparison of thermal responses between rest and leg exercise in water. *J. Appl. Physiol.* 59:248–253, 1985.
181. Toner, M. M., M. N. Sawka, and K. B. Pandolf. Thermal responses during arm and leg and combined arm-leg exercise in water. *J. Appl. Physiol.* 56:1355–1360, 1984.
182. Toussaint, H. M., L. Bruinink, R. Coster, et al. Effect of a triathlon wet suit on drag during swimming. *Med. Sci. Sports Exerc.* 21:325–328, 1989.
183. Trappe, T. A., D. L. Pease, S. W. Trappe, J. P. Troup, and E. R. Burke. Physiological responses to swimming while wearing a wet suit. *Int. J. Sports Med.* 17:111–114, 1996.
184. Trappe, T. A., R. D. Starling, A. C. Jozsi, et al. Thermal responses to swimming in three water temperatures: influence of a wet suit. *Med. Sci. Sports Exerc.* 27:1014–1021, 1995.
185. Veicsteinas, A., G. Ferretti, and D. W. Rennie. Superficial shell insulation in resting and exercising men in cold water. *J. Appl. Physiol.* 52:1557–1564, 1982.
186. Wallingford, R., M. B. Ducharme, and E. Pommier. Limiting factors in cold water survival swimming distance. *Eur. J. Appl. Physiol.* 82:22, 2000.
187. Weiler, J. M., and E. J. Ryan. Asthma in United States olympic athletes who participated in the 1998 olympic winter games. *J. Allergy Clin. Immunol.* 106:267–271, 2000.
188. Weller, A. S., C. E. Millard, M. A. Stroud, P. L. Greenhaff, and I. A. Macdonald. Physiological responses to cold stress during prolonged intermittent low- and high-intensity walking. *Am. J. Physiol.* 272:R2025–R2033, 1997.
189. Weller, A. S., C. E. Millard, M. A. Stroud, P. L. Greenhaff, and I. A. Macdonald. Physiological responses to a cold, wet, and windy environment during prolonged intermittent walking. *Am. J. Physiol.* 272:R226–R233, 1997.
190. Wigley, F. M. Raynaud's phenomenon. *N. Engl. J. Med.* 347:1001–1008, 2002.
191. Wilber, R. L., K. W. Rundell, L. Szmedra, D. M. Jenkinson, J. Im, and S. E. Drake. Incidence of exercise-induced bronchospasm in Olympic winter sport athletes. *Med. Sci. Sports Exerc.* 32:732–737, 2000.
192. Wilson, O., R. F. Goldman, and G. W. Molnar. Freezing temperature of finger skin. *J. Appl. Physiol.* 41:551–558, 1976.
193. Young, A. J. Homeostatic responses to prolonged cold exposure: human cold acclimatization. In: *Handbook of Physiology: Environmental Physiology*, M. J. Fregly and C. M. Blatteis (Eds.). Bethesda, MD: American Physiological Society, pp. 419–438, 1996.
194. Young, A. J., and D. T. Lee. Aging and human cold tolerance. *Exp. Aging Res.* 23:45–67, 1997.
195. Young, A. J., M. N. Sawka, L. Levine, et al. Metabolic and thermal adaptations from endurance training in hot or cold water. *J. Appl. Physiol.* 78:793–801, 1995.
196. Young, A. J., M. N. Sawka, P. D. Neufer, S. R. Muza, E. W. Askew, and K. B. Pandolf. Thermoregulation during cold water immersion is unimpaired by low muscle glycogen levels. *J. Appl. Physiol.* 66:1809–1816, 1989.
197. Zeitoun, M., B. Wilk, A. Matsuzaka, B. N. Knöpfli, B. A. Wilson, and O. Bar-Or. Facial cooling enhances exercise-induced bronchoconstriction in asthmatic children. *Med. Sci. Sports Exerc.* 36:767–771, 2004.