

# HEAT AND COLD

## A. Thermal Regulation and the Human Body

### 1. Human Body Temperature

The human body's core temperature varies from day to day, and from time to time, but these fluctuations are small, usually no more than 1.0°C. Humans are homeothermic and body temperature is regulated at about 37°C +/- 1°C. The thermoregulatory center in the hypothalamus plays a very active role in keeping body temperature in the normal range.

External (*climatic*) and internal (*metabolic*) heat sources influence body temperature. Heavy exercise, illness, and not only hot and humid but also cold and windy environments alter body temperature outside the normal range. Ambient temperature, humidity, air movement, and radiant heat from the sun, as well as warm and cold surfaces, contribute to climatic heat stress. Metabolic heat is produced by exercise.

Body temperature reflects a careful balance between heat production and heat loss. There is a continuous heat exchange between the body and the environment. Bi-directional routes for heat exchange are: convection (Cv), conduction (Cd), and radiation (R). There are also two uni-directional routes: metabolic heat (M) increases the thermal load; evaporation (E) decreases the load. The net heat storage (S) formula is:

$$S = M \pm C_v \pm C_d \pm R - E$$

When the net heat storage (S) is positive, body temperature will rise and when S is negative, it will go down.

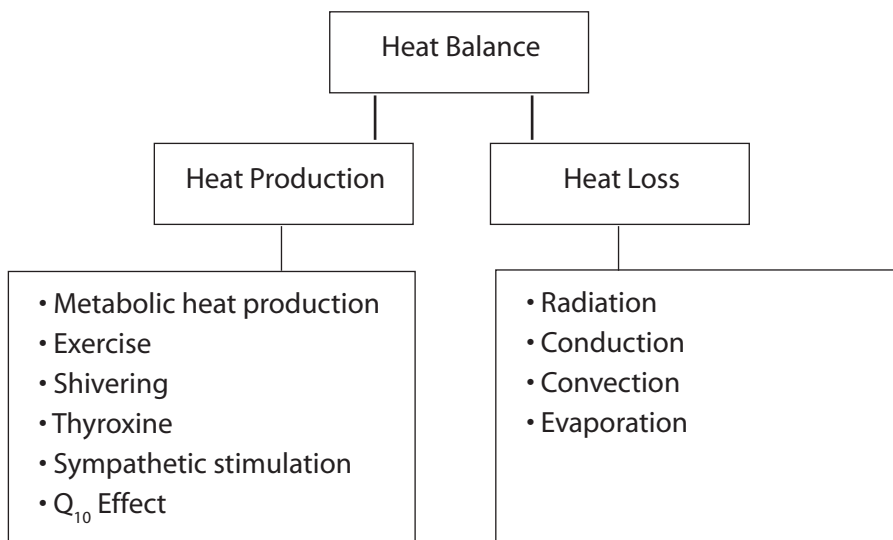


Figure 11-1. Heat exchange between the body and the environment.

## 2. Climatic Heat Stress

Air temperature alone is not an accurate index of climatic heat stress. Temperature and other important factors, including radiant energy, wind velocity and humidity, contribute to climatic heat stress. To determine the overall effect of these factors, a combination of measurements must be made. The Wet Bulb Globe Temperature (WBGT) index has been introduced to assess climatic heat stress. It consists of three thermometers: dry bulb (Tdb), which measures air temperature; wet bulb (Twb), which measures relative humidity; and black globe (Tg), a measure of solar radiation. The difference between the wet and dry bulb temperatures indicates the environment's capacity for cooling by evaporation. A very modern, simple and handy apparatus to measure the WBGT is available.

The WBGT provides a single temperature reading to estimate climatic heat stress and cooling capacity of the surrounding environment.

The Wet Bulb Globe Temperature (WBGT) =

$$(0.7 \times Twb) + (0.1 \times Tdb) + (0.2 \times Tg) \quad (\text{outdoors})$$

$$(0.7 \times Twb) + (0.3 \times Tdb) \quad (\text{indoors})$$

The importance of relative humidity is indicated by its 70% contribution to the WBGT.

The WBGT is the basis for recommendations by the American College of Sports Medicine (see Appendix 4, *ACSM Position Statement: Heat and Cold Illnesses During Distance Running* and Appendix 5, *IAAF Policy on Fluid Replacement*) for exercise under various environmental conditions for the fit general population. Highly trained elite athletes often are able to exceed these limits, but with appropriate cautions.

## 3. Metabolic Heat Stress

A large part of the energy that the body generates is degraded to heat. All body tissues produce heat that can be used to maintain body core temperature. When heat production exceeds the body's heat loss, body temperature rises. Intense exercise may increase the metabolic energy expenditure 20–25 times over resting levels. No more than 25% of this energy is utilised for muscular movement—the remainder is heat, which the body must dissipate. Metabolic heat is transferred by convection from working muscles to the blood stream, and thence to the body's core. Without adaptive mechanisms, even moderate exercise would elevate body temperature by 1°C every 5–6 minutes; thus, exercise would be limited to no more than 20–30 minutes before heat stress fatigue or life-threatening hyperthermia intervened.

In fact, the body is able to respond to a heat load through a variety of physiologic mechanisms: sweat rate, body and skin blood flow shifts, cardiac output, respiratory rate, and a sensation of heat intensity. Well-trained endurance athletes can sustain a core temperature of 39°–41°C for prolonged periods. The muscles' energy systems become more chemically effective with a small rise in muscle temperature. However, a critical thermal maximum is reached at 42°C (108°F), so there is a limited safety margin at intense levels of exercise. Heavier athletes run a higher risk of overheating than lighter athletes when exercising at the same rate.

a. Physiologic responses

Core temperature is sensed by the hypothalamic thermoregulatory center. This center then sets off a number of circulatory adjustments in an effort to dissipate heat. Primarily this involves increasing cardiac output and redistributing blood from the visceral organs to the working muscles and skin.

During heat stress, skin blood flow may be up to 20-fold higher than at rest level. Sweat glands become more active for increasing evaporative heat loss.

Four mechanisms are responsible for heat exchange at the skin surface:

- i. **Conduction** has a minimal effect on body heat transfer, as it depends upon direct contact between skin and a cooler object. Conduction could be utilised by immersion in water to cool or warm the body.
- ii. **Convection** is responsible for transferring heat from working muscles and the skin surface. The circulatory system transports the heat generated in the active muscles to the surface of the body. The air around the body is in constant motion and it sweeps away the warmed air molecules on the skin surface. It is dependent upon (a) the temperature differential between skin and the environment; and (b) the heat transfer coefficient, which varies with available body surface area and wind velocity. Minimal body fat and loose-fitting clothing enhance an athlete's convection potential.

Conduction and convection constantly remove body heat when air temperature is lower than skin temperature. Inversely, convection and conduction cause heat gain in a very hot environment.

- iii. **Radiation**, including solar radiation and radiant heat from tracks, roads, and surrounding structures, can be a major contributor to an athlete's heat load. At rest, radiation is the primary method for discharging the body's excess heat. The heat is given off in the form of infrared rays. Radiation heat loss or gain depends upon the temperature gradient between the skin and the environment.
- iv. **Evaporation** is the most important heat dissipation mechanism in warm environments. Though it accounts for only 20% of body heat loss at rest, more than 80% of body heat loss is achieved by evaporation when environmental temperature exceeds 20°C (68°F) (Table 11-1). Fit athletes can produce up to 30 ml of sweat per minute, but not all of this is available for heat elimination. Evaporative rate is determined by air velocity and the water vapor pressure gradient between the skin and the environment. This latter is determined by the **relative humidity** of the air. High humidity limits sweat evaporation and therefore, heat loss. Each liter of effective evaporated sweat removes 580 Kcal from the body. Evaporation plays a very important role in dissipating heat during exercise.

Table 11-1. Estimated heat loss at rest and during exercise at 70% VO<sub>2</sub> max.

<b>Mechanism of heat loss</b>	<b>Rest</b>		<b>Exercise</b>	
	<b>% total</b>	<b>Kcal/min</b>	<b>% total</b>	<b>Kcal/min</b>
Conduction and convection	20	0.3	15	2.2
Radiation	60	0.9	5	0.8
Evaporation	20	0.3	80	12.0

4. Acclimatisation to Heat and Maximising Heat Loss

Partial adaptation to heat stress occurs even with training at moderate temperatures, but full acclimatisation can be achieved only with repeated bouts of exercise in the heat. At least five to ten days of training in the heat are required for full acclimatisation. This should begin by training at a reduced intensity (60–70% of the usual load), so as to avoid heat-related disorders.

There are numerous adaptations that occur as a result of acclimatisation to exercise in the heat:

- a. Sweat rate in the skin areas exposed to heat is higher, thus enhancing evaporative capacity. An increased production of aldosterone can strongly stimulate the sweat glands and cause them to reabsorb more sodium and chloride.
- b. Earlier onset of sweating leads to a lower skin temperature, improved core-skin temperature gradient, and less demand for blood flow to the skin. This latter provides improved muscle blood flow and oxygen supply.
- c. Plasma volume is increased, due to an increased production of aldosterone and antidiuretic hormone (ADH). Aldosterone causes sodium and chloride retention by the renal and sweat tubules, and ADH increases renal water retention. Hence, there is a decrease in sweat sodium and chloride, but not potassium. More sodium is retained, which promotes water retention. Plasma and interstitial fluid volumes can increase 10–20%.
- d. Body core temperature can be kept lower, as heat dissipation is more efficient.
- e. Heart rate is lower at any given work load, as the core temperature is lower, plasma volume is higher, skin blood flow distribution is decreased, and there is improved stroke volume.
- f. The perception of heat stress is reduced.
- g. Onset of fatigue is delayed, as the rate of muscle glycogen utilisation is decreased.

Men and women acclimatise equally well. Training in a hot, humid environment is more stressful than training in hot, dry conditions.

## 5. Cold Exposure Thermoregulation and Minimising Heat Loss

The body's hypothalamic set-point for temperature regulation is about 37°C +/- 1°C. A decrease in skin or core temperature signals the thermoregulatory center in the posterior hypothalamus to set off a number of mechanisms to increase heat production. These include:

- a. Shivering—involuntary muscular contractions in response to cold. This can cause a 4–5 fold increase in heat production. Shivering results in decreased muscular coordination and impairs performance.
- b. Non-shivering thermogenesis. The sympathetic nervous system releases epinephrine and nor-epinephrine in response to cold exposure, causing anaerobic glycolysis and a release of free fatty acids from fat stores. This mechanism occurs in young children because of their rich brown fat.
- c. Increased thyroxine production. Hypothalamic thyrotropin-releasing hormone (TRH) rises, stimulating TSH release and ultimately elevated thyroxine production to increase general metabolic rate.
- d. Peripheral vaso-constriction. The sympathetic nervous system stimulates skin's smooth muscle contraction, thus shunting blood away from the skin and into deeper tissues.

The balance between heat loss and heat production is controlled by a number of factors. Generally, the greater the gradient between skin and environmental temperature, the greater the heat loss. However, a great number of anatomic and environmental factors affect the rate and degree of heat loss. For example, body size and body composition influence heat loss. Subcutaneous fat acts as an insulator. Smaller athletes such as children have a higher surface area/mass ratio, and may sustain greater heat loss. Clothing helps reduce heat loss.

The degree of heat loss also depends on air movement (convection), humidity, evaporation (sweating), and ambient temperature. Wind velocity exacerbates heat losses from convection, radiation, and evaporation. This is known as the Wind Chill Effect, and is expressed as the Wind Chill Factor (Table 11-2).

Exercise in the cold can affect muscle function. Muscle functions best at a temperature of 40°C. Cooling alters the nervous system and the muscle fiber's recruitment pattern. Muscle shortening velocity and power decrease when the temperature is lowered. Muscle glycogen utilisation is higher during exercise in the cold. Epinephrine and nor-epinephrine secretion increases markedly. However, FFA may not rise as much as in a normal environment, as blood flow to subcutaneous fat areas is lower, and less FFA are mobilised.

So long as clothing is adequate and metabolic rate remains high, the body temperature and function can be maintained. However, as fatigue develops and exercise intensity (i.e., race pace) slows, heat production declines and hypothermia may develop.

Table 11-2. Wind chill index.

Thermometer Reading °F (°C)											
	50 (10)	40 (4.4)	30 (-1.1)	20 (-6.7)	10 (-12.2)	0 (-17.8)	-10 (-23.3)	-20 (-28.9)	-30 (-33.3)	-40 (-40)	-50 (-45.6)
Wind speed (mph)	Equivalent Temperature (°F)										
5	48	37	27	16	6	-5	-15	-26	-36	-47	-57
10	40	28	16	4	-9	-24	-33	-46	-58	-70	-83
15	36	22	9	-5	-18	-32	-45	-58	-72	-85	-99
20	32	18	4	-10	-25	-39	-53	-67	-82	-96	-110
25	30	16	0	-15	-29	-44	-59	-74	-88	-104	-118
30	28	13	-2	-18	-33	-48	-63	-79	-94	-109	-125
35	27	11	-4	-20	-35	-51	-67	-82	-98	-113	-129
40	26	10	-6	-21	-37	-53	-69	-85	-100	-115	-132
	Minimal Risk		Increasing Risk					Great Risk			

c.f. °C = (°F - 32) / 1.8

## B. Thermal Regulation and Disorders

### 1. Heat Related Disorders

Heat related disorders occur when thermoregulatory mechanisms fail to compensate for elevations in core temperature caused by environmental or metabolic heat load. Heat related disorders might encompass a wide spectrum of symptoms of varying severity, ranging from heat cramps and dehydration to heat exhaustion and life-threatening heat stroke.

- a. **Heat cramps**, the least serious type of heat related disorders, are caused by loss of sodium and potassium associated with heavy sweating in unacclimatised individuals.
  - i. Signs/symptoms: painful skeletal muscle spasms, primarily of the muscles most heavily used during exercise (e.g. calves, abdomen).
  - ii. Treatment: move individual to a cooler location and administer fluid/electrolyte solutions or a saline solution, generally taken orally.
  - iii. Prevention: adequate fluid/electrolyte replacement, added salt to food; eat a balanced, high K<sup>+</sup> diet.
- b. **Dehydration** commonly accompanies exercise in warm, humid conditions, when fluid replacement is inadequate. It complicates heat exhaustion and heat stroke.
  - i. Signs/symptoms: fatigue, lethargy, irritability, loss of coordination, faintness, altered consciousness.
  - ii. Treatment: cool fluids; dilute electrolyte solutions.
  - iii. Prevention: pre-hydrate; adequate fluid replacement during activities.
- c. **Heat exhaustion** is a serious heat illness caused by increased exercise heat load plus dehydration. The pathophysiology of heat exhaustion is the inability of the cardiovascular system to adequately supply blood to organs, especially to the brain.
  - i. Signs/Symptoms: “core” (rectal) temperature elevated, usually not above 39.5°C (103°F); “goose flesh”, headache, lethargy, fatigue, dizziness, fainting, hypotension, rapid pulse, altered consciousness, nausea, vomiting, incoordination.
  - ii. Treatment: (See also Appendices 8 and 9, *Differential Diagnosis and Treatment of Exertional Heat Stroke and Heat Exhaustion on Site and in a Clinic Setting*).
    - Move to a cool, shaded area.
    - Remove excess clothing.
    - Elevate feet to avoid shock
    - Begin immediate cooling with cold or iced cloths, sponges, etc. to torso, axillae, groin, other exposed areas.
    - Begin hydrations with cool fluids, orally if possible, otherwise start IV fluids (Dextrose/0.5N saline).
    - Monitor vital signs, rectal temperature if possible.

- Transfer to hospital, when unconscious or incomplete response to therapy.
- iii. Prevention:
  - Avoid competition under adverse conditions, or adjust pace to existing conditions.
  - Utilise acclimatisation measures prior to competition.
  - Pre-hydrate; emphasise hydration during the competition.
  - Wear appropriate clothing, which will “breathe” and allow sweat to evaporate.
- d. **Heat stroke**, a life-threatening heat-related disorder and a medical emergency, is difficult to distinguish from heat exhaustion because sweating may continue. Heat stroke represents thermoregulatory failure, with reduction in skin blood flow in order to maintain the central circulation. Core temperature is more elevated, usually 40°C or higher.
  - i. Signs/symptoms: core temperature exceeding 40°C, cessation of sweating, rapid pulse, rapid respiration, hypotension, CNS symptoms predominate: unsteady gait, confusion, combative behaviour, reduced consciousness, convulsion, and coma. These signs and symptoms represent a medical emergency.
  - ii. Treatment: (see also Appendix 8)
    - Move to a cool, shaded area. Lay down with feet elevated.
    - Loosen or remove clothing.
    - Begin cooling at once. In the field, it may be necessary to assume that temperature is elevated, as taking a rectal temperature may not be feasible. Oral or axillary temperature is quite unreliable. Apply cool water, and fan to increase evaporation. Apply ice packs over major vessels in neck, axillae, groin. Cool to a rectal temperature of 39°C (102°F).
    - Start IV fluids (Normal saline)
    - Evacuate to a medical facility as early as possible. Manage as a medical emergency, with monitoring of cardiac, neurologic, and renal function, and electrolyte balance.

(For prevention and cautions related to thermal injuries, see Appendix 4, *ACSM Position Stand on Heat and Cold Illnesses During Distance Running* and Appendix 5, *IAAF Policy on Fluid Replacement*.)

## 2. Cold Injuries

Activities in water and in alpine locales, and prolonged activity can cause cold injuries. The cold stress conditions of concern are hypothermia and frostbite. Hypothermia affects cardiovascular, respiratory, CNS and neuromuscular systems. Hypothermia occurs when core body temperature decreases enough to affect body functions—usually below 35°C (95°F). It may be classified clinically as mild, moderate, or severe (Table 11-3).



Table 11-3. Categories of hypothermia.

Category	Rectal Temperature	Symptoms/Signs
Mild Hypothermia	33°–35°C	Symptoms/Signs: shivering, very cold, hunger, lethargy, confusion, muscle spasm and difficulty with motor tasks, decreased race pace, slurred speech, ataxic gait, slow reflexes.
Moderate Hypothermia	30°–33°C	Symptoms/Signs: may not be shivering, semi-conscious with confused actions and irrational behaviour, extremely tired, irritable and depressed, poor judgment, loss of memory, disoriented, poor coordination, muscle stiffness, slurred speech, slow and/or irregular pulse.
Severe Hypothermia	less than 30°C	Symptoms/Signs: loss of consciousness, pupils dilated, heartbeat faint or undetectable.

- a. **Hypothermia: Mild.** Rectal temperature 33°–35°C
  - i. Signs/symptoms: shivering, hungry, lethargic, confused, poor coordination, slurred speech, ataxia.
  - ii. Treatment: insulate the athlete with dry clothing, continue mild exercise, and administer warm liquids.
- b. **Hypothermia: Moderate.** Rectal temperature 30°–33°C
  - i. Signs/symptoms: semi-conscious, confused, irrational, disoriented, muscle stiffness, slow, irregular pulse.
  - ii. Treatment: exogenous heat via a warm shower, warm water bottles, body contact, inhale warm, moist air; warm liquids; the athletes should be handled with care due to myocardial irritability.
- c. **Hypothermia: Severe.** Rectal temperature below 30°C
  - i. Signs/symptoms: unconscious, pupils dilated, faint or absent heartbeat.
  - ii. Treatment: transported to a medical facility by emergency vehicle. The trunk must be rewarmed and continuous CPR may be necessary; handle the patient with care to avoid arrhythmias.
- d. **Frostbite** occurs when there is cold-induced peripheral vasoconstriction, leading to tissue freezing.
  - i. Prevention: appropriate clothing—mittens, footgear with dry wool or polypropylene socks, etc.

- ii. Treatment: leave parts frozen until thawing can be done without the possibility of re-freezing. Keep entire body warm. Avoid physical contact between injured tissue and surroundings. Thaw slowly and gently in warm water, 37°–40°C.
- e. Prevention of cold injuries
  - i. Administrative
    - Plan adequately to avoid wind-exposed race courses.
    - Caution athletes concerning wind-chill factor, adequate clothing, etc.
  - ii. Athlete preparation
    - Acclimatise to cold conditions—10 days is an ideal acclimatisation period.
    - Ensure adequate nutrition and use muscle glycogen loading to maximise heat production. Avoid caffeine and alcohol. Hydrate before and during a race.
    - Wear layers of material that will draw sweat from the skin and allow evaporation. Wear head cover.
    - Control pace to avoid late slowing and reduced heat production.

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# ALTITUDE

Altitude adversely affects performance in aerobic events (i.e., those lasting more than two minutes), because the partial pressure of oxygen decreases as barometric pressure falls. This leads to a decline in pulmonary diffusion of oxygen into the blood. A measurable effect on maximal oxygen uptake ( $\dot{V}O_{2\max}$ ) can be seen at elevations as low as 1524 m (5000 ft).

## A. Environmental Conditions at Altitude

### 1. Atmospheric Pressure

Atmospheric pressure decreases as altitude increases, but the percentage of gases in the air remains constant. Air always contains 20.93% oxygen, 0.03% carbon dioxide, and 79.04% nitrogen. The pressure that oxygen molecules exert ( $PO_2$ ) is directly related to the atmospheric pressure ( $P_b$ ). This change in the partial pressure of oxygen directly affects the transfer of oxygen between the lungs and the blood, and between the blood and the tissues (Table 11-4).

Table 11-4.Changes in the partial pressure of oxygen with increasing altitude.

Altitude	$P_b$ (mm)	$PO_2$ (mm)
0 (sea level)	760	159.2
1000	674	141.2
2000	596	124.9
3000	526	110.2

### 2. Air Temperature

Air temperature decreases about 1° C for every 150 m of ascent. This lower temperature also reduces the relative humidity of the air, and increases water losses by evaporation from the skin and the lungs. The increased respiratory rate and loss of respiratory water in dry air can lead rapidly to dehydration, especially during exercise.

### 3. Solar Radiation

Solar radiation increases at altitude, as less ultraviolet light is blocked by the reduced atmosphere and the lower water vapour pressure.

## B. Physiological Responses to Altitude

### 1. Respiratory Responses

#### a. Ventilation

As the partial pressure ( $PO_2$ ) of oxygen decreases, more air must be taken in to provide adequate tissue oxygenation. This increase in ventilation reduces the amount of alveolar and blood carbon dioxide, leading to respiratory alkalosis. The kidney compensates by excreting more bicarbonate ion, decreasing the blood's buffering capacity and reducing the alkalosis (compensated respiratory alkalosis).

#### b. Pulmonary Oxygen Diffusion

Oxygen diffusion across the alveolar-capillary membrane is dependent upon alveolar  $PO_2$ . This decreases as altitude increases, leading to a decrease in oxy-hemoglobin saturation. At sea level hemoglobin is 98% saturated, but this falls to 92% at 2400 m (8000 ft).

#### c. Muscle Oxygen Gas Exchange

The pressure gradient between blood and muscle oxygen concentration is 74mm Hg at sea level (94mm–20mm). This gradient is the major factor responsible for driving tissue oxygenation. At 2400 m the arterial  $PO_2$  is about 60mm Hg, while tissue  $PO_2$  remains at 20mm Hg, a gradient of only 40mm Hg, or a decrease of nearly 50%.

### 2. Cardiovascular Responses

#### a. Blood Volume

Plasma volume decreases soon after altitude exposure, and levels off after a few weeks. This leads to an increased red cell concentration (hematocrit), aiding oxygen delivery to tissues. Plasma volume is gradually restored, and erythropoietin from the kidney stimulates red cell production.

#### b. Cardiac Output

Cardiac output must increase at altitude to compensate for the reduced  $PO_2$  and decreased oxygen delivery to tissues. Initially this is accomplished by an increase in heart rate, as stroke volume is lower due to the decreased plasma volume. After a few days, tissue oxygen extraction improves (increased a-v  $O_2$  difference) and this reduces the cardiac demands. At maximal workloads, maximal stroke volume, heart rate, and tissue oxygen diffusion are reduced, thus total maximal  $VO_2$  and aerobic work are diminished.

### 3. Metabolic Adaptations

As oxidative pathways are limited at altitude, there is a shift toward anaerobic energy sources. At any given work level, lactic acid production is higher than at sea-level. However, at maximal workloads lactic acid is lower, possibly because work levels are too low to maximally activate all energy systems.

#### 4. Maximal Oxygen Uptake

Maximal oxygen uptake measures the ability of the body to take in, transport, and utilise oxygen. It decreases as altitude increases, but does not begin to fall until atmospheric  $PO_2$  drops below 125 mm Hg. This occurs at an altitude of about 1600 m (5250 ft).  $\dot{V}O_2$  max. is related to the decline in the barometric pressure and the partial pressure of oxygen ( $PO_2$ ).  $\dot{V}O_2$  max. decreases 11% for each 1000 metres above the 1600 metre level.

### C. Adaptations to Chronic Altitude Exposure

#### 1. Blood

Erythropoietin stimulates red cell production, and eventually a higher hemoglobin and hematocrit. Adequate dietary iron is essential to meet this demand for increased erythropoiesis.

#### 2. Muscle

Muscle cross-sectional area may decrease with chronic altitude exposure, though capillary density increases to deliver blood to the tissues. At very high altitudes (over 2500 m), levels of muscle enzymes decline, so that muscles are less able to generate ATP aerobically or anaerobically.

#### 3. Cardiorespiratory

Ventilation is stimulated by the hypoxia of altitude. This causes carbon dioxide removal and respiratory alkalosis. Bicarbonate is excreted and remains low, decreasing buffering capacity.

Muscle oxygen uptake decreases at altitude, and improves little with prolonged exposure. This may be due to the profound hypoxia that occurs during exercise at altitude and the consequent inability to train at an adequate intensity and volume.

### D. Effects on Training

Many athletes live at altitude, and others elect to train there in hopes of enhancing their performance at lower elevations. However, maximal aerobic capacity and submaximal aerobic training pace decrease at altitude, especially above 2500–3000 m. Prolonged stays at altitude may be detrimental to high-intensity endurance performance, so altitude training should be interspersed with periods near sea level.

A number of studies have shown that a programme of altitude exposure (hypoxia) interspersed with periods of near-sea-level exposure to permit intense training, when carried out for 3–4 weeks, enhances sea-level performance. This is most likely due to an erythropoietin-induced increase in red cell mass and aerobic capacity, although improved running economy may be a factor.

### E. Preparation for Competition at Altitude

Many major competitions, including the Olympic Games (1968) and the World Championships (1997) are conducted at altitude. While this provides an advantage

for sprinters, hurdlers, and jumpers, endurance events of 800 metres and longer are adversely affected. Therefore, a period of adaptation to altitude is essential in preparation for competition, if one lives at sea level. There is still much to be learned about optimal preparation, but a few principles seem to be well accepted:

1. The adaptation period at altitude should be at least 3 or 4 weeks prior to the start of competition.
2. Athletes should be in good general health, free of medical and orthopedic problems, and not iron deficient prior to beginning altitude training. Iron supplementation should be used if ferritin levels are low.
3. Training volume, intensity, nutrition, and health should be carefully monitored during the training and competition processes.
  - a. During the first week, aerobic adaptation should be stressed. Training volume should be moderate, about 75–80% of sea-level loads.
  - b. Intensity should increase in the second week, with mixed aerobic and anaerobic sessions.
  - c. During week three, emphasis is on maintaining speed, with aerobic work at the highest levels possible. Intensity of runs may be maintained by increasing rest time between interval runs.
  - d. Week four is for tapering and recovery prior to the start of competition.

## F. Altitude Illness

Rapid ascent to elevations of 1500m or more, especially above 2400m (8000 ft), may result in acute “mountain sickness” or, rarely, a severe altitude illness syndrome, such as high altitude pulmonary oedema (HAPE), or high altitude cerebral oedema (HACE). *Caution:* Athletes with sickle cell trait or G-6-P deficiency may sustain a life-threatening crisis with rhabdomyolysis when exposed to high altitude, especially with the added stress of exercise without prior adaptation.

Symptoms of altitude illness include headache, nausea, lethargy, anorexia, vomiting, and disturbed sleep patterns. Symptoms may begin within hours of ascent, peak in 1–2 days, and generally resolve in 3–4 days.

High altitude pulmonary oedema (HAPE) is a medical emergency. Symptoms and signs include those of altitude sickness plus cough, dyspnea, frothy sputum, chest pain, tachypnea, respiratory distress, and pulmonary rales. HAPE occurs more often in young, active people following heavy exercise in cold air.

High altitude cerebral oedema usually occurs after rapid ascent to 4000 m or more. Signs and symptoms include severe headache, ataxia, confusion, blurred vision, and altered consciousness. Rapid removal to a lower altitude is essential, plus oxygen and IV glucocorticoids.

The following may help prevent altitude illness:

1. A gradual ascent of no more than 300 m–600 m per day over 2400 m should be made.
2. Acetazolamide (Diamox) may be helpful prophylactically.

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# AIR POLLUTION

## A. Air Pollution

Athletes exercising in urban environments may be exposed to a variety of pollutants that may affect performance. The most common atmospheric pollutants include carbon monoxide, ozone, particulate matter, sulfur oxides (SO<sub>x</sub>), nitrogen oxides (NO<sub>x</sub>), and peroxy-acetylnitrate (PAN). The “pollution index,” calculated as pollutant concentration  $\times$  ventilation volume  $\times$  exposure time, is the best way to monitor potential threats to athletes.

- **Carbon monoxide (CO)** readily combines with hemoglobin to reduce tissue oxygenation, increase cardiac work, and impair psychomotor function. It is insidious, as it causes no local irritant symptoms to the airways.
- **Oxidants (ozone)** cause tracheal and bronchial irritation, chest tightness, and induce bronchospasm and reduced lung function (decreased FEV<sub>1</sub>). They affect endurance performance at levels of 0.1-0.3 ppm. Asthmatics may be more adversely affected and will need more thorough management.
- **Particulate matter** is composed of solid and liquid droplets, and may contain acids (nitrates and sulfate), organic chemicals, allergens (pollens and molds), metals, soil, and dust. Particles are less than 10 microns in size: “fine particles are defined as less than 2.5 microns, while “course” particles are 2.5–10 microns. High levels of particulate matter may affect the health of older people, increasing morbidity and mortality of those with pulmonary and cardiac disease, as well as affect young people with asthma. Long-term exposure may lead to a permanent decrease in lung function and chronic bronchitis.
- **Sulfur oxides (SO<sub>x</sub>)** irritate the upper airway, causing transient bronchospasm. They are a significant problem for asthmatics, even at low concentrations.
- **Peroxyacetylnitrate (PAN)** and **Nitrogen oxides (NO<sub>x</sub>)** cause symptoms similar to those triggered by ozone.

### 1. Prevention/Management

#### a. Administrative

Schedule events at locations and at times when pollutants are at the lowest levels—away from traffic, near parks and oceans. To minimise ozone exposure, race early in the AM or late PM.

#### b. Athlete Adaptation

Adaptation to ozone and sulfur oxide may occur, with less bronchospasm and irritative symptoms. Asthmatics may be treated with cromolyn sodium, and aerosol adrenal corticoids and/or beta-2 agonists. “Normal”

athletes may develop exercise-induced asthma (EIA) and require beta-agonist therapy.

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## JET LAG

Circadian rhythmic oscillations affect numerous physiologic functions that contribute to athletic performance. Circadian rhythms are expressed as oscillations in physiologic systems (body temperature, heart rate, hormone secretory rates), and responsiveness to internal stimuli (neuro-transmitters, electrolytes, metabolic substrates) or external stimuli (environmental factors, food, drugs, other stressors). Circadian rhythms are synchronised by periodic environmental changes (primarily the light-dark cycle), social interactions, and other environmental factors.

### A. Effects on Performance

Athletic performance can be affected by: 1) the time-dependent changes in circadian peak to trough, and amplitude of physiological variations, and by 2) disruption of circadian rhythms due to trans-meridian travel and subsequent changes in the sleep-wake cycle (i.e., “jet lag”). Circadian dysrhythmias (“jet lag”) can result in a wide variety of symptoms, including fatigue, insomnia, sleep disturbances, headaches, irritability, altered gastro-intestinal motility (constipation), and impaired athletic performance.

Factors that may affect the degree of circadian dysrhythmicity include the rapidity of travel and number of times zones crossed, direction of flight, rate of adaptation of various systems, synchroniser intensity, diet, and individual factors (personality, age, gender, chronotype).

#### 1. Distance and Speed

Crossing as few as two or three time zones may affect performance, but in general the more time zones crossed, the greater the disruption in circadian rhythms. Obviously, rapid travel by jet plane is more disruptive than intermittent or slower means of travel.

#### 2. Direction of Travel

East-bound travel is more disruptive than west-bound travel and recovery takes longer. This is because human physiological systems prefer a circadian cycle longer than 24 hours, and thus adapt better to a phase delay (lengthening) than to a phase advance (shortening). Physiological systems adapt 30–50% faster to westward travel than to eastward travel.

#### 3. Rate of Adaptation

Different physiological systems adapt to time-zone changes at different rates. Those that respond to environmental cues, such as heart rate, adjust more rapidly than internally controlled systems such as core temperature. The specific systems responsible for decreased athletic performance have not been identified, but are probably related to alterations in the sleep-wake cycle and the body temperature circadian cycle.

#### 4. Synchroniser Intensity

Appropriate exposure to external synchronisers, such as daylight, regular meals, social interactions, moderate physical activity, and the establishment of regular sleep-wake cycles can help shift circadian rhythms so that they are in phase with the local environment.

#### 5. Diet

Different foods cause different bodily reactions and can affect whether we feel sleepy or awake. Starchy and sweet foods provide quick but transient energy. They stimulate the indoleamine metabolic pathway and encourage higher concentrations of tryptophan, an essential amino acid, to enter the brain and be converted to sleep-inducing serotonin. As serotonin levels rise, sleep comes more readily. Thus, starchy and sweet foods ultimately tend to make one feel lethargic and can aid in sleep.

In contrast, high protein foods help stimulate the “get-up-and-go” catecholamine pathway (adrenaline pathway, starting from amino acid tyrosine and resulting in the synthesis of the neurotransmitters dopamine, norepinephrine, and epinephrine) and tend to make us more alert.

Thus, what and when one eats helps the biological clock function. Adjustments triggered by diet help the body adapt to travel across time zones by contributing, along with other “zeitgeber” (see below), to a shift of the biological from home time to destination time.

#### 6. Individual Differences

Individuals differ considerably in their ability to adapt to changes in circadian rhythms. About 20–30% have little difficulty, while a similar percentage never adjusts well. In general, the “lark” or morning type adapts poorly to westward travel, while the “owl” (extrovert) adapts less well to eastward flight.

Introverted and highly neurotic persons have more difficulty adjusting to jet-lag. Highly motivated individuals (such as athletes) usually can overcome any tendency to performance decrement through extra effort.

#### 7. Minimising Jet Lag’s Effects

Everyone has an internal “biological clock” guiding daily life. This clock prepares the digestive system to receive food at normal times and causes production of digestive juices even before we smell food. It also helps regulate energy, strength, and alertness, and causes one to become tired about the same time each evening.

Important steps to reduce jet lag involve adjustment of time cues, or “zeitgeber” (pronounced “tseitgaybur”). Zeitgeber are activities and inputs that help the body interpret its environment, determine whether it is day or night, and decide what activities (including conscious, unconscious, and biochemical activities) are appropriate for the time. Major zeitgebers are diet, light, exercise, drugs, and social interaction. Adjustment of these cues before and during travel help the body reset its internal clock more quickly and easily.

The body's sense of time is affected by both internal and external factors. The biological clock is coordinated by hormones, which are produced by endocrine glands in varying amounts and kinds throughout the day. Hormones help determine alertness, muscle proficiency, and mood, and their interplay with external time cues determines how strong the body clock's signals will be.

There are a number of steps that can help minimise the effects of jet lag (see Appendix 10, *Recommendations for Minimising Jet Lag*, for a summary of recommendations):

- a. Arrive at the competition site as early as possible. Allow one day for each time zone of eastward flight, and 0.6 days for each zone of westward travel.
- b. Adjust eating, sleeping, work, and training schedules in the direction of the new time zone, by 1–2 hours each day over a 4–5 day period. This may not be practical in a family or work situation.
- c. Arrange flights so as to arrive close to local bed-time.
- d. Eat high protein /low carbohydrate meals for 3 days prior to flight, and during flight. Try to follow a meal schedule that will be used at your destination. After arrival, starting with breakfast and lunch, eat high protein/low carbohydrate meals, and use tea or coffee to enhance synchronisation.
- e. Avoid alcohol as a sleep-inducer, as it interferes with REM sleep and delays sleep-wake cycle adaptation. Do not use coffee, tea, or caffeine-containing drinks during flight. Drink large amounts of caffeine-free drinks (juice, water) to prevent dehydration.
- f. At departure, set watch to destination time, and adjust sleep, meals, and other activities accordingly. East-bound travellers should avoid bright lights, movies, and socialising until breakfast of the day after arrival.
- g. Upon arrival, immediately adjust schedule (social contacts, bright sunlight, meals, training, etc.) to local time. Maintain regular sleep-wake schedules.

Stressors, such as competition anxiety, coping with climate and food changes, and other environmental and social factors can exacerbate the effects of “jet-lag.” Dealing successfully with these elements may minimise the effects of circadian dysrhythmias.

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