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# Human Adaptations to Heat and Cold Stress

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## Summary

Heat acclimation consists of adaptations that mitigate physiological strain of heat stress, which improve thermal comfort and exercise capabilities. Adaptations are induced by repeated heat exposures that are sufficiently stressful to elevate core and skin temperatures and elicit profuse sweating. Most adaptations to daily heat exposure occur during the first four days, and the remainders are complete by three weeks. Heat acclimation mediated adaptations include: lower core temperature, improved sweating and skin blood flow, lowered metabolic rate, reduced cardiovascular strain, improved fluid balance, and increased thermal tolerance (i.e., cellular stress protein adaptations). These adaptations vary somewhat depending if exposed to dry or humid heat.

Adaptations to chronic cold exposure can be categorized into three basic patterns: habituation, metabolic adaptations and insulative adaptations. The exact determinant of which pattern will be induced by chronic cold exposure is unclear, but the magnitude and extent of body cooling, frequency and duration of exposure, and individual factors all influence the adaptive process. Habituation is characterized by blunted shivering and cutaneous vasoconstriction; body temperature may decline more in the acclimatized than unacclimatized state. It is the most common cold adaptation and results from periodic short-term cold exposures. Metabolic adaptations are characterized by enhanced thermogenesis that develops when cold exposures are more pronounced, but not severe enough to induce significant declines in core temperature. Insulative adaptations are characterized by enhanced vasoconstriction and redistribution of body heat toward the shell that develops from repeated cold exposures severe enough to induce marked declines in core temperature.

## Introduction

Humans encounter thermal (heat & cold) stress from climatic conditions, insulation worn and body heat production. Alterations in body temperatures (skin, muscle & core) above and below “normal” levels can degrade exercise performance and cause thermal injury. Humans regulate core temperature within a narrow range (35° to 41°C) through two parallel processes: physiological and behavioral temperature

regulation. Physiological temperature regulation operates through responses that are independent of conscious voluntary behavior, and includes control of: a) rate of metabolic heat production, b) body heat distribution via the blood from the core to the skin, and c) sweating. Behavioral temperature regulation operates through conscious behavior, and includes actions such as modifying activity levels, changing clothes and seeking shelter. For humans, physiological thermoregulation is most important during heat stress and behavioral thermoregulation is most important during cold stress.

Humans demonstrate adaptations to repeated exposure to either heat (85) or cold (105) stress. In general, these adaptations act to defend body temperatures, reduce physiologic strain, and improve comfort / work capabilities and reduce susceptibility to thermal injury. This paper examines human thermoregulation and adaptations to repeated heat or cold stress. Throughout this paper, the terms acclimation and acclimatization will be used interchangeably. Acclimatization develops from challenges in the natural environment, and acclimation develops from experimental exposure to artificial conditions, both elicit similar adaptations. However, acclimatization can reflect adaptations to stimuli besides thermal strain, such as diet and activity level.

### **Adaptations to Heat Stress**

Humans have remarkable ability to adapt to heat stress, and given adequate water and protection from the sun, a healthy acclimated person can tolerate extended exposure to virtually any natural weather-related heat stress (85,97). Heat stress results from the interaction of environmental conditions (temperature, humidity, sun), physical work rate (body heat production) and wearing of heavy clothing / equipment that impedes heat loss. Environmental heat stress and exercise interact synergistically to increase strain on physiological systems (66). This strain is manifested by high skin and core temperatures, excessive cardiovascular strain and reduced performance. Heat acclimation results in biological adaptations that reduce these negative effects of heat stress. One becomes acclimated to the heat through repeated exposures that are sufficiently stressful to elevate both core and skin temperatures and provoke profuse sweating. These biological adaptations occur from integrated changes in thermoregulatory control, fluid balance, and cardiovascular responses.

*Induction & Decay.* The magnitude of biological adaptations induced by heat acclimation depends largely on the intensity, duration, frequency and number of heat exposures (85). Exercise in the heat is the most effective method for developing heat acclimation, however, even resting in the heat results in some acclimation. The full development of exercise-heat acclimation does not require daily 24-h exposure. A continuous, daily 100-min period of exposure appears to produce an optimal heat acclimation response in dry heat (57). Studies examining heat acclimation have generally used daily heat exposures; however, these are not necessary to produce heat acclimation. Fein and colleagues (20) examined the time course of biological adaptations to 10 days of heat exposure, when subjects were exposed to heat (47°C, 17% relative humidity) daily or every third day. Therefore, one group completed the acclimation program in 10 days and the other in 27 days. Both methods were equally effective in producing heat acclimation, but with daily heat exposures it required only one-third of the total time.

Heat acclimation is transient, it gradually disappears if not maintained by repeated heat exposure. The heart rate improvement, which develops more rapidly during acclimation, is also lost more rapidly than thermoregulatory responses. There is no agreement concerning the rate of decay for heat acclimation. Lind (56) believed that heat acclimation might be retained for two weeks after the last heat exposure, and then be rapidly lost over the next two weeks. Williams and colleagues (98) report some loss of acclimation in sedentary individuals after one week, with the percentage loss being greater with increasing time; and, by three weeks losses of nearly 100% for heart rate and 50% for core temperature. Physically trained and aerobically fit persons may retain benefits of heat acclimation longer (73).

*Actions & Mechanisms.* Table 1 provides a brief description of the actions of heat acclimation (86). Heat acclimation improves thermal comfort and submaximal exercise performance. These benefits of heat acclimation are achieved by improved sweating and skin blood flow responses, better fluid balance and cardiovascular stability, and a lowered metabolic rate (41,85).

Heat acclimation does not improve maximal intensity exercise performance. For example, heat stress mediated reductions in maximal aerobic power are not abated by heat acclimation (87). In addition, heat acclimation does not alter the maximal core temperature a person can tolerate during exercise in the heat (69,70). There is evidence, however, that persons who live and train over many weeks in the heat might be able to tolerate higher maximal core temperatures than persons heat acclimated over one or two weeks (82). Other studies (26,75) suggest that successful hot-weather athletes may be able to tolerate higher core temperatures.

**Table 1 - Actions of Heat Acclimation (86)**

<b>Thermal Comfort – Improved</b>	<b>Exercise Performance – Improved</b>
<b>Core Temperature – Reduced</b> <b>Sweating – Improved</b> Earlier Onset Higher Rate Redistribution (Tropic) Hidromeiosis Resistance (Tropic) <b>Blood Flow - Increased</b> Earlier Onset Higher Flow  <b>Metabolic Rate – Lowered</b>	<b>Cardiovascular Stability – Improved</b> Heart Rate - Lowered Stroke Volume – Increased Blood Pressure – Better Defended Myocardial Compliance - Improved <b>Balance - Improved</b> Thirst - Improved Electrolyte Loss – Reduced Total Body Water – Increased Plasma Volume – Increased & Better Defended

Heat acclimation mediates improved submaximal exercise performance by reducing physiologic strain during exercise. The three classical signs of heat acclimation are lower heart rate and core temperature, and higher sweat rate during exercise-heat stress. Skin temperature is lower after heat acclimation than before, and thus dry heat loss is less (or, if the environment is warmer than the skin, dry heat gain is greater). To compensate for the changes in dry heat exchange, there must be an increase in evaporative heat loss, in order to achieve heat balance. After acclimation, sweating starts earlier and at a lower core temperature, i.e., the core temperature threshold for sweating is decreased. Sweating rate is usually increased by the second day of heat acclimation (17,27,70,96,101). The sweat glands also become resistant to hidromeiosis and "fatigue" so that higher sweat rates can be sustained. Earlier and greater sweating improves evaporative cooling (if the climate allows evaporation) and reduces body heat storage and skin temperature. Lower skin temperatures will decrease the skin blood flow required for heat balance (because of greater core-to-skin temperature gradient) and reduce skin venous compliance so that blood volume is redistributed from the peripheral to the central circulation. All of these factors reduce cardiovascular strain and enhance exercise-heat performance.

On the first day of exercise in the heat, heart rate reaches much higher levels than in temperate conditions, and stroke volume is lower. Thereafter, heart rate begins to decrease as early as the second day of heat acclimation. These changes are rapid at first, but continue more slowly for about a week. There are probably numerous mechanisms that participate, and their relative contributions will vary, both over the course of the heat acclimation program and also among subjects (85,97). These mechanisms include: a) improved skin cooling and redistribution of blood volume (81); b) plasma volume expansion (90); c) increased venous tone from cutaneous and non-cutaneous beds (99); and d) reduced core temperature (85). In addition, myocardial changes reported from heat acclimatization include increased compliance (44) and isoenzymes transition reducing the myocardial energy cost (43).

The effects of heat acclimation on stroke volume and cardiac output responses to exercise-heat stress are not clear-cut. For example, two studies (78,101) report increased stroke volume with little change in cardiac output as heart rate fell; but another study (100) reports a decrease in cardiac output, associated with a decrease in "surface blood flow" (estimated calorimetrically) as heart rate fell, and little change in stroke volume; and still another study (102) reports a mixed pattern, with two subjects showing a steady increase in

stroke volume, one a transient increase, reversing after the sixth day, and one showing no increase. The reason for these differences is not clear: Rowell et al. (78) describe dry heat acclimation, and Wyndham (101) and Wyndham and colleagues (101,102) all describe humid heat acclimation.

Nielson and colleagues (69,70) examined stroke volume responses during exercise before and after heat acclimation. One study (69) had subjects acclimate for 9-12 days in hot-dry conditions while performing cycle ergometer ( $60\% \text{VO}_{2\text{max}}$ ) exercise. They reported that during exercise, heat acclimation increased stroke volume and increased cardiac output ( $\sim 1.8 \text{ L/min}$ ). The other study (70) had subjects acclimate for 8-13 days in hot-humid conditions while performing cycle ergometer ( $60\% \text{VO}_{2\text{max}}$ ) exercise. They reported that during exercise, heat acclimation did not alter stroke volume or cardiac output. Both studies reported plasma volume expansion of 9 to 13% with heat acclimation. It seems possible that dry and wet heat acclimation usually increased stroke volume responses, but that improved cardiac output responses are more likely to be observed with dry heat acclimation.

Heat acclimation can alter whole-body (85) and muscle metabolism (103). The oxygen uptake response to submaximal exercise is reduced by heat acclimation (83). Consistent with this is the observation that basal metabolic rate is decreased during warmer months (41). Lactate accumulation in blood and muscle during submaximal exercise is generally found to be reduced following heat acclimation (107). King et al. (50) and Kirwan et al. (51) both observed that heat acclimation reduced muscle glycogen utilization during exercise in the heat by 40-50% compared to before acclimation. Young et al. (107) also observed a significant glycogen-sparing effect due to heat acclimation, but the reduction in glycogen utilization was small, and apparent only during exercise in cool conditions. Glycogen utilization during exercise in the heat was negligibly affected.

*Fluid Balance & Blood Volume.* Fluid balance improvements from heat acclimation include better matching of thirst to body water needs, reduced sweat sodium losses, increased total body water and increased blood volume (59,81). Thirst is not a good index of body water requirements as *ad libitum* water intake results in incomplete fluid replacement or “voluntary” dehydration during exercise-heat stress. Heat acclimation improves the relationship of thirst to body water needs so that “voluntary” dehydration is markedly ( $\sim 30\%$ ) reduced (7,17). Therefore, heat acclimated persons will dehydrate less during exercise in the heat, provided that access to fluids is not restricted. This is an important adaptation as heat acclimation increases sweating rate and if fluid replacement is not proportionately increased then greater dehydration will occur.

Most studies report that heat acclimation increases total body water (81). The magnitude of increase ranges from 2.0 to 3.0 liters or  $\sim 5\%$  to  $7\%$  of total body water. This increase is well within the measurement resolution for total body water (81) and thus appears to be a real physiological phenomenon. The division of the total body water increase between intracellular fluid (ICF) and extracellular fluid (ECF) is variable: studies report that ECF accounts for greater, equal and smaller than its percentage increase in total body water after heat acclimation (81). Measures of ECF have relatively high variability, and therefore trends for such small changes are difficult to interpret. The extent of which ICF increases is unclear because typically it is calculated as the difference between total body water and ECF, and thus measurement variability inherent in both these techniques is compounded in the calculation of ICF. If total body water and ECF increase after heat acclimation, then expansion of blood volume might be expected.

Heat acclimation increases blood volume through differential effects on erythrocyte and plasma volumes. Erythrocyte volume does not appear to be altered by heat acclimation or season (80). Plasma volume expansion is usually, but not always, present after repeated heat exposure and heat acclimation (81). Heat acclimation studies report that plasma volume expansion, generally ranged from  $0\%$  to  $30\%$ , and the magnitude of increase is somewhat dependent on whether the person is at rest or performing exercise, the heat acclimation day and the hydration state when measurements are made (79). Plasma volume expansion seems to be greatest when performing upright exercise on about the fifth day of heat acclimation and when fully hydrated.

The mechanism(s) responsible for this hypervolemia are unclear, but may include an increase in extracellular fluid mediated by retention of crystalloids (primarily sodium chloride) and perhaps an increase in plasma volume selectively mediated by the oncotic effect of intravascular protein (58,59). Heat acclimated persons also exhibit a more stable plasma volume and more consistent intravascular fluid response to exercise-heat stress than do persons who are not heat acclimated (79). The increase in total body

water can be explained in part by increased aldosterone secretion and / or renal sensitivity to a given plasma concentration. Francesconi and colleagues (23) have shown that exercise - heat exposure markedly increased plasma aldosterone concentration which was subsequently abated by heat acclimation.

An unacclimatized person may secrete sweat with a sodium concentration of  $60 \text{ meq} \cdot \text{L}^{-1}$  or higher and therefore, if sweating profusely, can lose large amounts of sodium. With acclimatization, the sweat glands become able to conserve sodium by secreting sweat with a sodium concentration as low as  $10 \text{ meq} \cdot \text{L}^{-1}$  (1). This salt-conserving effect of acclimation depends on the adrenal cortex; and aldosterone, which is secreted in response to exercise and heat exposure as well as to sodium depletion, appears to be necessary for its occurrence. The conservation of salt also helps to maintain the number of osmoles in the extracellular fluid, and thus to maintain or increase extracellular fluid volume (71).

*Dry vs Humid Heat.* Although heat acclimation in a dry environment confers a substantial advantage in humid heat, the physiological and biophysical differences between dry and humid heat lead one to expect that humid heat acclimation would produce somewhat different physiological adaptations from dry heat acclimation; and although the pertinent literature is rather meager, there is evidence to support this expectation.

Fox et al. (22) compared the effects of acclimation to dry and humid heat on the inhibition of sweating. They acclimated resting subjects with controlled hyperthermia, maintaining core temperature near  $38.2^{\circ}\text{C}$  for 2 hours a day for 12 days, using dry heat for one group and moist heat for the other group. To collect sweat, both groups had their left arms in plastic bags, which created a warm, humid microclimate. After acclimation both groups showed similar decreases in heart rate and core and skin temperatures, with similar increases in sweating during an exercise-heat test. In a 2-hour controlled hyperthermia test while they rested in very humid heat, both groups had about the same whole-body sweat rates. The arms that were exposed to humid heat during acclimation had—compared to pre-acclimation responses—similar and large increases in their sweat production during this test, and sweat rates of these arms declined more slowly during the test. During the same test the right arms of the "dry" group, which had not experienced humid heat during acclimation, also had a higher initial sweat rate than before acclimation, but thereafter their sweat rate declined as fast as before acclimation, so that their total sweat secretion during the test was substantially less than that either of the contralateral arms or of the arms of the "humid" group. Thus most of the improvement in the ability to maintain high sweat rates in high humidity after acclimation apparently owed to a diminution of hydromeiosis.

Strydom and Williams (95) tested subjects' responses to 4 h of exercise in a humid environment both before and after a program of physical training, and compared their responses to those of another group of subjects who were well acclimated to humid heat. During the first hour of exercise, subjects in the training group showed better heat tolerance after training than before; and their responses after training approached those of the well-acclimated group. During the second hour of exercise, however, their heart rates and rectal temperatures increased more than those responses for the heat acclimated subjects, and by the end of the second hour their responses after training had come to appear more like their responses before training, and less like the responses of the heat acclimated subjects. Except during the first hour of the exercise-heat exposure, the physically trained subjects sweated considerably less than the heat acclimated subjects. Therefore, the probable reason for the greater physiological strain that the physically trained subjects experienced in the second hour and beyond was their inability to secrete and evaporate sweat at a rate sufficient to achieve thermal balance.

To achieve a high evaporative cooling rate in a humid environment, it is necessary to overcome the high ambient water vapor pressure by maintaining either a higher vapor pressure at the skin (which requires a higher skin temperature) or a larger wetted skin area, as compared to what would be necessary in a dry environment. Unless core temperature is allowed to rise along with skin temperatures, the higher skin temperature must be achieved by increasing core-to-skin thermal conductance, which requires a higher skin blood flow. Therefore, one expected difference between acclimation to humid heat and acclimation to dry heat is for the former to involve greater circulatory adaptations, to support higher skin blood flow with minimal circulatory strain.

Another difference that might be expected between acclimation to humid heat and dry heat is for the former to enable more efficient use of the skin as an evaporating surface. In humid heat, a greater portion of the sweat production is on the limbs after acclimation than before (85). We are not aware of any reports of

changes in the regional distribution of sweating after acclimation to dry heat. Before acclimation, mean sweating intensity (i.e., sweat rate per unit area) is much lower on limbs than on the trunk, so acclimation tends to make the sweating intensity more uniform over the skin surface. This is an advantage in humid heat, because it increases the wet body surface area, and therefore sweat evaporation rate, and probably reduces the extent to which sweating in some regions is in excess of the rate it can be evaporated.

Research has not fully evaluated the magnitude of cross-acclimation that exercise combined with either dry or humid heat confers during exercise in the other hot climate. Studies indicate (which generally have inadequate designs and limited data) that some cross acclimation can occur between humid heat and dry heat exposure. Passive dry heat or passive humid heat acclimation elicited similar core temperature levels during exercise in both hot climates (21). Exercise-dry heat acclimation conferred an advantage (over no heat acclimation) during exercise in humid heat (7,21) and vice versa (16). In addition, Shapiro and colleagues (91) reported that exercise ( $\sim 35\%$   $\text{VO}_{2\text{max}}$ )-dry heat acclimation elicited equal or greater core temperature during exercise in humid heat than a matched ( $\text{WBGT} = 34^\circ\text{C}$ ) dry heat climate. Unfortunately, that study did not report pre-acclimation data and employed only dry heat acclimation. Sawka and colleagues (84) found that exercise ( $\sim 29\%$   $\text{VO}_{2\text{max}}$ ) in matched ( $\text{WBGT} = 32^\circ\text{C}$ ) hot-dry and hot-wet climates resulted in similar core temperature levels both before and after completing a heat acclimation program (which consisted of daily alternating dry heat and humid heat exposures).

Griefahn and Schwarzenau (28) compared the physiologic time course of acclimation to humid-heat, dry-heat and radiant-heat at equivalent ( $33^\circ\text{C}$ )  $\text{WBGT}$  temperatures. Eight subjects complete a 15-day exercise heat acclimation program in each climatic condition. An unknown number of subjects participated in one to all three of the heat acclimation programs that were spaced by at least 52 days. These investigators reported that humid-heat elicited a more rapid acclimation and less physiologic strain (core temperature, heart rate and sweating rate) than dry-heat exposure. Unfortunately, the experimental design (lack of matched groups or cross over testing) did not allow cross acclimation effects' to be evaluated nor did the authors speculate their findings to that issue.

No study directly compared deacclimation for matched groups after humid heat and dry heat acclimation. Pandolf and colleagues (73) acclimated soldiers to dry heat ( $49^\circ\text{C}$ , 20%) and studied their deacclimation over 3-weeks. They reported that 13% and 4% of the core temperature 'advantages, and 23% and 29% of the heart rate advantages were lost after one and three weeks of deacclimation, respectively. Williams and colleagues (98) acclimated African miners to humid heat ( $35^\circ\text{C}$ , 80%) and studied their deacclimation over three weeks. They reported that 26% and 45% of the core temperature advantages and 65% and 92% of their heart rate advantages were lost after one and three weeks of deacclimation, respectively. Together these studies suggest that deacclimation might occur more rapidly for humid than dry heat.

*Aerobic Fitness.* In addition to improving aerobic power, endurance training in temperate climates reduces physiological strain and increases submaximal exercise capabilities in the heat and endurance-trained individuals exhibit many of the characteristics of heat-acclimated individuals during exercise in the heat (5). In addition, aerobically fit persons develop heat acclimation more rapidly than less fit persons, and high aerobic fitness might reduce susceptibility to heat injury / illness (25). A person's maximal aerobic power accounts for approximately 44% of the variability in core temperature after 3 h of exercise in the heat, or the number of days required for complete development of heat acclimation (5). However, endurance training alone does not totally replace the benefits of heat acclimation produced by a program of exercise in the heat (5).

Some investigators believe that for endurance training to improve thermoregulatory responses during exercise in the heat, the exercise training sessions must produce a substantial elevation of core temperature and sweating rate. Henane and colleagues (33) compared thermoregulatory responses of six skiers ( $\text{VO}_{2\text{max}} = 67 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) with those of four swimmers ( $\text{VO}_{2\text{max}} = 66 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ), found that skiers were more heat tolerant and better acclimatized than swimmers, and attributed the difference to a smaller increase in the swimmers' core temperature produced during training in cold water. In agreement, Avellini et al. (6) found that four weeks of training by cycle exercise in  $20^\circ\text{C}$  water increased  $\text{VO}_{2\text{max}}$  by 15%, but did not improve thermoregulation during exercise-heat stress. Thus, high  $\text{VO}_{2\text{max}}$  is not always associated with improved heat tolerance.

To achieve improved thermoregulation from endurance training in temperate climates, either strenuous interval training or continuous training at intensities greater than 50%  $\text{VO}_{2\text{max}}$  should be employed. Lesser training intensities produce questionable effects on performance during exercise-heat stress (5). The endurance training must last at least one week (67) and some authors show that the best improvements require 8-12 weeks of training (5).

*Thermal Tolerance.* Thermal tolerance refers to cellular adaptations from a severe nonlethal heat exposure that allows the organism to survive a subsequent and otherwise lethal heat exposure (42,64). Thermal tolerance and heat acclimation are complimentary as acclimation reduces heat strain and tolerance increases survivability to a given heat strain. For example, rodents with fully developed thermal tolerance can survive 60% more heat strain than what would have been initially lethal (60). Thermal tolerance is associated with heat shock proteins (HSP) binding to denatured or nascent cellular polypeptides and providing protection and accelerating repair from heat stress, ischemia, monocyte toxicity, and UV radiation in cultured cells and animals. HSPs are grouped into families based upon their molecular mass. These HSP families have different cellular locations and functions that include processing of stress-denatured proteins, management of protein fragments, maintenance of structural proteins, and chaperone of proteins across cell membranes.

The HSP responses will increase within several hours of the stress and will last for several days after the exposure. After the initial heat exposure, mRNA levels will peak within an hour and subsequent HSP synthesis depends upon both severity of heat stress and cumulative heat stress imposed (60). Both passive heat exposure and physical exercise will elicit HSP synthesis; however, the combination of exercise and heat exposure elicits a greater HSP response than either stressor does independently (93). Recent research has identified ~130 genes that are up-regulated and ~89 genes down-regulated during heat stress (24). The contribution of most of these genes on thermal tolerance has not been determined, but certainly more than HSP responses are involved.

## **Adaptations to Cold Stress**

Human thermoregulatory adaptations to chronic cold exposure are more modest and less understood than adaptations to chronic heat (104). Where chronic heat exposure induces a fairly uniform pattern of thermoregulatory adjustments, chronic cold exposure induces three different patterns of adaptation. Habituation is characterized by blunted physiological responses during cold exposure. Metabolic adaptations are characterized by enhanced thermogenic responses to cold. Insulative adaptations are characterized by enhanced body heat conservation during cold exposure.

*Habituation.* Habituation, the most commonly observed cold adaptation, is characterized by blunted shivering, blunted cutaneous vasoconstrictor response, or both. Circumpolar residents such as the Inuits (2,32,35), other Native North Americans from the Arctic (18,45) and Norwegian Lapps (3) respond to whole-body cold exposure in a similar manner as persons from temperate climates. That is, metabolic heat production increases due to shivering, and convective heat loss decreases due to vasoconstriction of peripheral blood vessels. However, these responses may be less pronounced in circumpolar residents, as demonstrated in studies of Norwegian Lapps (3). The Lapps exhibited a smaller increase in oxygen uptake, indicative of less shivering, compared to control subjects. Lapps also maintained warmer skin during cold exposure than unacclimatized persons. Other circumpolar residents also exhibited blunted shivering (2) and blunted vasoconstrictor responses to cold (35,77) compared to control subjects.

The warmer skin in cold-exposed circumpolar residents than control subjects results from altered vasomotor responses. Inuits maintained higher hand blood flow than unacclimatized subjects during two-hour water immersions at temperatures ranging from 45°C down to 5°C, and the difference was greatest at the colder temperatures (12). Elsner *et al.* (19) observed that during hand immersion in cold water Native Americans from the arctic exhibited greater hand heat loss than unadapted control subjects. Forearm blood flow during arm immersion in cold water was greater in Inuits than control subjects (11). Collectively, these observations indicate that cold-induced vasoconstriction is less pronounced in circumpolar residents than in unacclimatized persons.



The blunted shivering and vasoconstrictor responses that develop with habituation, might lead to a greater fall in core temperature during cold exposure. This is evident in the studies of the Lapps, however, not all cold habituated circumpolar residents manifest hypothermic habituation. Americans native to Arctic regions and unacclimatized subjects do not differ in core temperature response to cold exposure (2,32,35,45,77).

The blunted response to cold is less apparent in young circumpolar residents suggesting that the adaptation is developed over time rather than inherited (63). Also, other ethnic groups from temperate climates whose occupations necessitate frequent cold exposure of the hands, and people who sojourn in circumpolar regions experience cold habituation (9,10,13,54,62,68), even when those cold exposures are rare and brief (10,62). Thus, habituation can occur even when cold exposures are too mild or brief to cause increased body heat loss or a fall in body temperature. Cold acclimation studies confirm this suggestion.

Attempts to induce cold acclimation by repeated cold-air exposure have employed a wide range of temperatures and exposure durations. Brief ( $\leq 1$ -hr) cold air exposures, repeated over a two-week period, resulted in blunted shivering but had no effect on body temperature during cold exposure (4,34,92). In studies employing longer exposure durations and a longer acclimation period, reduced shivering was accompanied by blunted vasoconstriction (61). Habituation of both shivering and vasoconstrictor responses can lead to more pronounced declines in body temperature during cold exposure than occur in unadapted persons exposed to the same conditions (14,49,52). Hence, this is termed hypothermic habituation.

Superficially, habituation may not seem beneficial since the thermoregulatory adjustments do not help maintain normal body temperature during cold exposure. However, people living in regions experiencing the most extreme cold weather on earth generally have adequate clothing and shelter to protect them from the cold, so they probably do not experience significant whole-body cooling, thus, explaining the lack of more dramatic thermoregulatory adjustments. On the other hand, periodic short-term exposure of small portions of the body would be common, such as when gloves are removed to complete a task requiring dexterity or when individuals moved through unheated corridors of a polar base. Indeed, when whole body cooling is unlikely, warmer skin and reduced shivering would help conserve energy, improve comfort and prevent peripheral cold-injuries.

*Metabolic Adaptations.* Thermoregulatory responses to cold in the Alacaluf people have been cited as evidence for a metabolic cold adaptation (29,30). These nomadic Native Americans lived on coastal islands off the southern tip of South America, where the climate was rainy and cool (lows from 0 to 8°C and highs from 5 to 15°C). Overall, when they were studied for signs of cold acclimatization, the Alacaluf's way of life was similar to Inuits of the North American Arctic. While the environment was less severe, the Alacaluf's clothing (loin cloth and cloak) and shelter (lean-tos built from scrap lumber) were less protective than that of the Inuits.

During a standardized overnight cold exposure, Hammel et al. (30) observed that metabolic heat production was initially higher in Alacaluf than unacclimatized subjects. Some researchers consider this evidence of enhanced thermogenesis, or metabolic acclimatization, induced by chronic cold. However, in contrast to the progressive rise in metabolic heat production exhibited by unacclimatized subjects during the overnight cold exposure, Alacaluf subjects exhibited a progressive fall in heat production, so that the Alacaluf and non-adapted subjects reached similar metabolic rates by the end of the cold exposure (30). Therefore, these people may, in fact, have been exhibiting effects of cold habituation (i.e. blunted shivering) rather than an enhanced thermogenic response.

One other study suggests that an enhanced metabolic response to cold can develop due to repeated cold exposure. Scholander *et al.* (88) studied eight students who camped six weeks in the Norwegian mountains during autumn when it was moderately cold, and rain, sleet and snow were frequent. To increase cold stress, the students had only lightweight summer clothing and minimal shelter. After completing this acclimation period, the campers exhibited a greater increment in metabolism upon cold exposure than unadapted subjects. However, Scholander *et al.* (88) failed to measure the responses before the campers underwent the acclimation. It is unclear whether control and acclimation groups were matched for confounding factors such as body composition, physical fitness or age. Therefore, the possibility that repeated cold exposure humans can induce enhanced thermogenic response remains open to question.

*Insulative Cold Adaptations.* Studies of the Aborigines living in the central Australian desert suggested an insulative pattern of cold acclimatization (31,36,89). Night-time lows in the central Australian desert reach 0°C in winter and 20°C in summer; low humidity and clear atmosphere facilitated evaporative and radiative cooling. When these studies were being completed, the central Australian Aborigines were nomadic people who lived out of doors and wore no clothing. They slept on bare ground and their only protection from the cold was a small fire at their feet and windbreak made from light brush.

While metabolic rate increased in unadapted European subjects sleeping in the cold, the Aborigine's metabolic rate remained unchanged as ambient temperature fell at night (29,36,89). In contrast to habituated circumpolar residents, the Aborigines exhibited a greater fall in skin temperature than did Europeans which was attributed due to a more pronounced cutaneous vasoconstrictor response to cold (36,89). Additionally, the Aborigine's rectal temperature also fell more than in control subjects (31). However, thermal conductance (metabolic heat production divided by the core to skin temperature gradient) was less in the Aborigine than unacclimatized Europeans suggesting that the lower thermal conductance of the Aborigine reflected an enhanced vasoconstrictor response to cold. Alternatively, the Aborigines may have exhibited a lower thermal conductance simply because their shivering had become habituated, but unlike the Inuits and Lapps, vasoconstrictor responses had not.

Long-distance swimmers, surfers and scuba divers show a blunted or delayed shivering during cold-water immersion (15,94) suggesting that they have become cold habituated. However, studies of professional breath-hold divers of Korea, the Ama, and their counterparts in Japan, suggest development of a more complex adaptation to repeated cold-water immersion. Traditionally, the divers wore only a light-weight cotton bathing suit which offered little insulation, and they dove year round in water as cold as 10°C in the winter and 25°C in the summer (39,40). During these dives, they experienced marked whole-body cooling. The divers continued working in the water until their core temperature fell by 2°C (47,48). These people's willingness to repeatedly subject themselves to such stressful conditions alone seems evidence for their acclimatization to cold.

Kang et al. (46) observed that Korean diving women exhibited a seasonal variation in basal metabolic rate (BMR) consistent with a metabolic acclimatization. In the summer, when water temperatures were warmest, the Ama's BMR was lowest. Throughout the fall, the Ama's BMR increased, becoming highest in the winter when water temperatures were coldest. Non-diving control subjects from the same community exhibited no seasonal fluctuation in BMR. While the elevated BMR during winter appeared related to increased cold stress, the practical value of an increased BMR was negligible (39,46). Other observations are consistent with the development of cold habituation in the diving women. The Korean diving women tolerated much colder water without shivering than non-divers of comparable fat thickness (37,38). Although the Ama's shivering responses indicate that they had become cold-habituated, their vasomotor responses and skin heat flow during cold exposure suggest that a more complex acclimatization.

The Ama diving women appeared to have developed an insulative form of cold acclimatization; that is, mechanisms for body heat conservation were enhanced. Maximal tissue insulation was greater in divers than in non-divers with comparable subcutaneous fat thickness (38). The mechanisms for the insulative acclimatization remain unidentified. However, assuming that skin thickness contributes negligibly to insulation, the increased insulation in divers must derive from their control of circulation to the peripheral shell.

Unfortunately, follow-up studies are no longer possible. Since 1977, the divers have used wet suits, and modern divers have substantially more subcutaneous fat than the first diving women studied (74). Thus, modern divers experience less body cooling than traditional divers and insulative acclimatization is no longer apparent (74). This suggests that the stimulus for the different pattern of cold acclimatization exhibited by the traditional divers as opposed to circumpolar residents or Aborigines was the more substantial whole-body cooling experienced by the traditional divers. Acclimation studies tend to support this thesis.

Repeated cold-water immersion induces different acclimation patterns, depending on cold intensity, exposure duration and length of acclimation period. Brief immersions induce habituation, even when only a few immersions are completed (53,55,76). For example, Radomski and Boutelier (76) had subjects immerse themselves in 15°C water 20-60 minutes a day for 9 days, and observed shivering and vasoconstrictor responses to cold became blunted, allowing a greater fall in rectal temperature. This hypothermic habituation

also diminished the sympathetic response to cold. When the immersion durations are increased and the immersions repeated over a longer acclimation period, acclimation patterns besides habituation are induced.

Young et al. (105,106) studied the effects of an acclimation program consisting of 90 minutes of immersion in 18°C water, repeated five days per week for eight weeks. During each immersion, subjects experienced about a 1°C decrease in rectal temperature. Before and after acclimation, physiological responses were measured while the subjects were exposed to cold (5°C) air. Some acclimation effects appeared consistent with hypothermic habituation. Metabolic heat production increased more slowly during cold-air exposure following acclimation, and the fall in rectal temperature during cold-air exposure was greater and more rapid. However, other adaptations suggested the development of an insulative acclimation.

Following repeated cold-water immersion, cold-air exposure caused skin temperature to fall about 4°C lower than before acclimation (105). The greater fall in skin temperature during cold exposure suggests that a more pronounced cutaneous vasoconstrictor response to cold had developed. The increment in plasma norepinephrine concentration elicited by cold air exposure was more than two-fold greater following acclimation suggesting increased sympathetic nervous responsiveness to cold. In addition, a smaller increment in blood pressure during cold exposure was observed after acclimation, while cardiac output responses to cold were unaffected (65). The blunting of the systemic pressure response to cold indicated that subcutaneous vascular beds are better perfused following acclimation. Thus, as was suggested to have occurred in the Korean diving women, acclimation by repeated cold-water immersion may enable better heat conservation by improved insulation at the shell surface, while perfusion of the subcutaneous shell is more optimally maintained than before acclimation.

The lower skin temperatures during cold air exposure following acclimation (9,105) have two implications. First, at a given air temperature, lower skin temperatures reduce the thermal gradient for heat transfer between skin and air, which improves insulation. Second, the magnitude of the acclimation effect on skin temperature maintained during cold exposure exceeds the magnitude of the acclimation effect on core temperature maintained during cold air exposure. Therefore, the core-to-skin thermal gradient is enlarged. A larger thermal gradient between core and skin would favor redistribution of body heat from the core to the subcutaneous muscle shell, while lower skin temperature due to enhanced cutaneous vasoconstriction in cold air would limit heat loss from the body's shell.

Recently, O'Brien and colleagues (72) examined the relative importance of skin vs core temperature for stimulating insulative acclimation. Subjects completed 5 weeks of daily 1 hour water-immersions (20°C) while resting or performing exercise. Skin temperature decrements were similar during immersion for both groups. Rectal temperature fell by ~0.8°C for the resting immersions group but was maintained in the exercise immersion group. Physiological responses during resting cold-air (5°C) exposure were evaluated before and after the acclimation program. Neither group demonstrated insulative acclimation. However, the data suggested that core temperature reductions during acclimation sessions may be a needed stimulus for development of increased sympathetic responses to cold, while decreased skin temperature during acclimation sessions is sufficient stimulus for increased vasoconstrictor responses to cold. Finally they concluded that the duration (>60 min) of the core temperature reduction might be an important for inducing insulative acclimation.

*Determinants of the Pattern of Adjustments.* Figure 2 provides a theoretical schematic depicting the development of different patterns of cold adjustments (104). Brief, intermittent cold exposures appear sufficient to induce habituation of shivering and vasoconstrictor responses to cold, even when only very limited areas of the body surface are exposed and whole body heat losses are probably negligible. More pronounced physiological adjustments are observed only when the repeated cold-exposure causes significant body heat loss. Insulative adjustments appear to develop when repeated cold exposures are too severe for body heat loss to be offset by increased metabolic heat production; that is, when cold causes deep body temperature to decline significantly. The possibility that an enhanced thermogenic capability can develop in humans in response to chronic cold cannot be dismissed. It is tempting to speculate that the stimulus for this metabolic pattern of cold adaptation is prolonged periods in which significant body heat loss was experienced, but under conditions in which body heat production increased sufficiently to prevent a significant decline in deep body temperature. This speculation is not unjustified, since the metabolic pattern of cold adjustments has only been reported in studies in which acclimatization or acclimation was induced by exposure to such conditions, i.e. prolonged exposure to moderately cold air.

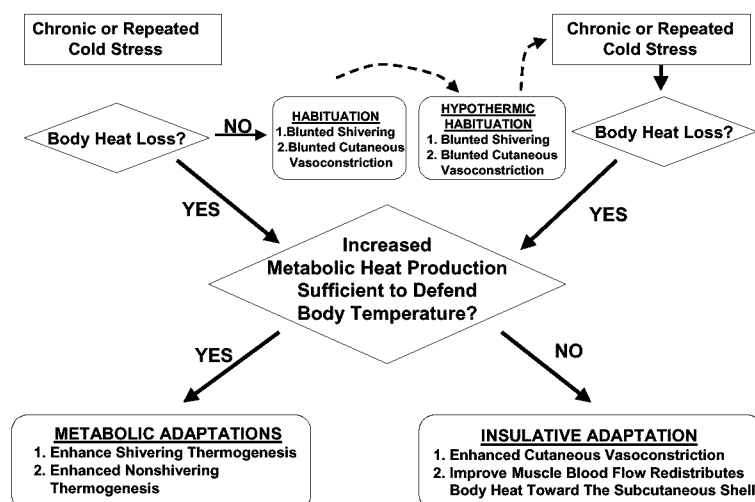


Figure 2. Flowchart illustrating a theoretical scheme to explain the development of different patterns of cold acclimatization / acclimation observed in humans (104).

Alternative explanations for the different patterns of cold adaptations have been proposed. Bittel (8) speculated that body composition and physical fitness determined the acclimation type. His data suggested that lean, fit individuals develop metabolic adjustments and fat, less fit individuals develop insulative adjustments. However, he studied too few subjects to statistically substantiate this hypothesis (8). Skreslet and Aarefjord (94) studied three scuba divers during a standardized cold-water immersion test before and at two week intervals throughout a 45 day period of daily diving in 2-4°C sea-water. Initially, all subjects responded to cold with an increase in metabolic heat production, which in two out of three was sufficient to prevent rectal temperature from falling. After two weeks of diving, all three divers exhibited shivering habituation, and the two divers who tolerated the first immersion without a decline in rectal temperature now experienced a decline. After 45 days of diving, the subjects tended to maintain higher rectal temperatures and lower torso and thigh skin temperatures during immersion than during the initial test. Skreslet and Aarefjord (94) hypothesized that different cold adjustment patterns did not represent development of mutually exclusive physiological states, but rather, different stages in the progressive development of complete cold acclimatization. Thus, their divers initially responded to whole-body cold exposure by shivering; eventually, however, this response disappeared and insulative adaptations developed to help limit body heat loss.

### Disclaimer

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