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EXERCISE IN THE COLD

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EXERCISE IN THE COLD

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Humans have developed the technical capability to protect themselves, from a thermoregulation point of view, against almost any terrestrial extreme. With respect to the extreme cold, it is understood that the best technique to ensure survival resides in enhancing one's insulation, or reducing heat losses, by using a wide variety of new high-tech clothing and equipment. Excellent examples of the use of such technologies are displayed by winter Olympians, skiers, trekkers, joggers, and, in particular, polar adventurers. However, there are situations where technologic and behavioral strategies to increase insulation are simply not available, not appropriate, have lost their effectiveness, or have already been maximized. Faced with high rates of heat losses, survival would then reside in one's physiologic ability to maintain body temperatures via vasoconstriction and an enhanced metabolic heat production. This chapter focuses on thermogenesis during cold exposure, key factors that influence one's resistance to cold, hypothermia and its treatment, and finally implications for the winter athlete.

THERMOGENESIS TO SUSTAIN HOMEOTHERMY

Components of Thermogenesis

Although no one debates the importance of thermogenesis, or production of heat, in the cold, the various components and mechanisms involved in daily thermogenesis are rarely highlighted and often misconstrued. Resting metabolic rate (RMR) accounts for the major portion of the daily energy expenditure while resting at thermal neutrality (TN) (Table 1). The thermic effect of food, exercise-induced thermogenesis, and thermoregulatory thermogenesis (TT) are the other three components of daily thermogenesis. Briefly, the thermic effect of food is made up of obligatory thermogenesis (a phase formerly known as the specific dynamic action, related to the absorption, breakdown, and storing of ingested nutrients) and facultative thermogenesis (a phase related to sympathetic nervous system activity). Exercise-induced thermogenesis represents the additional energy expenditure associated with standing, walking, and exercising. This component can vary from a negligible portion of the daily energy expenditure of a bed-ridden patient, to its highest in a marathoner, where such activity can burn as many as 8,400 kilojoules (2,000 kcal). The last component is TT. At thermal neutrality, TT is negligible. But in emergency situations where cold exposure is lengthy, energy expenditure can be as high as that of the marathoner mentioned. This is explained by the fact that although the cold-induced increase in metabolic rate can be relatively small compared to that of exercise, TT is increased *continuously*, in contrast to the *intermittent* nature of exercise.

Table 1 Components of Thermogenesis

Resting metabolic rate (RMR)
Thermic effect of food (TEF):
Obligatory thermogenesis
Facultative thermogenesis
Exercise-induced thermogenesis
Thermoregulatory thermogenesis:
Shivering
Nonshivering

TT can originate from shivering and/or nonshivering thermogenesis. Shivering is a form of involuntary rhythmic muscular contraction where no useful work is done. It is triggered by acetylcholine, and involves cellular processes similar to those occurring in other forms of skeletal muscle contraction. It is important to point out here that the conversion of chemical energy to mechanical work, which includes shivering, is not an efficient process, since about 75% of its energy is released as heat and only 25% is converted to high-energy compounds. So many muscle groups are recruited even during low intensity shivering that increased energy is demanded to fuel this muscular activity. Metabolic rate can be increased by up to five times RMR during shivering. Whether nonshivering mechanisms make a significant contribution to TT in humans and in which particular tissues, is unclear. Mechanisms unrelated to shivering can include ion pumping (i.e., Na/K pump) and futile metabolic cycles.

Fuel Utilization

Exercise and cold exposure are two distinct physiologic conditions where energy expenditure is enhanced: they have many similarities, in particular with respect to metabolic fuel utilization. In rats, cold exposure greatly enhances turnover, tolerance, oxidation, and uptake of plasma glucose. It has even been suggested that cold exposure stimulates peripheral tissue glucose uptake primarily via insulin-independent pathways, possibly similar to the well-known "insulin-like" effect of exercise. Increments in lipid metabolism and, in particular, FFA turnover have also been documented. In contrast, aside from changes in circulating substrates, very little was known about fuel metabolism in cold-exposed humans until very recently.

Using the well-known indirect calorimetry and nonprotein respiratory exchange ratio technique, it has been demonstrated recently that the cold-induced increase in heat production (fasting seminude subjects at rest for 2 hours at 10°C, 1 $m \cdot s^{-1}$ wind) is associated with 588% and 63% increases in carbohydrate (CHO) and lipid oxidation, respectively, and an unchanged rate of protein oxidation. The proportion of substrate utilization is also dramatically altered. At TN, the greatest proportion of energy expenditure is derived from lipids (59%), but in the cold, there is a marked shift to CHO (51% of total energy expenditure). Entirely similar results have been observed with respect to CHO oxidation during cold water immersion. Attempts to determine the source of the substrates oxidized in the cold have been particularly revealing.

In humans, the utilization of circulating CHO is accelerated in the cold even in the presence of reduced blood insulin levels, suggesting a greater sensitivity of peripheral tissues to insulin. In addition, cold exposure increases the utilization of intramuscular glycogen (Fig. 1). This can be a factor limiting cold tolerance since low glycogen levels prior to cold water immersion (90 minutes in stirred 18°C water) significantly accelerate body cooling. Faster body cooling has also been observed

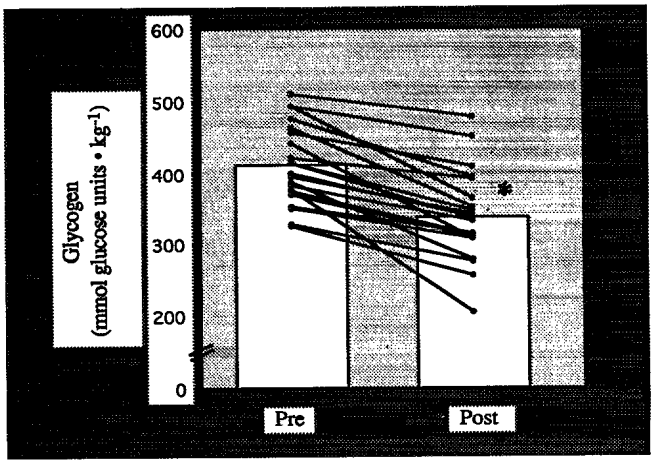

Figure 1 Muscle glycogen levels before and after cold water immersion (90 minutes at 18°C seminude). (From Martineau L, Jacobs I. Muscle glycogen utilization during shivering thermogenesis in humans. J Appl Physiol 1988; 65:2046–2050; with permission.)

when hypoglycemia is induced in cold-exposed individuals, suggesting that both low blood glucose and low muscle glycogen reserves should be avoided in the cold. It certainly appears as though there is a CHO dependence during TT, although the exact reasons remain to be elucidated.

What is the origin of the fatty acids oxidized in the cold? Recent cold exposure experiments have shown that blocking fatty acid release from white adipose tissue did not alter metabolic rate, lipid oxidation, or the rate of body cooling, suggesting that fatty acids from another source were being oxidized. Since plasma triglycerides (TG) were already known to represent another important source of fatty acids for oxidation during prolonged exercise, it was hypothesized that the same concept could apply to cold exposure. Unfortunately, plasma TG clearance was not found to be affected by the cold. It remains to be determined whether intramuscular TG (the only other potential source of fatty acids) represents a key source of readily available fatty acids for oxidation in the shivering muscles.

FACTORS THAT INFLUENCE RESISTANCE TO COLD

Heat Balance

It has been mentioned earlier that to prevent hypothermia, or at least delay its onset, it is essential that heat production be increased and/or heat losses be reduced. How do these *heat*-related concepts fit together and what is the link between body *heat* and body *temperatures?* The concept of whole body heat exchange is detailed in the heat balance equation below, which incorporates all routes for the gain and loss of heat [all terms are in $W \cdot m^{-2}$ of body surface area (1 W = 1 $J \cdot s^{-1}$)]:

$$\dot{S} = \dot{M} - (\dot{R} + \dot{C}) - \dot{E}_{persp} - \dot{C}_{resp} - \dot{E}_{resp}$$

where $\dot{M}$ is the metabolic rate, $\dot{R} + \dot{C}$ is the rate of dry heat exchange by radiation and convection, $\dot{E}_{persp}$ is the rate of evaporative heat loss from the skin, $\dot{C}_{resp}$ and $\dot{E}_{resp}$ are respectively the rates of convective and evaporative heat loss by the respiratory tract and $\dot{S}$ is the rate of heat debt (determined as the minute by minute balance of heat gains and heat losses). A negative $\dot{S}$ signifies a negative heat storage or a positive heat debt. Integrated over time, a total S (sum of instantaneous $\dot{S}$ in $W \cdot m^{-2}$, converted to kcal or kilojoules) will eventually correspond to a change in core temperature (T_{core}). Both the total S and the change in T_{core} have been used as indices of cold tolerance or resistance, although S is inherently a more robust measure, since it is based on more parameters than T_{core} alone.

As knowledge of the utilization of metabolic fuels during cold exposure expanded, it became increasingly evident that an enhancement of metabolic heat production could ensure warmer body temperatures by reducing the cumulative heat debt, therefore enhancing the individual's resistance to cold. Three types of interventions may be considered: the use of pharmacologic agents, dietary supplements, and exercise.

Pharmacologic Agents

The use of pharmacologic agents to enhance cold resistance is not a new idea. As early as 1942, it was reported that the ingestion of caffeine reduced the drop of mean skin temperatures (T_{sk}) in men who were exposed to cool ambient temperatures. Relatively similar results were found with the use of thermogenic mixtures of ephedrine and xanthines. Recent studies have established that β-adrenergic drugs (such as ephedrine) and xanthines (such as caffeine and theophylline) are effective thermogenic antiobesity agents. Studies involving mixtures of ephedrine/caffeine and ephedrine/caffeine/theophylline have documented a similar slowing of the drop of T_{sk} in the cold, accompanied by a warmer T_{core} and a reduced heat debt, these changes being consequent to a greater substrate oxidation and metabolic rate (Figs. 2 and 3). Although ephedrine/xanthine mixtures increase cold resistance, further work is required to determine their effectiveness during longer exposures and at deeper levels of hypothermia.

Dietary Supplements

The influence of dietary supplements in the cold has received a fair amount of attention, probably due to an abundance of literature about their usefulness during exercise. Moreover, energy substrate mobilization has been postulated as one factor limiting for TT. It was thus thought that enhancing energy substrate mobilization by feeding could enhance cold resistance. Although this theory seems in accord with the known facts in experimental animals, the corresponding metabolic data in humans is surprisingly unconvincing. We carefully reinvestigated the links between energy substrate mobilization, TT, and cold tolerance. Ingestion of various dietary supplements (1,400 kJ or ~ 340 kcal of high-CHO or pure CHO) during either mild or more severe cold significantly enhanced energy substrate mobilization, exactly as expected. However, there were no associated changes in T_{core}, T_{sk}, M, or heat debt. We were unsure whether the above dosage of substrates was optimal, and a higher dose was tested in another study where cold-exposed men ingested as much as 3,000 kJ (710 kcal) of a high-CHO supplement in an effort to optimize substrate mobilization and thermogenesis. This high-energy supplement again had no beneficial effect on any thermal parameter, though it did increase CHO mobilization and oxidation at the expense of lipid mobilization and oxidation.

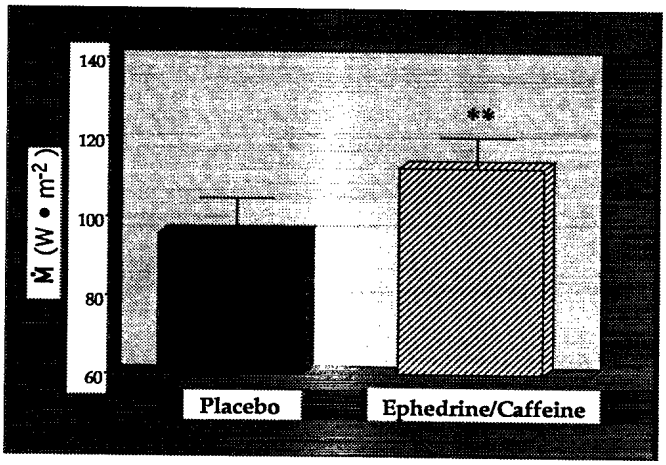

Figure 2 Average energy expenditure in cold air (3 hours at 10°C, 1 $m \cdot s^{-1}$ wind, seminude) following the double-blind ingestion of either a placebo or an ephedrine/caffeine capsule (From Vallerand AL, et al., J Appl Physiol 1989; 67:438–444; with permission.)

We hypothesized that dietary supplements might still have a beneficial effect at the higher metabolic rate associated with exercise in the cold. Results showed that ingesting a high-CHO supplement during intermittent exercise in the cold produced no beneficial effect on body temperatures or heat debt; it only increased CHO mobilization and oxidation at the expense of lipid mobilization and oxidation, without altering thermogenesis. Therefore it seems that dietary supplements alone are not as effective as originally thought in enhancing TT and cold resistance. Although energy substrate utilization is required to fuel thermogenesis in the cold, energy substrate mobilization does not appear to be a limiting factor for humans under normal conditions. Whether similar conclusions would be reached during conditions of energy deficiency in the cold, remains to be demonstrated.

Exercise

Although significant metabolic heat is produced through shivering alone, exercise-induced thermogen-

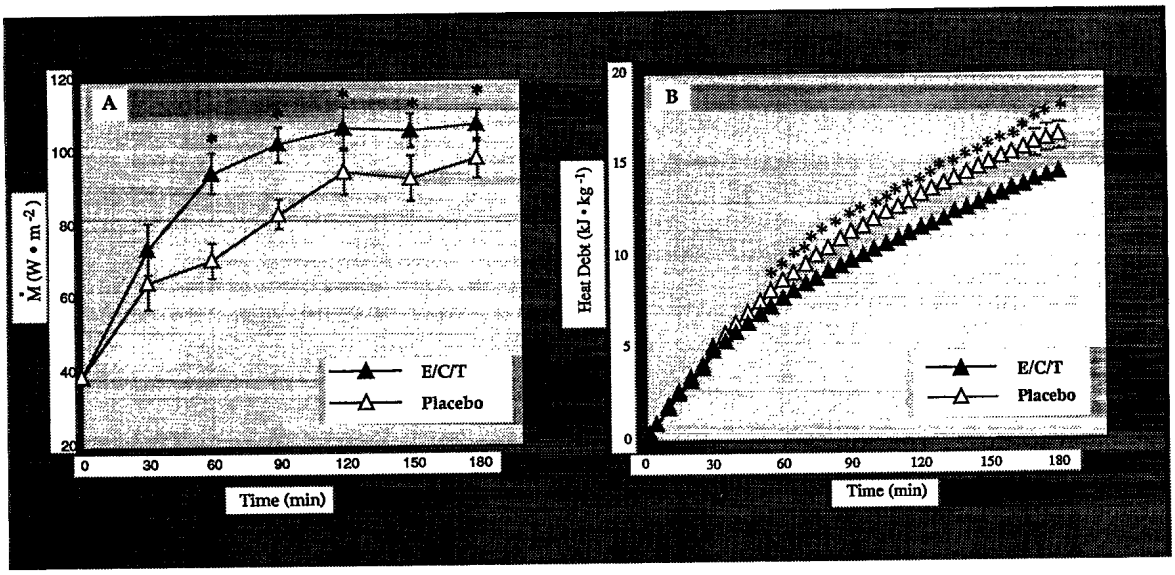

Figure 3 Average energy expenditure in cold air (3 hours at 10°C, 1 $m \cdot s^{-1}$ wind, seminude) following the double-blind ingestion of either a placebo or an ephedrine/caffeine/theophylline capsule (ECT). (From Vallerand AL. Effects of ephedrine/xanthines on thermogenesis and cold tolerance. Int J Obes 1992; 17:S53–S56; with permission.)

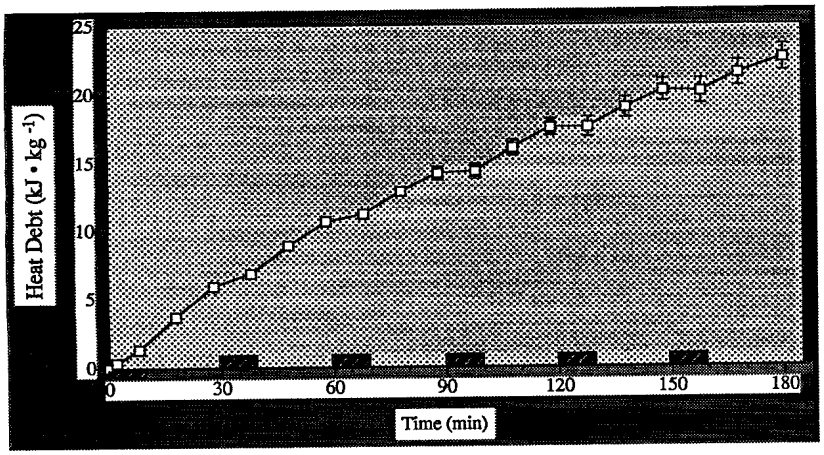

Figure 4 Cumulative heat debt in the cold air (3 hours at 0°C, 1 $m \cdot s^{-1}$ wind) where intermittent exercise was performed (alternating 10 minutes treadmill walking, 20 minutes resting). Dark shaded areas on the x-axis represent the exercise periods.

esis can also become quite effective in defending the body against cold. Good examples are found in the swimmers who have crossed the English Channel (~ 18 hours at 16°C), the North American Great Lakes (~ >20 hours at 15° to 18°C), or even Glacier Bay, Alaska (~ 0.5 hours at 2° to 3°C). What is remarkable is that these feats have usually been performed while wearing minimum insulation! It is in our interest to try to explain these remarkable thermal performances, because there are unfortunately more unsuccessful immersions in cold water than the above hand-picked examples. First, it is likely that these participants, experienced and highly-trained swimmers, were somewhat habituated to cold water. Secondly, they probably had a substantial amount of insulative body fat. And thirdly, they probably knew exactly what their optimal swimming speed was in order to maintain body heat balance under the prevailing environmental conditions. The latter is not easily accomplished because water has a specific heat about 4,000 times that of air and a thermal conductivity about 25 times greater. When body heat travels from the core to the skin surface, heat is rapidly transferred to the water, quickly bringing the skin very close to water temperature. Any exercise intensity that inappropriately increases heat losses, particularly in cold water, accelerates body cooling.

In direct contrast to exercise in cold water, exercise in cold air consistently provides an optimal means of maintaining thermal balance. A good example is depicted in the experiment of Figure 4, where each intermittent bout of exercise almost flattened the heat debt profile in the cold (3 hours at 0°C), whereas the heat debt increased during each resting period. Nude men can maintain an elevated T_{core} when exercising at 10°C and can maintain thermal balance down to an air temperature of -10°C when the thermogenesis of mod-

erate exercise exceeds heat losses. In air as cool as -30°C, exercise-induced thermogenesis can sustain a stable T_{core} when wearing heavy protective clothing. The problem with such clothing is the possible accumulation of sweat in the garment, the consequent high evaporative cooling and loss of insulating properties. Exercise in the cold can either accelerate or reduce the drop in T_{core}, depending on the balance between the increments in heat production versus the corresponding increments in heat losses. Another important aspect of thermal protection by exercising in the cold is simply how long the exercise can be maintained.

Interactions between exercise and cold stress depend greatly on the intensity of the cold exposure. Some situations result in combined shivering and exercise-induced thermogenesis. If the cold stimuli are strong enough and the exercise-induced thermogenesis is insufficient, additional thermogenesis from shivering helps to maintain T_{core}. For this reason, exercise in the cold delays the onset of any increase in plasma lactate, reduces plasma lactate levels during submaximal exercise, and increases plasma norepinephrine levels, in contrast to the values observed during exercise at TN. Further, the rate of oxygen consumption is higher during light exercise in the cold and part of the extra work is fueled by a greater glycogen utilization.

HYPOTHERMIA

Accidental hypothermia can be classified according to its severity and duration. Experts usually recognize mild hypothermia as starting at a T_{core} of about 35°C, moderate hypothermia between 30° and 34°C and severe hypothermia at <30°C. In the same fashion, the description of acute hypothermia is reserved for a duration of <6 hours, subacute hypothermia persists for 6 to 24 hours and chronic hypothermia for >24 hours. T_{core} is best determined with a rectal, oesophageal, or tympanic probe. Under field conditions, however, one may have to rely on oral readings. Nevertheless, efforts should be made to avoid being the author of an important case report where the victim's T_{core} could not be determined properly for lack of a suitable T_{core} probe. Although most electronic hand-held temperature indicators work very well and seem indestructible, they may become inaccurate or fail to function if left in the cold.

If T_{core} cannot be assessed, you can suspect mild hypothermia if your patient shivers vigorously and is still alert. However, if shivering is weak, if they make little attempt to protect themselves from the cold, and if they show profound mental confusion, you can suspect moderate to severe hypothermia; such patients must be treated as a medical emergency. There is much interindividual difference in response. One individual with 32°C of T_{core} may be conversant, whereas another will already be somnolent at 35°C. There are further differences in T_{core} according to the site chosen for measurement. Knowing these limitations, experts generally agree that hypothermia victims are likely to demonstrate the following symptoms, at a T_{core} of:

- <35°C: conscious and alert, with greatly increased shivering and vasoconstriction
- 32° to 34°C: conscious with mild/moderate clouding of mental capacities, but a very good chance of recovery.
- 30° to 32°C: a matter of serious concern due to severe clouding or loss of consciousness, and a slow but progressive reduction in shivering intensity.
- 30°C: unconscious, with acidosis, slow respiration, and limited heat production; the heart muscle does not respond either to medication or to electrical stimulation; remember that a 2°C afterdrop induced by rapid rewarming attempts can bring T_{core} to a dangerously low 28°C.
- 28°C: ventricular fibrillation is so common that it represents the main cause of death during the rewarming of such victims.
- 25°C: the patient slowly changes from homeotherm to poikilotherm, due to a marked depression in both heat production and heat conservation mechanisms: the victim is now at the mercy of the environment.

Note that low body temperatures do not themselves cause death, since therapeutic hypothermia to about 18° to 20°C has been used in surgery. It is therefore essential to re-warm and resuscitate any hypothermic victim before they are legally pronounced dead.

Treatment

Experts still debate the optimal method of rewarming following hypothermia. Although the list of possible methods is rather long, they can be divided into three general classes: passive rewarming, active external rewarming, and active internal rewarming. Passive rewarming consists of insulating the patient and letting shivering thermogenesis slowly rewarm a mildly hypothermic victim. Active external rewarming includes such methods as hot baths, radiant heat, hot water bottles, plumbed garments, electric blankets, warm water, and air mattress held at various temperatures and for varying periods of time. Methods of active internal rewarming comprise inhalation rewarming (breathing warm humidified air), warming by IV fluids, irrigation (nasogastric, bladder, pleural, colonic), microwave rewarming (in various volumes and/or temperatures and in varying combinations), and finally, for severe cases, extracorporeal circulation (use of a heat exchanger). In young, healthy and well-motivated experimental subjects, exercise-induced thermogenesis has been employed successfully as a rewarming technique following hypothermia as low as 31.2°C (inclusive of afterdrop). Even though exercise tends to exaggerate the extent of the afterdrop, this is compensated by a faster rate of rewarming thereafter. This is a bold and challenging new approach, since until now it was commonly believed that muscular activity could not be used as a method of rewarming below a T_{core} of 33°C.

RECOMMENDATIONS FOR THE WINTER ATHLETE

The interaction of exercise and cold produces significant physiologic changes that affect the winter athlete. Some recommendations follow:

- Cold weather competitions should be well marshalled so that hypothermic victims can be identified quickly and treated accordingly.
- Individual winter athletes should check weather conditions and dress appropriately to prevent hypothermia. A longer warm-up (15 minutes) can be valuable.
- It is advisable to run with a friend, or at least to inform others about your route or your expected time of arrival.
- The increased glycogen utilization associated with cold exposure could affect the endurance athlete. Further, the continuing demand for CHO in the face of glycogen depletion could lead to hypoglycemia. This will affect significantly not only muscle fatigue, temperature regulation, skilled performance, but also cerebral metabolism. It can also leave the winter athlete vulnerable to injuries.
- The winter athlete must pay attention to the body's warning signals about local cooling. Disregarding pain, numbness, and fatigue can be deadly. The same can be said about sweating in the cold: sweat destroys clothing insulation and is likely to increase evaporative cooling greatly. Persistent shivering after competition or exercise should be interpreted as indicating more extensive core cooling than was thought and should be a reason for concern.
- Since cold-induced diuresis affects blood volume, stroke volume, and heart rate at submaximal work intensities, it is important to stay well hydrated. It is also wise to avoid a negative energy and CHO balance: both have been associated with lessened resistance to cold.
- Both whole body and local cold acclimation are clearly of benefit; beneficial responses develop more readily at rest in the cold than during exercise. Note that the body has to be readapted to the cold every winter, though the extent of such readaptation may vary according to geographic location.
- Finally, hypothermic victims in isolated areas typically struggle until exhaustion in the mistaken belief that their only hope lies in reaching their destination. It is crucial to remember that an emergency bivouac offers a much better chance of survival, preventing exhaustion and further degradation of clothing insulation.

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