

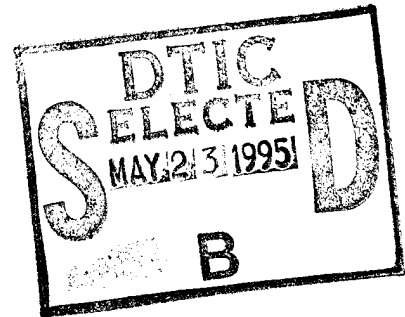
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ENVIRONMENTAL CONSIDERATIONS FOR EXERCISE

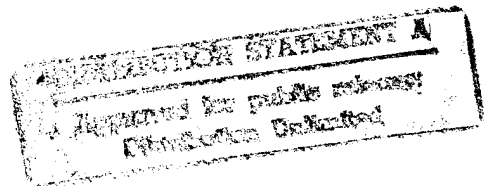
BY

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May 1995



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EXECUTIVE SUMMARY

Exercise in either hot or cold climates, at high altitude or in air polluted environments can reduce exercise tolerance and physical performance. This report discusses environmental considerations for exercise. It reviews the general physiological responses at each environmental extreme and presents special considerations for exercise in hot and cold climates, at high altitude and air polluted areas. It also discusses, albeit briefly, medical management of persons who develop heat, cold, or high-altitude related illnesses. The report concludes with a discussion of the interaction of the individual environmental extremes on exercise and physical performance.

INTRODUCTION

Exercise testing and physical training are integral components in the physical rehabilitation process. Regular physical exercise is also an essential component in any preventive medicine program. The stress imposed by the exercise intensity, however, is only one consideration in the exercise testing and prescription process. An equally important stress to consider is that imposed by the environment (i.e. heat, cold, altitude and air quality). The environment can alter the physiological responses of the muscular, cardiovascular and thermoregulatory systems, and, either singly or in combination with exercise, induce potentially hazardous health conditions.

The purpose of this report is to provide the reader with information on the physiological responses which occur when humans are exposed to heat, cold, altitude and air pollution. The chapter focuses on the impact each environmental extreme has on the physiological responses to exercise, exercise performance and exercise prescription. In each section, albeit briefly, special considerations are discussed which can modify the physiological responses to the environment, as well as effective ways to prevent, recognize and treat illnesses arising from environmental exposure.

TEMPERATURE REGULATION AND ENERGY BALANCE

Humans, like other homeothermic animals, must maintain body temperature within a relatively narrow range in order to function optimally and/or survive. Deviations in body temperature $\pm 4^{\circ}\text{C}$ from 37°C (normal resting core temperature) are known to reduce physical and cognitive performance, and deviations of $+6^{\circ}\text{C}$ or -12°C are usually lethal (1).

Body temperature is physiologically regulated by integrating both thermal and non-thermal information within the anterior and pre-optic areas of the hypothalamus and sending the appropriate efferent signals to alter heat loss and gain with the environment. Thermal signals include afferent input from temperature sensitive nerve endings located in the brain, body core, and skin. Non-thermal signals include afferent information from the cardio-pulmonary and arterial baroreceptors, and osmoreceptors. Passive heat transfer properties also affect heat movement within the

body. These passive properties include body mass, body mass/surface area ratio, and body composition.

Despite its complex nature, body temperature regulation can be viewed simply as the process of balancing heat production with heat loss. This is accomplished by physiological processes which control the rate of heat production, the transfer of metabolic heat from the body core to the skin surface and the transfer of heat from the skin surface to the surrounding environment. Deep body (core) temperature remains stable whenever body heat production equals body heat loss. Core temperature increases when heat production exceeds heat loss and falls when heat losses exceed heat production.

During exercise, metabolic substrates are broken down to provide energy for cellular metabolism. However, only 20-23% of the substrate's energy is used to accomplish work. The remaining 77-80% becomes heat that must be dissipated to prevent dangerous elevations in core temperature. Heat production, which can increase 20 times the basal rate, can be conveniently estimated from the measured oxygen uptake and the useful work that is accomplished. The magnitude of heat storage (i.e., increase in body core temperature) will depend on the magnitude of heat production, the ability to transfer the heat to the skin surface and the capacity of the environment to accept heat from the skin surface. In hot environments, the heat produced during exercise may be compounded by heat gained from the environment and further raise core temperature. In cold environments, the heat produced by exercise and/or involuntary rhythmic contractions of skeletal muscle (shivering) may be needed to attenuate reductions in body temperature.

The majority of heat generated within the body is convected from the inner core to the skin's surface via the circulation. Heat is then dissipated to the environment by three primary means: radiation, convection and/or evaporation. The relative contribution of each pathway is dependent on the environmental conditions as summarized in Figure 14-1. Any imbalance between the rate of heat dissipation and heat production will result in a change in heat storage or heat debt. To better understand the different pathways for heat loss, a brief description of each is described below:

Radiation is the heat gained or lost at the skin surface due to the arrival or release of energy in the infrared and related portions of the electromagnetic energy spectrum from or to the surrounding radiant surfaces. Radiant heat exchange is independent of ambient air temperature, depending upon the temperature gradient between the emitting and receiving surfaces.

Convection is the heat lost or gained due to the mass transfer or conduction of heat at the boundary layer of air (or water) moving past the skin's surface. In thermoneutral conditions and still air, convection accounts for 12-15% of total heat loss. However, convective heat loss increases rapidly with air movement. Water is approximately 40 times more effective in convecting heat from the body than is air. As a consequence, cold exposure combined with body surface wetting causes substantial heat loss and is more likely to result in hypothermia than cold air alone.

Evaporation is the heat lost by vaporization of water from the skin surface. Evaporation is the most effective means of heat loss in terms of absolute quantities of heat, and is the most important route of heat loss during exercise-heat stress. Evaporation is dependent upon the water vapor pressure gradient between the skin and the air, and the air velocity moving over the skin surface. It does not require a positive skin to ambient air temperature gradient. Thus, under conditions in which there is a positive water vapor pressure gradient between the skin and ambient air, secreted sweat will evaporate, removing heat from the body. In high ambient humidity conditions, the evaporative capacity is reduced and secreted sweat will drip from the skin surface without dissipating body heat.

HEAT EXPOSURE

CORE TEMPERATURE RESPONSES TO EXERCISE-HEAT STRESS

During physical exercise in ambient conditions that allow steady-state heat balance, core temperature initially increases rapidly and then increases more slowly until heat loss equals heat production (Figure 14-2). The initial increase in core

temperature is due to a lag in activating the heat dissipation mechanisms (i.e. sweating, cutaneous vasodilatation). The magnitude of increase in core temperature is proportional to the metabolic rate (e.g., the exercise intensity), and nearly independent of environmental conditions within a range of climatic conditions termed the "prescriptive zone" (2-6); (Figure 14-3). The prescriptive zone refers to a range of ambient air conditions in which there is little or no difference in steady-state exercise core temperature despite varying the ambient air conditions. During exercise in climatic conditions above the prescriptive zone, the steady-state core temperature is elevated in proportion to the climatic conditions. Therefore, steady-state body core temperature will be similar during exercise in temperate (20 to 28°C) environment, but will be elevated when performing the same exercise in hot environments. Equally important, Figure 14-3 also illustrates that the upper end of the prescriptive zone is shifted to a lower ambient temperature condition with increasing exercise intensity. This means that core temperature will begin to be affected by the environmental conditions at a lower ambient temperature conditions when performing high intensity exercise than during moderate or low intensity exercise.

The core temperature increase during endurance exercise does not reflect a failure of the temperature control system. On the contrary, core temperature is finely regulated even under extremes of exercise and environmental temperature. During exercise, the core temperature increase is the result of integrated physiological responses which optimize skeletal muscle perfusion and heat transport without compromising arterial blood pressure. Under conditions in which skin blood flow is compromised (i.e. dehydration, high exercise intensity) the core temperature increase widens the temperature gradient between the body core and skin; preserving heat transport to the peripheral circulation despite lower skin blood flow.

There are climatic conditions, however, in which steady-state core temperature values cannot be attained. Whenever heat production exceeds the environment's capacity to accept heat, core temperature will progressively increase and persons are at increased risk for heat exhaustion and heat stroke. The athlete, race organizer, and trainer must evaluate the ambient weather conditions prior to beginning exercise and modify the exercise intensity, duration, as well as fluid requirements to reduce the risk of heat injury. The American College of Sports Medicine has recommended that

endurance athletic events be rescheduled when the ambient wet bulb globe temperature ($WBGT = 0.7 \cdot T_{wb} + 0.2 \cdot T_g + 0.1 \cdot T_{db}$; where T_{wb} = wet bulb temperature, T_g = black globe temperature and T_{db} = dry bulb temperature) exceeds 28°C (7).

CARDIOVASCULAR RESPONSES TO EXERCISE-STRESS

When exercising in warm or hot ambient temperatures, the cardiovascular system is challenged with simultaneously delivering sufficient muscle blood flow to support metabolism and sufficient cutaneous blood flow to support heat loss from the body while still maintaining blood pressure. Because the blood flow demand to these vascular beds can exceed the capacity of the cardiovascular system, reductions in blood flow to the skin and/or muscle may be necessary to preserve cardiovascular stability.

Several adjustments occur during exercise-heat stress to optimize the balance between cutaneous and skeletal muscle blood flow. First, blood flow to the splanchnic organs and kidney is reduced; with the magnitude of reduction proportional to the exercise intensity and environmental heat stress (8). The lower blood flow to these organs reduces their blood volume, helping maintain central blood volume, cardiac filling pressure and arterial blood pressure. Second, at the onset of exercise, a generalized venoconstriction occurs in proportion to the severity of exercise (9,10). The venoconstriction acts to reduce skin blood volume, enhancing central blood volume and cardiac filling (8).

The cardiovascular system has finite limits in its ability to distribute blood volume during exercise-heat stress. The redistribution of blood into the cutaneous circulation during heat stress can compromise cardiac filling, cardiac output and blood pressure. Figure 14-4 illustrates the cardiovascular responses of six subjects during graded exercise in a neutral (25.6°C) and hot (43.3°C) environment (8). When exercising in the hot environment the subjects had higher heart rates, but lower central blood and stroke volumes compared to the neutral environment. While cardiac output was similar between the hot and neutral environments during the two lower exercise intensities, cardiac output was lower in the hot environment at the two higher exercise intensities. The lack of ability to redistribute sufficient blood volume to preserve

cardiac output during heat stress is further evidenced by the 5 to 8% lower maximal oxygen uptake during exercise in hot environments (11-15). The greater cardiovascular strain can reduce exercise tolerance and impair endurance exercise performance.

METABOLIC RESPONSE TO EXERCISE-HEAT STRESS

Whether heat stress increases the caloric requirement to perform a specific task remains unclear. While several investigators have reported elevated metabolic rates with exercise-heat stress (16-20) others have reported no change or lower metabolic rates (21-24). One explanation for the discrepancy may be contributions of anaerobic metabolism to total energy cost, as the investigations reporting similar or lower metabolic rates during exercise-heat stress also reported increased plasma or blood lactate concentration (22-24).

The impact of exercise-heat stress on muscle blood flow and metabolism have been examined in several studies. Exercise-heat stress does not appear to impair either blood flow nor oxygen delivery to the working muscle as two recent studies reported similar leg blood flow and leg oxygen uptake during moderately-intense exercise in hot (40°C) and temperate (20-23°C) climates (20,25). Exercise-heat stress also appears to have little impact on muscle glycogen utilization (20,24,26). Therefore, there is little evidence to suggest that hot ambient air temperatures impair oxygen delivery to skeletal muscle nor increase skeletal muscle glycogenolysis during exercise.

SKELETAL MUSCLE STRENGTH AND FATIGUABILITY

Several investigations have reported that sprinting and jumping performance are improved after warming skeletal muscle (27,28). Bergh and Ekblom (27) found that maximal torque and power output during knee extensor exercise were positively related to muscle temperature, with each measure changing $\sim 5\% \cdot ^\circ\text{C}^{-1}$ over a muscle temperature range of 30-39°C. Other investigators report no difference in maximal isometric force (29-31) and a reduction in submaximal isometric endurance (29,32-34) following skeletal muscle heating. One explanation for these disparate findings may

be the magnitude of increase in muscle temperature accrued with heating, as there is an optimal skeletal muscle temperature for isometric endurance (32-34). Therefore, muscle performance can be compromised if its temperature becomes excessively elevated during exercise-heat stress.

SPECIAL CONSIDERATIONS

Acclimatization

Physical tasks that are relatively easy to perform in cool weather can become extremely difficult to complete during initial exposures to hot weather. Depending on the exercise intensity and accompanying physiological strain, heat exposure may result in higher core temperature, a more rapid heart rate, narrowed pulse pressure, headache, dizziness, cramps, dependent edema, flushing of face and neck, and orthostatic hypotension (35-37). In addition, these symptoms may be accompanied by a feeling of lassitude and increased fatigue. Fortunately, repeated exposure to hot environments produces adaptations which improve an individual's work capacity during subsequent heat exposures. The physiological adjustments that improve work capacity in the heat tolerance are collectively termed heat acclimatization.

As depicted in Figure 14-5, most of heat acclimatization occurs during the first week of heat exposure and acclimatization to a given environmental condition is virtually complete after 10 days of exposure. Heat acclimatization leads to marked reductions in exercise heart rate, core temperature, skin temperature and the core temperature at which cutaneous vasodilatation and sweating begin (38). There appears to be little change in cardiac output, mean arterial pressure, pulse pressure, or total vascular conductance with acclimatization (38).

The reductions in heart rate and core temperature are greater and occur more quickly with the combination of exercise and heat stress rather than heat stress alone (39). Furthermore, heat acclimatization only occurs when an individual is "stressed" by the environment. Little acclimatization to the hot midday weather will occur if an individual trains in the cool hours of the morning or evening and spends the rest of the day in an air conditioned room.

Regular exercise in a cool environment has been shown to improve one's ability to thermoregulate during exercise-heat stress (40,41). The exercise-induced "internal" heat stress (e.g. increased core temperature) stimulates adaptation of the peripheral circulation and evaporative capacity similar to that which occurs with heat exposure. As a result, aerobically trained persons store less body heat during exercise than untrained persons. However, regular exercise in a cool environment does not fully acclimatize an individual for exercise in a hot environment, as regular training in a hot environment can further reduce the thermal and cardiovascular strain associated with exercise in the hot environment (42-45). Therefore, if exercise is to be performed in hot weather, it is optimal to acclimate to that climate.

Dehydration

Dehydration, or body water deficits, accrue during exercise-heat stress if sufficient fluids are not ingested to offset sweat losses. Dehydration results in elevated body core temperature and heart rate during exercise (41,46-48), with the magnitude of these increases graded in proportion to the amount of dehydration (Figure 14-6) (41,47). Dehydration will negate the thermoregulatory advantage of heat acclimation and aerobic training (49,50). Dehydration also increases perception of effort (47) and reduces endurance exercise performance (41,46,51). In addition, a recent study (52) showed that dehydrated subjects incurred exhaustion from heat strain at lower core temperatures than when normally hydrated during uncompensable heat stress; suggesting that dehydrated persons have a reduced tolerance to exercise-heat strain.

To minimize dehydration, fluids should be ingested during prolonged exercise. The increase in core temperature during exercise is smallest when fluid intake most closely matches sweat loss and no dehydration occurs (47,53). In hot climates, this may require persons to drink 1.0 to 1.5 liters of fluid per hour. The timing of rehydration during exercise is not critical to thermoregulation if the volume of fluid intake is adjusted accordingly; as the rise in core temperature is dependent on the magnitude of fluid loss and not the timing of fluid intake (54). The composition of the fluid replacement solution can vary as well. Commercial carbohydrate-electrolyte solutions with up to 8% carbohydrate appear to be equally effective as water for

attenuating increases in core temperature and heart rate during prolonged exercise (55).

While dehydration produces graded increments in core temperature during exercise-heat stress, and can impair endurance performance, it remains unclear whether drinking large volumes of fluid to prevent dehydration actually improves athletic performance. Drinking large volumes of fluid during prolonged exercise may force athletes to slow to consume the beverage and subsequent race pace may be compromised due to gastric bloating and discomfort (56). The athlete will need to determine whether the time lost as a result of drinking larger volumes of fluid is compensated for by the physiological benefits obtained by attenuating dehydration during prolonged exercise.

Aging

Studies have documented that older populations have a greater incidence of heat intolerance compared to young adults (57). The elderly also exhibit a larger than normal basal body temperature fluctuation compared to young adults (58). This latter liability appears largely due to a reduced circulatory effectiveness in controlling heat transport between the body core and the cutaneous circulation. Research now suggests, however, that aging *per se* is not responsible for the greater incidence of heat intolerance in the elderly (59,60). When matched for activity and aerobic fitness, and exercise is performed at the same relative percentage of maximal aerobic capacity, older adults have similar increases in core temperature compared to young adults. In addition, aging *per se* does not impair the ability to acclimatize to hot environments (59,60). The greater incidence of heat intolerance observed in the elderly is now thought to result from functional changes accompanying a more sedentary lifestyle, obesity, a reduced sweating response, cardiovascular insufficiency, and the effects of medication (57).

Gender

Both men and women have similar thermoregulatory responses during exercise and moderate heat stress when matched for fitness and training (61-63). Yet,

differences between genders do exist. Women appear to sweat more efficiently than men in humid climates, as more of their secreted sweat is evaporated and less sweat drips from the body prior to evaporation (63,64). The female menstrual cycle produces regular fluctuations in basal body temperature. During the luteal phase of the menstrual cycle, basal body temperature is roughly 0.4°C higher than during the follicular phase (65). Whether the phase of the menstrual cycle alters the ability to regulate body temperature remains unresolved. While several investigations have reported that the luteal phase simply produces a graded increase in exercise steady-state core temperature compared to exercise during the follicular phase (65,66), a separate investigation found that females were unable to achieve a steady state core temperature during the luteal phase (67). The ability of women to effectively train and compete throughout their menstrual cycle, however, suggests that body temperature is adequately regulated during the luteal phase, albeit at a higher core temperature.

Clothing

Clothing impairs the ability to dissipate heat during exercise-heat stress as clothing creates a barrier for the transfer of heat between the skin and the environment. The properties of the clothing, such as its insulation properties, air and vapor permeability, as well as the proportion of the body surface covered, will determine the impact of the clothing on body temperature regulation and exercise-heat tolerance. Warm weather clothing should be loose fitting to permit free circulation of air between the skin and the environment. Further, it should be light colored to reflect radiant light and minimize radiant heat gain in sunny weather. The Protective clothing worn by fire fighters and soldiers on the chemical/biological battlefield greatly increase the thermal and cardiovascular strain of exercise, and reduces physiological tolerance to heat strain (68,69).

Exercise Training in Hot Environments

Exercise training can be conducted safely in hot environments if precautions are taken to minimize the risk of heat illness. Persons exercising in hot environments should attempt to attenuate dehydration by drinking fluids before and during exercise. Individual fluid requirements can be estimated from body weight lost during exercise.

Proper clothing should be worn to minimize added heat stress during the exercise bout. Finally, persons exercising in the heat should adjust the exercise intensity to accommodate the added circulatory strain accompanying exercise in hot environments.

Heart rate remains a reliable indicator of cardiovascular strain during exercise-heat stress. As such, use of exercise heart rate to set the exercise intensity is a good method for reducing thermal strain in hot climates. However, it should be realized that this method will reduce the training stimulus placed on skeletal muscle.

Populations Susceptible to Heat Intolerance

While the etiology of heat intolerance is poorly understood, there are a number of congenital and acquired risk factors that have been associated with reduced heat tolerance (Table 14-1). The identification of susceptible persons is important to reduce the risk of heat injury during work in hot weather.

Congenital anomalies underlying heat intolerance usually impair the ability to produce and secrete sweat onto the skin surface. Ectodermal dysplasia is the most common form of congenital anhidrosis. It is attributed to an autosomal dominant or x-linked recessive trait and, therefore, affects only males (70). The sweat glands of those afflicted are either absent, functionally impaired, or histologically present but not innervated. Patients with cystic fibrosis and Parkinson's disease have altered neural sweat gland regulation, which may predispose them to heat intolerance during environmental heat stress (71-73).

Several skin disorders, such as miliaria rubra (heat rash), impede heat transfer, increase heat storage and impair performance (74-76). Psoriatic patients have been shown to have reduced sweating and elevated core temperatures during heat stress (76). Burn victims have an impaired ability to sweat, and heat tolerance is negatively correlated to the burnt skin area (57). Conversely, mild sunburn does not seem to increase the thermal strain accompanying exercise in hot climates (77).

Obese individuals appear to be less tolerant to heat stress (78,79). The lower heat tolerance in this population may be due to their lower cardiovascular fitness compared to lean individuals (as evidenced by a higher heart rate at rest and during exercise) as well as their lower surface area to body mass ratio (57).

Spinal cord injured persons cannot dissipate body heat as effectively as able-bodied persons during exercise-heat stress. They cannot redistribute blood flow as effectively as able bodied persons and maximal whole body sweat rate is impaired due to insensate skin. The magnitude of this impairment is dependent on the level of the spinal lesion.

Patients with multiple sclerosis typically demonstrate adverse reactions to heat exposure. External heat, either in the form of climatic conditions or therapeutic modalities may exacerbate clinical symptoms and induce fatigue (80).

Various pharmaceutical agents impair heat transfer to the environment (81-83). For example, anticholinergic drugs, such as Atropine, increase core temperature and reduce exercise tolerance time when working in hot environments (84). Acute amphetamine poisoning commonly leads to hyperpyrexia and fatal heat stroke (57). Neuroleptic drugs, such as Chlorpromazine, can also impair heat transfer (81).

HEAT ILLNESS

There are several illnesses that may occur during exercise-heat stress. The clinical features and treatment of these illnesses has been discussed in detail elsewhere (85,86)

Heat Cramps

This condition can arise after heavy and prolonged sweating. Etiology is unknown but is associated with low serum electrolyte concentration. It is characterized by skeletal muscle twitching, cramps and spasm. It generally occurs in the arms, legs and abdomen. The incidence of heat cramps can be minimized by insuring acclimatization and inclusion of salt supplementation at mealtime.

Heat Exhaustion

This disorder is the most common encountered form of heat illness. It is a "functional" illness and is not associated with organ damage. Heat exhaustion arises when the cardiovascular system is unable to meet the competing demands of thermoregulatory skin blood flow, skeletal muscle and vital organ blood flow. Individuals become fatigued, with orthostatic dizziness and ataxia. Common features include dyspnea and tingling in hands and feet. Persons may be disoriented, uncoordinated mentally dull. Acute management should be focused on reducing cardiovascular demand and reversing water and salt deficits.

Heat Stroke

At presentation, the distinction between heat exhaustion and heat stroke is very difficult. Heat stroke is distinguished from heat exhaustion by the presence of tissue injury. The degree of injury appears dependent on both the magnitude of core temperature elevation and the duration of exposure. Pre-existing conditions (e.g. hypokalemia, endotoxemia, tissue ischemia) probably have a role in evolution of heat stroke. It is a serious illness that requires medical attention. If heat stroke is suspected and temperature is elevated, active cooling should be initiated and the person transported to medical facilities.

Miliaria Rubra and Miliaria Profunda

Miliaria rubra (heat rash, "prickly heat") is a papulovesicular skin eruption that can arise when active eccrine sweat glands become occluded by organic debris. It generally occurs in humid climates or to skin sufficiently encapsulated by clothing to produce high humidity conditions. It is treated by cooling and drying the skin, treating itching symptoms and controlling for infection. Exercise-heat exposure should be avoided until the rash has disappeared.

Miliaria that becomes generalized and prolonged is termed miliaria profunda. It can lead to anhidrotic heat exhaustion as the occluded sweat glands no longer produce sweat for evaporation. The lesions are truncal, noninflamed and papular.

They are also asymptomatic. Persons with miliaria profunda are less heat tolerant compared to the general population.

Heat Tetany

A rare condition which occurs in individuals acutely exposed to overwhelming heat stress. It appears to be caused by hyperventilation. Persons present with respiratory alkalosis, carpopedal spasm and syncope. They should be removed from the heat and treated for hyperventilation.

COLD EXPOSURE

THERMOREGULATION IN COLD CLIMATES

Cold weather need not be a deterrent for outdoor exercise. Physical exercise can be performed safely in extremely cold conditions if sufficient clothing is worn to maintain body core temperature and protect the skin from cold injury. It does require special consideration for the exercise participant, however, as inadequate clothing can lead to rapid reductions in body temperature and commensurate impairment of exercise performance.

Behavioral thermoregulation (i.e., seeking shelter and clothing) is man's most effective means to tolerate cold exposure. Cold weather clothing serves to add an insulation layer between the body core and the ambient air/water; enabling persons to tolerate extremely cold ambient temperatures without becoming cold. The amount of clothing necessary to maintain thermal balance is inversely related to metabolic rate.

Humans have two primary physiological responses to cold exposure, both which attempt to prevent or minimize a fall in body core temperature. The initial response is cutaneous vasoconstriction. This reduces blood flow to the periphery and increases insulation. The second response is to increase metabolic heat production by shivering and/or physical activity.

Peripheral Vasoconstriction

Cutaneous vasoconstriction is sympathetically-mediated. It reduces heat transfer from the core to the periphery and adds an insulative layer between the body core and the environment. Depending on the magnitude of cold and the duration of exposure, cold exposure can also induce limb skeletal muscle vasoconstriction (87). Peripheral vasoconstriction is initiated with the onset of skin cooling, however, muscle blood flow is maintained until cold strain is severe enough to compromise core temperature (87).

In many individuals, the hand and feet demonstrate cyclic blood flow when exposed to cold. This phenomenon, often referred to as the "hunting reflex" is now more commonly termed, "cold induced vasodilatation" or "CIVD". It occurs due to rhythmic cycling of sympathetic vasoconstrictor stimulation. The cyclic warming and cooling of these peripheral tissues is generally thought to be beneficial for preventing peripheral cold injuries and improving manual dexterity. It is not observed in all persons, however, and can be highly variable within an individual. Exercise can modify peripheral vasoconstriction. If exercise heat production exceeds the rate of heat loss, peripheral vasoconstriction will attenuate and cutaneous blood flow will increase. During moderate cold stress, exercise can completely abolish the peripheral vasoconstriction. However, the peripheral vasoconstrictor drive will persist if the heat produced during exercise is lower than the rate of heat loss.

Thermogenesis

At rest, humans increase heat production primarily through shivering, i.e., the involuntary contraction of skeletal muscle. Shivering is stimulated when cold stress reduces skin and/or core temperature. The magnitude of shivering is dependent on the relative intensity of the cold stress as shivering is proportional to the decrement in body temperature (88,89). To support shivering, oxygen uptake increases up to a value of $\sim 1.5 \text{ l}\cdot\text{min}^{-1}$ as body temperature declines (87-89). Some heat may also be released from other metabolic yet non-shivering sources, such as digestion, although the contribution appears limited (90). Physical exercise can produce substantial heat and offset heat loss in cold weather. As discussed, physical activity can increase

metabolic heat production 15 fold. Endurance trained athletes may, during competition, exercise for several hours at metabolic intensities which produce 700-800 watts of heat (47,54). This rate of heat production enables these individuals to exercise comfortably with minimal clothing in cold air temperatures.

CARDIOVASCULAR AND METABOLIC RESPONSES TO EXERCISE DURING COLD STRESS

The peripheral vasoconstriction induced by cold exposure redistributes blood from the periphery to the body core. As a result of this redistribution of blood, end-diastolic volume and stroke volume are elevated during cold exposure. Cardiac output at a given oxygen uptake remains unchanged, however, as heart rate is reduced during cold exposure (91). Diastolic blood pressure is increased ~10 torr as consequence of peripheral vasoconstriction (87).

Cold exposure can increase caloric expenditure during submaximal exercise. However, the impact of cold exposure on metabolic rate is dependent on exercise intensity and the effect of cold exposure on core temperature. At rest and during low intensity exercise, it is not uncommon for oxygen uptake to be elevated compared to temperate conditions (91-93). During more intense exercise, oxygen uptake is generally similar in cold and temperate conditions. This relationship is likely due to the additive energy cost imposed by shivering during low intensity exercise.

Cold weather clothing and winter terrain (e.g. snow and slush) increase caloric expenditure during exercise/activity. Bulky and heavy cold weather clothing increase the energy cost of exercise up to 10-20% (94,95), depending on the number of clothing layers and exercise task. Similarly, walking or running through snow increases the caloric cost compared to exercise on a smooth, firm surface (96). As illustrated in Figure 14-7, walking in moderately deep snow at 2.5 mph increases the energy cost approximately 3 fold compared to walking on pavement.

EFFECT OF BODY COOLING ON PHYSICAL PERFORMANCE

While some studies report no reduction in physical performance during cold exposure, it should be noted that the persons tested were often not cold. It is only when body core and/or muscle temperatures are reduced that decrements in physical performance occur.

Whole body cooling can reduce maximal oxygen uptake (93,97-100). Experiments which have lowered core temperature 0.5 to 2.0°C have reported 10-20% reductions in maximal oxygen uptake. The reduction may result from reduced maximal cardiac output (86) as maximal heart rate is 10-30 b·min⁻¹ lower after body cooling (87,91). In addition, blood temperature reductions shift the oxygen dissociation curve to the right, reducing oxygen delivery to the cells.

Body cooling has been shown to reduce endurance exercise performance (97,101,102). This may be due to increased metabolic strain as blood lactate begins to accumulate at lower workloads and the rate of accumulation is greater during incremental exercise in the cold (99,100). In addition, since maximal oxygen consumption is reduced in the cold, any given submaximal intensity represents a higher percentage of maximal oxygen consumption and, therefore, a greater relative exercise intensity.

The discomfort of severe cold stress may be, in part, responsible for reduced tolerance to exercise-cold stress. Adolph and Molnar (103) found that their subjects became confused and stuporous when resting or working in the cold. They speculated that the pain and discomfort accompanying cold exposure were important causes for termination. Similarly, Doubt (104) reported that 14 of 63 trials in which subjects were immersed in 5°C water were terminated prematurely because of the intolerable discomfort associated with low finger and toe temperatures.

Cold exposure can impair muscle strength, power and fatigability. Investigations have reported that peak power output, sprint performance and jumping ability are lowered by muscle cooling (27,105). Similarly, during the initial minutes of recovery from sustained cold water immersion, there is a reduction in maximal isometric strength and faster time to fatigue during submaximal isometric contractions (29,34,106). Cooling might reduce muscle strength and endurance by interfering with

muscle relaxation (106). However, there is also evidence that cooling impairs muscle excitation-coupling as supramaximal stimulation of motor nerves produces no muscle action potential when skin temperatures are reduced below 6°C (107,108). Regardless of the mechanisms, muscle performance may be impaired immediately following cold exposure if cold exposure has lowered muscle temperature.

During recovery from muscle cooling, strength can be higher compared to temperate conditions. Johnson et al. (109) examined the impact of cold exposure on muscle strength by measuring maximal handgrip strength prior to immersing the forearm for 30 min in 10-15°C water and during a 3 h recovery period. They reported a significant reduction in maximal handgrip strength immediately following cold exposure. However, maximal handgrip strength was 17-21% higher than control measures from 100 to 180 min of recovery. In similar experiments, Oliver et al. (110) observed an acute reduction in plantar flexion strength after pre-cooling the calf in 10°C water for 30 minutes. During a three-hour recovery period, plantar flexion strength became 33-40% higher than control measures from 120 to 180 min of recovery. This muscle strength increase occurred despite the fact that muscle temperature remained below baseline levels during recovery. The mechanism(s) responsible for these strength gains remain unclear. Limb cooling is known to decrease muscle spindle discharge and the monosynaptic reflex (111,112). Possibly, the sensitivity of the spindles is suppressed by limb cooling, enabling potentiation of muscle force production during recovery/rewarming.

SPECIAL CONSIDERATIONS

Ambient Weather Conditions

The magnitude of environmental cold stress is determined by the ambient temperature, wind velocity and wetness. The rate of body cooling is inversely related to the air temperature at temperatures below the critical temperature where heat production cannot balance heat loss. The addition of air motion accelerates body cooling, as the movement of air past the skin surface replaces the warmer insulating air surrounding the body with colder air. As a consequence of air movement, body cooling can occur rapidly in relatively mild ambient air temperatures. The wind chill

index (Table 14-2) provides a useful index for the combined effects of ambient temperature and air velocity on subjective comfort and potential for peripheral cold injury. Skin wettedness increases the effective wind chill and the rate of body heat loss.

Water immersion increases the rate of body cooling compared to ambient air exposure. Water has a 25-fold greater heat transfer coefficient compared to air. As a consequence, body cooling occurs much more quickly, and begins at a higher absolute temperature, in water than air. Low intensity exercise in water can actually speed the development of hypothermia as physical movement increases conductive heat transfer from the active limb muscles to the water with little metabolic heat left to warm the body core (113). However, moderate intensity exercise (heat production approximately 400 watts) can retard or prevent the reduction in core temperature which occurs when resting in 20-26°C water (114).

Clothing

Clothing is an effective method for attenuating body heat loss during cold exposure. By choosing clothing with high insulation, man can remain comfortably warm even in extreme cold conditions. For optimal effectiveness, the insulative value of the clothing should be balanced with metabolic heat production. It is important not to wear too much clothing during cold exposure as this can lead to increases in body core temperature and the stimulation of sweating. Profuse sweating is not advisable in cold climates as many clothing materials lose their insulative properties when wet and peripheral cold injury is more likely when skin is wet. Similarly, if too little clothing is worn, hypothermia and its consequences can occur. It is generally recommended that persons wear several layers of clothing during cold exposure. This method of dressing enables the individual to add or remove clothing as exercise intensity and weather conditions dictate.

As discussed, the energy cost of exercise can be 10-20% higher when wearing cold weather clothing. The greater energy cost means that exercise at the same work rate requires a greater percentage of a person's maximal oxygen uptake. The greater relative exercise stress may shorten time to fatigue.

Body Morphology

Both body morphology (e.g. mass, surface area, etc.) and composition affect the body cooling rate during cold stress. Persons with small body mass and/or high surface area to mass ratios cool faster than larger persons with similar body morphology (114,115). Similarly, persons with thicker layers of subcutaneous fat, and therefore, greater peripheral insulation, cool more slowly than those with less fat (91,92,116,117). A thick subcutaneous fat layer also enables individuals to tolerate lower ambient temperatures before initiating shivering (118). Differences in body size and composition are considered to be largely responsible for much of the inter-individual variability in physiological responses to cold exposure.

Gender

Whether gender alters the ability to defend body temperature during cold stress remains unresolved. While there are well documented differences between men and women during cold stress, these differences may not be due to differences between gender *per se*, but rather due to differences in body mass, body composition, surface area and aerobic power between genders. No study examining the physiological responses to cold exposure, to date, has matched men and women for weight, fitness, body surface area and fatness.

During cold water immersion, women generally lose heat at a faster rate than men (92). This can be explained, in part, by females having a smaller body mass and greater surface area to body mass ratio. However, women show less increase in metabolic rate for the same magnitude of body cooling (119); suggesting that they have a blunted response to reductions in core temperature.

During cold air exposure, women generally maintain body core as effectively as men (119). They accomplish this by maintaining a lower skin temperature compared to men during cold stress (119). The lower skin temperature not only reduces heat transfer to the periphery, it also serves to increase the insulative barrier between the body core and the environment.

Aerobic Fitness/Training

The physiological adaptations which occur with regular physical activity can be improve tolerance to cold environments. Because endurance exercise training increases maximal aerobic power and endurance performance, trained individuals can sustain relatively high exercise intensities for prolonged time periods. This ability is of great value when high rates of heat production are necessary to prevent hypothermia during cold exposure.

It is controversial whether exercise training produces adaptations which improve thermoregulatory responses during cold exposure. There is evidence that endurance trained persons have a reduced sensitivity to cold stress as suggested by a delayed onset of shivering during body cooling (120). However, Bittel et al. (121) found that aerobically fit persons had greater metabolic heat production during cold exposure as well as an increased shivering sensitivity, i.e., the onset of shivering began at warmer skin temperatures. Similarly, skin temperatures during cold exposure have been reported to both increase (113,122) and decrease (123) after physical training. Young et al. (124) have recently shown that aerobic training improves the vasoconstrictor response to cold exposure. Obviously more study is needed to clarify the role of fitness/training on the physiological responses to cold exposure.

Aging

The risk of hypothermia is widely considered to be greater in older compared to young adults, and hypothermia is thought to contribute to the increased winter mortality rate in the elderly (125-127). Studies have reported that older persons are less able than young adults to defend body temperature during cold stress (128). This has been attributed to a reduced ability to vasoconstrict peripheral arterioles during cold exposure (129,130) and a smaller rise in heat production during cold stress (131). Older persons have also been reported to have less CIVD response in comparison to younger persons (132). The differences between the older and younger adults in these studies may not have been due to aging *per se*, but due to other factors, i.e., differences in body morphology, fitness and health. When these confounding

variables are experimentally controlled, the rate of body cooling appears similar between old and young adults in some (133,134) but not all studies (135)

Nutrition Status

Persons who become hypoglycemic during cold exposure have a reduced tolerance to cold stress and are more susceptible to cold injury (136-138). Persons with hypoglycemia exhibit either attenuated shivering or no shivering during cold stress (137-139) and therefore, lower rates of heat production. Interestingly, Gale et al. (139) demonstrated that intravenous glucose infusion restored shivering, within seconds, after euglycemia was restored in persons rendered hypoglycemic during cold stress. The restoration of shivering occurred even in a limb removed from circulation by arterial occlusion. These findings suggest that hypoglycemia affects temperature regulation through a central rather than peripheral mechanism. Regardless of the mechanism, hypoglycemia can have traumatic consequences for persons working in cold environments and should be prevented whenever possible.

Both carbohydrate and fat are oxidized to meet the metabolic cost of shivering. The increase in plasma catecholamine concentration that typically occurs with cold exposure facilitates the mobilization of both glycogen and triglyceride stores (140). Debate exists, however, whether muscle glycogen is an important substrate during shivering as some studies (141-143) report reductions in muscle glycogen concentration after cold exposure while others do not (144).

Acclimatization

Human cold acclimatization has been examined in a variety of populations. Studies of individuals who have a lifetime of repeated cold exposure indicate that cold acclimatization can occur in humans. For example, Australian aborigines, African bushmen, and women breath-hold divers of Korea have different whole body thermoregulatory responses during cold stress compared to persons unaccustomed to cold climates (90,145,146). Similarly, persons whose occupations require repeated limb exposure to cold temperatures have made peripheral adjustments (e.g. enhanced CIVD response) to attenuate the impact of cold during exposure (147,148). The

magnitude of physiological adaptation to cold exposure appears very modest, however, when compared to the marked physiological adaptations associated with heat acclimatization.

People with Chronic Disease/Disability

Individuals with chronic diseases and disabilities may be at more risk for cold-induced injury. Spinal cord injured persons cannot vasoconstrict peripheral arterioles in the insensate skin and have a blunted shivering response during cold exposure. Both of these factors cause their core temperature to decline during even moderate cold stress (149). Therefore, care must be taken to ensure these people wear adequate clothing during cold exposure to protect against cold injury and hypothermia.

Persons with cardiovascular disease are at more risk of having angina during cold exposure since cold increases blood pressure and myocardial oxygen demand. The positive relationship between the incidence of stroke and myocardial infarction with sudden reductions in air temperature (150) is well recognized.

Cold exposure can elicit asthma-like symptoms. It was generally thought that cold exposure increased respiratory resistance due to intrathoracic airway cooling. Evidence now suggests that the increased pulmonary resistance is due to respiratory tract dehydration (151). Medication can prevent the asthma-like symptoms in most sufferers (151).

Persons with multiple sclerosis have improved motor coordination after moderate levels of body cooling. Therefore, cold exposure may be an effective method to temporarily reduce spasticity in this population. Cold appears to exert its effects on motor control by reducing muscle spindle firing and gamma spasticity (111,112). These cold-induced effects decrease the strength of the stretch reflex and reduce resistance to passive movement. The improvements in motor control may persist for several hours following cold exposure.

Drugs

Alcohol can predispose persons to cold injury (136,152,153). Alcoholic beverages, for example, can induce hypoglycemia which, in turn, suppresses shivering (136-138,152). Several prescription medications, such as barbiturates, phenothiazines, reserpine and narcotics act directly on the hypothalamus and interfere with normal temperature control (154).

COLD DISORDERS

Accidental Hypothermia

This condition can occur whenever body heat loss exceeds body heat production. Predisposing factors include, insufficient clothing, inadequate nutrition and fluid intake, infection, alcohol and drug use (155). Persons with mild hypothermia (core temperature 32-35°C) have the ability to spontaneously rewarm and generally do not present with cardiac arrhythmias. Clinical features include, persistent shivering, cool pale skin, bradycardia and hypotension. Peripheral pulses are weak and hypopnea may be present. Persons may be withdrawn and irritable, confused and lethargic. Acute treatment should be directed at warming the individual. Since persons may be hypoglycemic, warm sweet beverages are recommended. Persons with severe hypothermia (core temperature < 28°C) require active rewarming and immediate medical attention.

Freezing Injury (Frostbite)

Frostbite occurs when skin tissue freezes. It primarily afflicts the digits, ears and exposed facial tissue. Initially the frozen tissue is cold, hard and bloodless. The severity of frostbite is classified by depth of the injury. First degree frostbite afflicts the epidermal tissue. Post-thawing the skin may become wheal-like, red and painful. It may become edematous, but does not blister. Second degree frostbite affects the superficial dermis and is characterized by blister formation. Third degree frostbite extends to the reticular layer. It is characterized by edema with hemorrhagic bullae. Permanent tissue loss is possible. Fourth degree frostbite involves the full thickness

of skin. Thawing does not restore muscle function. Affected area will show early necrotic change. There will be permanent tissue and functional loss. Affected area should be warmed gradually (temperatures greater than 39°C can aggravate injury). During initial treatment, affected areas should be protected from physical injury and excessive cold or heat exposure. Elevate tissue to reduce swelling. It can be minimized with proper clothing, insuring adequate diet and hydration, and avoiding unduly prolonged cold exposure.

Non-Freezing Injuries

Sustained exposure to cold-wet conditions can induce skin injury. Chilblain is a skin condition which primarily affects the hands and feet. Persons present with red, swollen, tender areas on the dorsum of the extremities between the joints. The affected area should be warmed and carefully dried. Persons should be treated for infection and re-exposure avoided until the affected region is healed. Trenchfoot can occur with prolonged exposure to wet-cold conditions when circulation is restricted. The tissue becomes pale, anesthetic, pulseless and immobile. The skin is frequently macerated and slightly edematous. After warming there is marked hyperemia, pain and blister formation. Immersion foot can occur after prolonged immersion in cold water. The clinical features are similar to trenchfoot. Acute treatment for both trenchfoot and immersion foot is directed at warming (passively) the affected feet, protecting the area from physical injury, controlling for infection, and treating concomitant hypothermia and dehydration. The non-freezing injuries can be reduced by frequent hand and foot inspection, and by regularly warming and drying exposed limbs.

HIGH TERRESTRIAL ALTITUDE

Improved access to mountainous areas has increased the number of people visiting and living at high altitude. It is now possible for travelers to fly to a major airport and quickly drive to a high altitude destination. In the mountain states of Utah, Colorado, Wyoming and New Mexico, for example, major roads reach 12,000 ft (3650 m), some towns are located above 10,000 ft (3050 m), and lodges at 9000 ft (2750 m)

and higher. In these states, approximately seven million people ascend to altitudes above 8500 ft (2590 m) each year (156).

Most people who ascend to high altitude are unaware of the physiological impact altitude exposure can have on exercise performance and their health. Altitude exposure impairs exercise performance and can produce debilitating illness and death. It has been estimated that 25% of travelers to altitudes greater than 8,200 ft (2,500 m) will develop acute mountain sickness (157); a short duration debilitating illness that can ruin a long-planned vacation. Fortunately, the human body can adapt to the stress of high altitude and preventive measures can prevent many of the altitude-induced illnesses.

PHYSICAL IMPACT OF ALTITUDE EXPOSURE

Ascent to high altitude impairs the ability to transport oxygen to the cell. With ascent from sea level to altitude, the partial pressure of oxygen (PO_2) declines proportionately to the reduction in barometric pressure. The lower atmospheric PO_2 not only reduces the volume of oxygen inspired at a specific pulmonary ventilation, it also reduces the PO_2 at each step of the oxygen transport chain. The result is an impaired ability to transport oxygen to the cell and the development of hypoxia. Typical reductions in PO_2 at several steps of the oxygen transport chain are presented in Table 14-3. Secondary problems accompanying altitude exposure are the reduction in air temperature and humidity, decreased ability to sleep, reduced appetite, dehydration and increased exposure to UV radiation.

PULMONARY VENTILATION AT HIGH ALTITUDE

One of the first observable physiological responses to hypoxia is an increase in pulmonary ventilation. The increased ventilation serves to increase alveolar PO_2 and reduce the alveolar CO_2 partial pressure; thereby improving arterial oxygen saturation. The increased ventilatory volume is achieved through an increased tidal volume but the respiratory rate may also increase. The increase in ventilation can be detected when the inspired PO_2 drops below 110 mm Hg or the arterial PO_2 is less than 60 mm

Hg (158,159). The increase in pulmonary ventilation is considered by many to be the most important response to hypoxia (156).

The magnitude of hyperventilation is dependent on competing signals. The initial increase in ventilation is stimulated by peripheral chemoreceptors within the aortic and carotid bodies as total denervation of the carotid and aortic bodies significantly attenuates or abolishes the ventilatory response to acute hypoxia (158). The increase in ventilation, however, produces hypocapnia and arterial alkalosis, which act to brake further elevations in ventilation.

Altitude acclimatization produces additional increases in minute ventilation, reaching maximum after four to seven days at the same altitude (Figure 14-8, (160)). One mechanism, facilitating this adaptation is the elimination of excess bicarbonate by the kidneys during the initial days at altitude. By removing excess bicarbonate, the kidneys attenuate the arterial alkalosis and the accompanying hypoventilatory drive. It has also been suggested that there may be an increased sensitivity to hypoxia during the initial weeks at altitude (161).

Hyperventilation not only improves arterial oxygen saturation, it also facilitates transport of oxygen to the peripheral tissues. The systemic alkalosis accompanying hypocapnia increases oxygen affinity to hemoglobin, promoting binding of oxygen at the lung. In addition, hypoxia and alkalosis activate the glycolytic enzyme, phosphofructokinase, leading to an increased synthesis of 2,3 diphosphoglycerate (2,3 DPG). 2,3 DPG helps oxygen to dissociate at the tissue level by decreasing hemoglobin's affinity to oxygen.

CIRCULATORY RESPONSES AT HIGH ALTITUDE

Hypoxia alters the cardiovascular responses both at rest and during exercise. Acute exposure to altitude produces a mild increase in blood pressure, moderate increase in heart rate and cardiac output, and increased venous tone (156). Heart rate at rest may initially rise 20% or more (157). Initially, cardiac output is elevated secondary to tachycardia with normal stroke volume, but cardiac output becomes similar or lower than control values after approximately 1 week of exposure. The decline in cardiac output occurs due to reductions in stroke volume (162-165). The pulmonary circulation is also affected by hypoxia as pulmonary vascular resistance and pulmonary arterial pressure are elevated with acute altitude exposure (166).

During acclimatization, two additional circulatory adaptations occur to enhance oxygen transport. The first adaptation is an increase in hemoglobin concentration secondary to reduced plasma volume. The hemoconcentration is apparent within 1-2 days of exposure and persists for several weeks. It arises as a consequence of the diuresis of excess bicarbonate (167) and plasma volume may be reduced as much as 10 to 20% (300-600 ml) (161). The trade-off to this mechanism, however, is that the lower blood volume reduces stroke volume, increasing circulatory and thermoregulatory strain during exercise (168). A later adaptation is an increase in red cell volume. Within two hours of hypoxemia, plasma erythropoietin concentration is elevated; stimulating red blood cell production in the bone marrow. Within four to five days of stimulation, new red blood cells are in the circulation (156). Over a time period of weeks to months, red cell mass increases in proportion to the degree of hypoxemia (169) and plasma volume returns to near sea level values (161).

Maximal oxygen uptake is not measurably altered between sea level and 4,900 ft (1,500 m). Above 4,900 ft (1,500 m), however, there is a linear decline in maximal oxygen uptake at a rate of 10% per 1,000 m (170). At 14,100 ft (4,300 m), maximal oxygen uptake is reduced approximately 27-28% (171). There appears to be no gender influence on this response (172). While there is evidence that individuals with a high aerobic capacity have larger decrements in maximal oxygen uptake with altitude (171), there is large inter-individual variability in response to hypoxia. Acute hypoxia appears to reduce maximal oxygen uptake by impairing maximal oxygen

carrying capacity as maximal values for heart rate, stroke volume, cardiac output, venous O₂ content and ventilation are not reduced from sea level values (164,173-175) and the magnitude of reduction in maximal oxygen uptake is similar to the reduction in arterial O₂ content (173,175).

Altitude acclimatization has little effect on maximal oxygen uptake. Studies generally report either no change in maximal oxygen uptake or only modest increases (3-5%) following short-term altitude acclimatization (161,165,176-180). The modest results may be due, in part, to detraining as training time and intensity are reduced due acute mountain sickness and hypoxia. It may also be due to a lower maximal cardiac output, as maximal heart rate is reduced following acclimatization to high altitude (181,182) and this is not completely reversed by breathing normoxic air nor with atropine infusion (181).

The reduced oxygen carrying capacity at high altitude also affects the physiological responses during submaximal exercise. At high altitude, the same absolute power output elicits a greater percentage of maximal oxygen uptake than at sea level (Figure 14-9). As a consequence of the lower maximal oxygen uptake at altitude, heart rate, ventilation and lactate concentration are higher during hypoxia than at sea level at the same power output (174,175). During exercise at the same percent of environment specific maximal oxygen uptake, however, heart rate, ventilation and lactate concentration are similar between hypoxia and sea level conditions (183-186).

EXERCISE PERFORMANCE AT HIGH ALTITUDE

Acute exposure to hypoxia impairs endurance performance. Hypoxia reduces the time to exhaustion during exercise at a given absolute power output, with the magnitude of decrement proportional to the magnitude of hypoxia. Reducing the workload to elicit the same percentage of environment specific maximal oxygen uptake results in similar time to exhaustion during high altitude as observed during sea level exercise (165,186).

Acclimatization to high altitude can dramatically improve time to exhaustion during submaximal exercise (165,186,187) as well as athletic performance during

sporting events (178). For example, Maher et al. (186) reported a 45% increase in time to exhaustion during cycle exercise between the second and 12th day at 4300 m, and Horstman (165) found a 60% increase in treadmill running time between the second and 16th day of acclimatization at the same altitude.

The improved endurance performance appears to be due, at least in part, to altered substrate utilization and less disturbance of cellular homeostasis. Following high altitude acclimatization, there is a less reliance on anaerobic energy metabolism, as evidenced by lower plasma ammonia and lactate concentrations (186,188,189), less glycogen utilization (188), and a reduced lactate production (189). There is also less disturbance of cellular homeostasis as the [ATP]/[ADP] ratio is higher and the inorganic phosphate concentration lower, after high altitude acclimatization (190). Since intracellular acidosis (which accompanies lactic acid formation) and elevations in inorganic phosphate concentration have been shown to induce fatigue in skeletal muscle (191), less reliance on anaerobic metabolism should enhance endurance performance.

An improved intracellular buffering capacity may be another possible mechanism for the improved endurance performance following high altitude acclimatization. Mizuno et al. (187) reported that two weeks of exercise training at altitude (8,850 ft; 2,700 m) produced a 6% increase *in vitro* skeletal muscle buffer capacity. In addition, they reported a strong relationship ($r=0.83$) between improved skeletal muscle buffer capacity and short duration (range 240 to 380 sec) running endurance time (average improvement +17%) after high altitude acclimatization.

Ascent to high altitude does not impair skeletal muscle strength but does increase fatiguability of skeletal muscle during small muscle mass activity (172,192). Acclimatization to high altitude, however, improves skeletal muscle endurance capacity (192); presumably due to improved oxygen delivery and less disturbance of intracellular homeostasis.

NEUROPSYCHOLOGICAL BEHAVIOR AT HIGH ALTITUDE

Acute hypoxia exposure can impair neuropsychological function. Relatively small reductions in arterial oxygen saturation significantly impair mental and motor coordination, personality and judgement (193). The ability to do mathematical calculations, make decisions and perform coding and conceptual reasoning tasks are reduced when inspired PO_2 is reduced below 110 torr. Short term memory begins to be affected at inspired PO_2 below 118-127 torr; declining progressively with further reductions in O_2 saturation.

Several of the perceptual and motor decrements associated with acute altitude exposure are lessened with acclimatization. Decrements in ability to perform simple mental tasks, such as coding, are attenuated with as little as 4 days of exposure. Perception of effort during exercise at a given percent maximal oxygen uptake is lower following acclimatization to altitude. However, it has been suggested that the improvements may be more related to recovering from mountain sickness than acclimatization *per se* (161).

SPECIAL CONSIDERATIONS AT HIGH ALTITUDE

Exercise Training

Because high altitude reduces maximal oxygen uptake and results in elevated heart rate at a given oxygen uptake, exercise prescriptions based on tests at sea level will need modification. Heart rate remains a reliable indicator of cardiovascular stress during exercise, but exercise at a given heart rate will provide less absolute stress to skeletal muscle. Therefore, to provide the same overload to skeletal muscle exercise will need to be performed at a greater absolute heart rate.

Optimal training for improving athletic performance at altitude depends on the altitude of residence and the athletic event. For aerobic activities (events lasting longer than three to four minutes) at altitudes above 2,000 m, acclimatization for 10 to 20 days is necessary for maximal performance (156). Highly anaerobic activities at intermediate altitudes only require arrival at the time of the event (156).

The effects of high altitude training on sea level athletic performance remain controversial (194). Studies have suggested that sea level performance can be improved by living and training at altitude (176,180,187,195). These studies, however, have often lacked a control group performing similar training at sea level. Studies containing the proper control groups (177,179,196) have not found altitude training to be superior to sea level training with regard to sea level performance. Regardless, any benefit appears dependent on choosing an altitude that maximizes physiological stress but minimizes the detraining which is inevitable when maximal oxygen uptake is limited (altitudes greater than 4,900 to 6550 ft (1,500 to 2,000 m)). Levine et al. (194) recently showed that athletes train faster and at greater oxygen uptakes at low altitude (3900 ft, 1,200 m) than at higher altitude (9,150 ft, 2,800 m); suggesting that altitude exposure may compromise training overload. They also found that athletes who lived at altitude, but trained near sea level, had improved maximal aerobic power (+5%) and 5,000 m running time (30 seconds) compared to a control group who lived and trained near sea level. Levine et al. (194) suggest that living at altitude stimulates beneficial changes for athletic performance and training near sea level provides optimal exercise training.

People with Chronic Diseases

There remains little information on the impact of altitude on health of people with chronic diseases, nor is much known about the impact of medicines during hypoxia. The first 5 to 10 days at altitude are the most dangerous period for those with cardiovascular disease. It is known that patients with coronary artery disease have decreased exercise tolerance, and earlier onset of angina and ST-segment changes during exercise at altitude (197-199). Those who can achieve 9 min on the Bruce protocol without anginal symptoms will probably be safe at altitude (157). Those with severe angina and limitation of effort at sea level should not go to altitude as hypoxia will increase cardiac energy demand and precipitate anginal episodes. Those afflicted should choose an itinerary with access to easy descent and medical help. A good suggestion is to simulate the activity at moderate altitudes before attempting a specific activity at higher altitudes.

Individuals with mild to moderate lung disease may tolerate modest altitudes but have a higher incidence of acute mountain sickness (200). Patients with primary pulmonary hypertension do not do well at altitude. Diabetics may experience problems regulating insulin dose because of varying energy expenditure and food intake. More frequent insulin dosing may be needed as well as more frequent blood glucose determinations. The presence of sickle cell disease is a contraindication for ascent to altitude as ascent to altitudes as low as 6320 ft (1925 m) are associated with an increased incidence of clinical symptoms (157).

HIGH ALTITUDE ILLNESS

Acute mountain sickness

This is a common illness that arises after rapid ascent (<24 hours) to altitudes above 8,200 ft (2,500 m); with symptoms beginning within hours to days after exposure. Persons often present with headache, anorexia, insomnia, nausea, and malaise (156,157,201). In moderate forms, there may be vomiting, unrelieved headache and decreased urine production. Severe mountain sickness produces altered consciousness, localized rales, cyanosis and ataxia. It is most common among those who ascend quickly and those who have a past history of acute mountain sickness. Vigorous physical activity during ascent or within 24 h of ascent increases the incidence and severity of symptoms. Whether physical fitness attenuates the symptoms remains questionable. It is most likely in those who retain fluid at altitude and have a blunted ventilatory response (167,202). The symptoms will typically improve over a few days if hypobaric stress is not increased.

High Altitude Pulmonary Edema

This illness occurs in 5 to 10% of those with acute mountain sickness and has a high mortality rate (157). It often occurs the second night after ascent to altitude and is thought to be due to hypoxia-induced pulmonary hypertension and increased permeability of the pulmonary capillary endothelium. It is characterized by elevated pulmonary artery pressures and marked ventilation-perfusion mismatch; a patchy edema is typically seen on chest roentgenograms. The bronchoalveolar fluid has a

high protein content and contains increased numbers of macrophages, leukotrienes and evidence of complement activation. Features include decreased exercise performance, dry cough, fatigue, tachycardia, rales in the middle right lobe, and tachypnea. Later cyanosis, extreme weakness, productive cough, and dyspnea may be present at rest. Mental confusion, irrational behavior and coma can also occur. It is most common in people who ascend rapidly, perform strenuous exercise upon arrival, are obese, are of male gender and have a previous history of pulmonary edema. High altitude pulmonary edema is a medical emergency requiring rapid descent and medical attention. Acute treatment should be directed at increasing oxygen availability and decreasing pulmonary artery pressure.

High Altitude Cerebral Edema

This illness usually occurs several days after the onset of mild acute mountain sickness and arises as result of increased brain cell volume. It's symptoms include impaired judgement, inability to make decisions, irrational behavior, severe headache, nausea and vomiting, truncal ataxia, severe lassitude, and progression to comma. Cerebral edema is most common in people who ascend rapidly to significant altitudes; it rarely occurs at altitudes below 10,000 feet. High altitude cerebral edema is a medical emergency requiring rapid descent and medical attention. Acute treatment should be directed at increasing oxygen availability and reducing cerebral edema.

High Altitude Peripheral Edema

Altitude-related edema of the hands and face may occur in up to one-third of travellers to high altitude (201). It is thought to be due to hypoxia-induced retention of sodium and water. The condition is benign, but may cause persons enough discomfort to degrade physical performance. It occurs most often in women and in those with a previous history of high altitude peripheral edema. The condition can be treated successfully with diuretic therapy. Descent is the definitive treatment.

AIR POLLUTION

There are a several chemical compounds present in the atmosphere that can directly impact on our health. These compounds, collectively termed air pollutants, have been classified as either primary or secondary pollutants. Primary pollutants are those emitted directly into the environment. They are produced primarily from the combustion of petroleum based fuels and include carbon monoxide, sulfur oxides, nitrogen oxides, hydrocarbons, and particulates. Secondary pollutants are those which develop from the interaction of the primary pollutants with the environment and include ozone, peroxyacetyl nitrate, sulfuric acid, aldehydes and sulfates. The smog or brown cloud associated with large metropolitan areas usually contains both primary and secondary pollutants.

The quantity of air pollution inhaled during exercise is determined by several factors. The concentration of both primary and secondary air pollutants is influenced by the climatic season, the ambient weather conditions, and the topography of the region. Ozone typically reaches peak concentration in the late morning and early afternoon time periods of summer and lowest at night during the winter months. The highest carbon monoxide concentrations are typically during peak traffic periods. The geography of the region also affects the dispersion of the air pollutants. The relatively high ozone concentrations in the inland valleys of Los Angeles, for example, are the result of prevailing sea breezes pushing the pollutants inland against the mountain ranges that surround the city. Additional factors which determine whether air pollutants will impact our health is whether breathing is done nasally or orally, and the quantity of air inspired. Oral ventilation removes less air pollutants than nasal breathing; resulting in greater exposure to air pollutants per unit time. Since physical exercise increases the rate and depth of inspiration, more air pollutants are inspired during exercise than at rest.

Certain individuals are more susceptible to the adverse effects of air pollutants. It is well documented that sub-threshold concentrations of air pollutants for healthy adults can compromise respiratory and cardiovascular function in children and the elderly, persons with respiratory disorders (e.g. asthmatics, respiratory disease,

chronic obstructive pulmonary disease (COPD) and persons with ischemic heart disease²⁰³⁻²⁰⁶.

In many regions of the country, the air quality meets the national air quality standards (Table 14-4). However, in many areas of Southern California, the concentration of air pollutants often exceed national air quality standards. For example, in Los Angeles County, California, each of the 16 EPA testing stations exceeded the national standard for ozone (0.12 ppm for 1 h) on one or more occasions during a two year period spanning 1991-1993; with several locations exceeding the national standard 50 days or more during the calendar year. Similar results have been reported for the other counties along the coast of Southern California.

Unfortunately, while a great deal of research has investigated the impact of individual air pollutants on pulmonary function and the ability to tolerate the stress of exercise, relatively few studies have investigated the interaction of these pollutants on exercise performance. There is even less information addressing the interaction of air pollutants and other environmental stressors (i.e. heat, cold, altitude) on pulmonary function and exercise performance. The goal of this section of the chapter is to discuss the physiological impact of several major air pollutants, both alone and in combination.

CARBON MONOXIDE

Carbon monoxide is the most frequently occurring air pollutant in urban environments. It is released primarily from auto emissions, but is also a bi-product of industrial combustion and cigarette smoke. Carbon Monoxide impacts our health and physiological function by reducing the oxygen carrying capacity of the blood, as hemoglobin has a 200-fold greater affinity for carbon monoxide than oxygen. Thus, when the carboxy-hemoglobin (COHb) concentration is elevated there is less hemoglobin available to transport oxygen for cell respiration.

While normal resting COHb concentration is approximately 1%, many behaviors can significantly increase COHb concentration. Driving with an open window during peak traffic hours, for example, can raise COHb concentration to 5% (205). Aerobic

exercise under similar conditions can further increase COHb as the greater minute ventilation increases the amount of carbon monoxide inhaled per unit time. Nicholson and Case (207) reported that 30 min of running in New York City resulted in COHb concentrations of 4-5%. Smoking 3 cigarettes over a short time period can increase COHb to approximately 5% (45).

During light to moderate intensity aerobic exercise (30 to 75% maximal oxygen uptake), healthy individuals can tolerate COHb concentrations of up to 20% with no reduction in physical performance (208-210). However, during exercise at 70-75% maximal oxygen uptake, individuals with elevated COHb have increased heart rates, greater respiratory distress and higher blood lactate concentrations compared to control conditions (208,211). In addition, there is evidence that high COHb (25 to 35% COHb) concentrations may result in higher core temperatures during prolonged exercise (212).

Carbon monoxide exposure appears to have little effect on maximal oxygen uptake up to 4% COHb (213). Beyond this apparent threshold, however, maximal oxygen uptake declines approximately 0.9% with every 1% increase in COHb up to 35% COHb (214). Carbon monoxide appears to reduce maximal oxygen uptake by reducing blood oxygen transport capacity. Maximal cardiac output is unaffected by carbon monoxide exposure (213).

Persons with cardiovascular and/or pulmonary disease have a reduced ability to perform exercise when COHb is acutely elevated. Several investigations have reported that patients with angina have a decreased exercise time to angina onset, and systolic blood pressure and heart rate are lower at the onset of angina; suggesting angina occurs at a lower myocardial oxygen uptake (205,206,215-217). Carbon monoxide exposure has also been shown to reduce the exercise tolerance of persons with chronic bronchitis, emphysema (218) and anemia (219).

SULFUR OXIDES

Sulfur oxides are products of fossil fuel combustion and are upper respiratory tract irritants which can cause reversible bronchoconstriction and increased airway

resistance. The dominant forms of sulfur oxides are sulfur dioxide, sulfuric acid and sulfate, with 98% of the sulfur released into the atmosphere in the form of sulfur dioxide (203).

In healthy adults, sulfur dioxide does not impair pulmonary function until the concentration exceeds 1 to 3 ppm (220). However, in asthmatics and others with pulmonary hyperactivity, the threshold for airway restriction is less than 1 ppm (221-224); with one study reporting progressive bronchoconstriction when exposed to 0.1 to 0.5 ppm sulfur dioxide (223). To these authors knowledge, no study has evaluated whether sulfur dioxide exposure impairs either endurance exercise performance or maximal oxygen uptake.

Repeated exposure to sulfur dioxide does induce adaptation. Industrial workers routinely exposed to 10 ppm sulfur dioxide retain normal pulmonary function when exposed to 5 ppm sulfur dioxide pulmonary function (225). Andersen et al. (226) conducted studies investigating the physiological responses which occur when breathing either 1, 5 or 25 ppm sulfur dioxide for 6 h. They reported that the subjects tolerated each concentration of sulfur dioxide "very well", but investigators who occasionally entered the climatic chamber reported the 25 ppm sulfur dioxide concentrations to be "almost intolerable." These adaptations to sulfur dioxide occur in both healthy and asthmatic individuals (227).

NITROGEN OXIDES

Nitrogen oxides develop from high temperature combustive processes involving nitrogen and oxygen. The concentration of nitrogen oxides is elevated during peak traffic periods, at airports, and with smoke accompanying cigarette smoking and fire fighting. There are several forms of nitrogen oxides. They include: nitrous oxide, nitric oxide, nitrogen dioxide, dinitrogen dioxide, dinitrogen pentoxide and nitrate ions. Nitrogen dioxide is known to be harmful to health; as acute exposure to high nitrogen dioxide levels (200 to 4000 ppm) can cause severe pulmonary edema and death (228). At lower concentrations (2-5 ppm), nitrogen dioxide increases airway resistance and reduces pulmonary diffusion capacity (229,230). Fortunately, the ambient air concentration of nitrogen dioxide is generally less than 1 ppm.

For healthy persons, nitrogen dioxide concentrations of less than 0.7 ppm appear to have no adverse effects on pulmonary function or exercise performance (220,231). Aging does not appear to reduce tolerance to nitrogen dioxide (232). However, people with COPD, and presumably other respiratory diseases, have reduced pulmonary function when exposed for 4 h to 0.3 ppm nitrogen dioxide (232).

PRIMARY PARTICULATES

Primary particulates include dust, soot and smoke (203,233). These pollutants are of physiological importance because they can impair pulmonary function after they are inhaled into the lung. The fine dust from charcoal (234) and cigarette smoke (235) have been shown to increase airway resistance and reduce forced expiratory volume. The lung depth reached by the dust is determined by the particle size. The dispersion of dust within the lung is influenced by the tidal volume, frequency of breathing, and whether inhaled nasally or orally (220). Because aerobic exercise increases ventilation and oral inhalation, it is likely that exercise increases the effective dose of this pollutant.

Unfortunately, there is little information on the physiological consequences of particulate inhalation during exercise. Klausen et al. (45) have suggested that inhalation of particulates may reduce endurance exercise time. In their study, subjects performed a maximal exercise test under control conditions, after inhalation of the smoke of three cigarettes and after sufficient carbon monoxide inhalation to raise their COHb concentration the same magnitude as when they smoked three cigarettes. Interestingly, Klausen and colleagues found that raising the COHb concentration produced a 7% reduction in maximal oxygen uptake in both treatment groups, but endurance time during the maximal oxygen uptake test was reduced 20% after smoking but only 10% after carbon monoxide inhalation. They interpreted the results to suggest that the decrease in maximal oxygen uptake after smoking or carbon monoxide inhalation was due to reduced oxygen carrying capacity of the blood, while the decrease in endurance time was a combined effect of the carbon monoxide concentration and the increased cost of breathing caused by the smoke particulates.

OZONE

Ozone is produced in oxygen containing atmospheres primarily from the interaction of hydrocarbons and nitrogen dioxide in the presence of solar UV radiation. As a consequence, ozone is most prevalent in urban areas and reaches its highest concentration during the mid-day hours.

Ozone is a potent airway irritant causing reflex bronchoconstriction in upper airways. The most common subjective symptoms are a cough, substernal soreness, and dryness of the upper respiratory passages. These symptoms can occur after brief exposures to low concentrations of ozone (0.05-0.10 ppm) and can persist for several hours after exposure (203). Furthermore, there may be heightened sensitivity to ozone during the initial hours following ozone exposure (236).

During light to moderate intensity exercise, exposure to ozone in the range of 0.2 to 0.4 ppm reduces pulmonary function and enhances subjective discomfort (237) but does not appear to impair exercise performance. These ozone concentrations can produce cough, substernal pain, wheezing, and malaise during exercise (238); with the incidence of symptoms increasing in proportion to the ozone concentration (239,240).

During high intensity aerobic exercise (75-85% maximal oxygen uptake), ozone inhalation can impair endurance exercise performance. Schelegle and Adams (241) evaluated the effect of 0, 0.12, 0.18 or 0.24 ppm ozone exposure on endurance performance of ten highly trained athletes during 60 min of simulated competition (the last 30 min were at 86% of maximal oxygen uptake). All subjects completed the protocol when breathing 0 ppm ozone, whereas one, five, and seven subjects did not complete the exercise when exposed to 0.12, 0.18 and 0.24 ppm ozone, respectively. Follinsbee (242) reported that inhalation of 0.21 ppm ozone during 60 min exercise at 75% of maximal oxygen uptake induced significant reductions in pulmonary function and six of seven subjects developed subjective distress (symptoms included tracheal irritation, substernal soreness, chest tightness and shortness of breath). Adams and Schelegle (243) also documented decrements in pulmonary function and subjective discomfort. In addition, they reported that four of ten subjects couldn't complete the exercise trials when breathing 0.35 ppm ozone. Similarly, Gibbons and Adams (244)

reported that three of ten subjects discontinued exercise prematurely when exposed to 0.3 ppm ozone during 60 min of moderate intensity (66% maximal oxygen uptake) exercise in a 35°C environment.

Ozone inhalation can reduce maximal oxygen uptake. Follinsbee et al. (245) reported a 10% reduction in maximal oxygen uptake when exposed to 0.75 ppm ozone. Similarly, Gong (240) reported that 0.20 ppm ozone reduced maximal oxygen uptake (-16%), maximal ventilation (-18%) and exercise tolerance time (-30%) during a graded exercise test following 1 h of exercise. Not all investigators have reported reductions in maximal oxygen uptake after ozone exposure (246,247). One explanation for these divergent findings may be the duration of ozone exposure prior to initiating the maximal exercise test, as the studies reporting no change in maximal oxygen uptake exposed the subjects to ozone only during the exercise test.

There is evidence that repeated ozone exposure produces adaptations which attenuate ozone-induced reductions in pulmonary function (237,248-256) and decrements in maximal oxygen uptake and exercise time to exhaustion (257). This adaptation generally occurs within 2-5 days of repeated exposure (237,251-256,258) and persists for 7-20 days upon discontinuation of exposure (255,258); with the least persistent adaptation in persons sensitive to ozone. However, acclimation to ozone does not attenuate decrements in pulmonary function when exposed to higher ozone concentrations (254). The time course of adaptation is similar between genders (237).

INTERACTIONS BETWEEN VARIOUS AIR POLLUTANTS

Because polluted atmospheres contain many contaminants, it is important to investigate whether the combination of various pollutants interact to affect pulmonary function and exercise performance in either an additive, synergistic or subtractive manner. The majority of research has focused on the interaction of various air pollutants with ozone. While the early work of Hazucha and Bates (259) suggested that exposure to 0.37 ppm ozone and 0.37 ppm sulfur dioxide resulted in synergistic reductions in pulmonary function, subsequent investigations have found only additive effects (248,260-262). Koenig et al. (263) recently reported that asthmatic patients

who received prior exposure to ozone had a greater decrement in pulmonary function during subsequent exposure to sulfur dioxide, at sulfur dioxide dosages that are subthreshold for normal subjects. They concluded that ozone exposure made the asthmatic subjects more susceptible to low concentrations of sulfur dioxide.

Experiments which have evaluated the interaction of nitrogen dioxide and ozone suggest no interaction between the two compounds as the effects of the combination produced no greater effects than exposure to ozone alone (238,239,264). The combination of nitrogen dioxide and sulfur dioxide appears to produce only additive effects. Pulmonary resistance is increased abruptly after sulfur dioxide exposure with no persistent effects; nitrogen dioxide has a more delayed and persistent effect (265). Combining subthreshold doses of nitrogen dioxide (0.5 ppm) and sulfur dioxide (0.3 to 0.5 ppm) during 2 h of light intensity exercise does not induce deficits in pulmonary function in either healthy or asthmatic subjects (222).

Particulates interact with carbon monoxide to produce greater reductions in endurance time than carbon monoxide alone (45). However, particulates do not appear to accentuate the effects of ozone on pulmonary function in either normal subjects or asthmatics (266-268).

INTERACTIONS BETWEEN ENVIRONMENTAL STRESSORS

COLD AND ALTITUDE

Ascent to high altitude is typically accompanied by reductions in ambient temperature and humidity, and increased wind velocity. These added environmental stressors will exacerbate the physiological stress of hypoxia alone, potentially compromising exercise performance and/or health. Since temperature typically falls ~10°C with every 1,500 m rise in elevation, even moderate increases in elevation can significantly magnify the degree of cold stress (269). Therefore, persons travelling to high altitudes should bring adequate clothing to protect themselves against environmental cold stress.

The combined effects of cold and hypoxia on injury and exercise performance remain poorly understood, due to lack of experimental information. However, it is likely that the combined stress of cold and altitude exposure predispose persons to injury and premature fatigue as: a) both body cooling and hypoxia impair mental function and decision making ability; b) the lower maximal oxygen uptake at altitude reduces a persons ability to increase heat production and defend core temperature; c) both cold and hypoxic stress are associated with hemoconcentration and peripheral vasoconstriction, which may contribute to peripheral cold injury; and d) since both cold and hypoxia increase blood lactic acid concentration at a given exercise intensity, premature fatigue may occur during exercise.

AIR POLLUTION AND HEAT

Since elevated concentrations of air pollutants are often associated with excessive heat and humidity (270), which are known to impair exercise performance, it is likely that the combined stresses of excessive heat, humidity and poor air quality further impair exercise performance. Gibbons and Adams (244) found that subjective symptoms increased when ambient temperature was 35°C rather than 25°C and exposed to either 0.15 ppm or 0.30 ppm ozone during 60 min of moderately intense exercise. Furthermore, they reported that 3 of 10 subjects were unable to complete the 1 h exercise bout (66% maximal oxygen uptake) when heat and/or ozone were present. Similarly, Follinsbee (271) reported greater reductions in pulmonary function when exposed to 0.5 ppm ozone during exercise in a hot climate compared to a cool climate. However, the same investigator subsequently found no temperature interaction when subjects were exposed to 0.5 ppm ozone and 0.5 ppm nitrogen dioxide (264).

Rather surprisingly, there is no evidence that hot ambient temperatures and carbon monoxide produce either additive or synergistic effects on maximal oxygen uptake or performance. Since both hot ambient conditions and increased COHb have individually been shown to reduce maximal oxygen uptake, it would be expected that the combination would produce additive reductions on maximal oxygen uptake and performance. The only studies to date, however, reported no additive effect on maximal oxygen uptake when heat and carbon monoxide were combined (272-274).

AIR POLLUTION AND COLD/ALTITUDE

Exercise in cold environments can induce reflex bronchoconstriction and cold-induced asthma in approximately 12% of the population (275,276). Unfortunately, no studies have evaluated the interaction of cold temperatures and pollution exposure on exercise performance in this population.

Several studies have examined the impact of carbon monoxide at high altitude (220,277,278). Carbon monoxide exposure at altitude further lowers oxygen transport capacity. The effect appears to be additive, however, as a given COHb at altitude and sea level produces a similar decrement in endurance tolerance time and maximal oxygen uptake at both altitudes (278). Yet, since it takes a smaller concentration of carbon monoxide at high altitude to elicit the same COHb concentration (220), persons at high altitude are more sensitive to the effects of carbon monoxide.

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