# BIOPHYSICAL AND PHYSIOLOGICAL BASIS OF HUMAN COLD ACCLIMATIZATION

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(*Received* 19 *Sept.* 1967 : *Revised* 27 *Dec.* 67)

On exposure to cold, the problem is to maintain internal temperature of the human body in the presence of an increased thermal gradient between the core and the external environment. The ability to maintain homeothermy in the cold environment is enhanced in the acclimatized man. Superimposed upon the adaptive responses of the whole body to cold exposure are the adaptive responses of the extremities to avoid severe cold injury. The two major methods of adjustment to cold exposure are metabolic adjustments and peripheral cooling. Metabolic adjustments involve an increase in heat production in response to a cold stress such as shivering and non-shivering thermogenesis and voluntary muscular activity. Peripheral cooling reduces the loss of heat from the skin by effectively increasing the thickness of relatively cooler peripheral tissues. The available literature on human cold acclimatization has been surveyed with a view to explain the biophysical and physiological mechanisms involved in the process of acclimatization.

The changes in the physiological responses induced by the environmental variations may be called acclimatization of the human body to a complex of environmental factors e.g., cold, humidity, wind velocity etc. General hypothermia serves as the most effective stimulus to bring about acclimatization to cold[1]. A high degree of resistance to cold may be developed by continuously lowering temperature of the air in contact with a large surface of the naked human body. Similar results are obtained by water procedures such as sponging down with cold water, showers, and bathing at a continuously lowering temperature in natural reservoirs[2]. During the period of exposure to cold, exercise and muscular activity is to be regarded as an intensive interoceptive heat stimulus. This inhibits by way of cortical induction the effect of extero-ceptive thermal cold stimuli. Thus physical exertion in the cold does not produce the sensation of cooling or lead to an increased resistance to cold despite numerous repetitions.

In recent years numerous studies on human cold acclimatization have been reported. Gupta *et al*[3] observed acclimatization to cold in Indian troops by daily exposed to an environmental temperature of 10—15°C for three hours every day for twenty days. They further observed that the cold acclimatization achieved was not adversely affected by reversion to routine duties with normal snow clothing[4]. From a comparative study of physiological responses to cold, of newcomers and residents of high altitude, Davis *et. al.* concluded that acclimatization to cold does take place although the natives have superior adjustments,[5'6]. Bal Krishna *et. al*[7] studied the peripheral responses of cold acclimatization in army personnels and natives, and observed a better thermoregulatory efficiency in the cold acclimatized troops and the natives. Investigations in polar regions showed significant

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changes suggestive of general cold acclimatization in men's responses to a test cold exposure. Decreased heat production during winter was observed by LeBlanc[8] in the arctic and by Milan & Wyndham *et. al*,[9,10] in the antarctic. Davis[11] observed decreased shivering in the arctic. Skin temperatures were increased or unchanged[8—11] and rectal temperature was unchanged except in LeBlanc's study in which it decreased[8]. A highly significant improvement occurred in the subjects' ability to maintain rectal temperature and was attributed to general cold acclimatization. The experimental evidence by ANARE suggested a smaller heat debt was incurred in antarctica and it was concluded that tissue insulation increased as a result of vasomotor changes. Heat production, shivering and skin temperature did not change significantly in response to a standard cold exposure[12,13]. The experiments were repeated after five years and the findings were confirmed[14]. A comparison of the thermal comfort and the clothing worn both indoors and outdoors in periods before and after mild winter in the antarctic suggested that acclimatization to cold had occurred[15].

Studies before and after six weeks of strenuous outdoor work and cold exposure—often in wet clothing—in the Antarctica showed that physical fitness increased significantly. The response of rectal temperature and shivering to a two-hour period of whole body cooling did not change significantly. Finger temperatures fell more rapidly. There was less cold induced vasodilation and the gradient of skin temperature between elbow and the finger increased significantly suggesting that heat was conserved by means of heat exchanges and enhanced vasoconstriction. Discomfort from cold did not change while subcutaneous fat and arterial blood pressure decreased significantly[16]. It appeared that reduction in insulation caused by loss of fat was balanced by an increase in insulation of other tissues.

The effects of immersion in cold water of the long distance swimmers were studied by Carlson *et. al.*[17], Pugh & Edholm[18]. It was established that the swimmers can endure prolonged immersion in cold water as a result of the protection afforded by an increased thickness of their subcutaneous fat layer and also by a conditioning regimen that trains them to swim for long periods of time at a very high rate of metabolic heat production. Further there is a preferential laying down of fat subcutaneously rather than in the deep fat depots.

Another example of cold acclimatization is the diving women of Korea or Ama who harvest plant and animal life from the coastal waters of the Korean Peninsula. The air temperature approaches zero degree and the water temperature reaches 10°C during winter. So these subjects experience the most severe form of cold stress that human subjects voluntarily tolerate. The studies of Hong[19] showed an increase of 30 per cent in the resting metabolism and an elevation of the tissue insulation in winter. The threshold of shivering response was elevated. Threshold is related inversely to the sensitivity and hence sensitivity to cold shivering is decreased in the Ama.

## INITIAL RESPONSES TO ACUTE COLD EXPOSURE

The intensity of cold and the duration of the test cold exposure used by the various workers differ widely, which makes it difficult to compare the results and a detailed mathematical analysis is not possible. However there is similarity in the physiological response to acute cold exposure. Folk[20] has summed up the human responses to cold as follows:

On exposing a nude man in a cold room, the following events take place (*i*) There will be cutaneous vasoconstriction. This will permit the skin and deeper layers under the skin to cool. The surface to environment heat loss is decreased. The effective thickness of the

body shell is increased and the conductivity from the interior is decreased. This is accomplished by a shift of the blood from the shell area to the core area and there is an increase of blood into the viscera. (*ii*) There is an increase in the heart rate irrespective of the massive vasoconstriction. (*iii*) There is an acceleration of the pulmonary respiration. (*iv*) Piloerection in the skin showing as goose pimples. This reduces heat loss from one sixth to one third of its value (*v*). Next is release of norepinephrine at the muscle bed and of epinephrine from the adrenal medulla. (*vi*) All the items so far are the familiar signs of sympathetic nervous discharge. These will be followed by neurohumoral activation of the hypothalamus bringing about the release of anterior pituitary hormones especially TSH and ACTH. All above results from stimulation of the skin cold receptors which bring about reflex responses all leading to heat conservation. (*vii*) Increased electrical activity of skeletal muscle leads to full development of the shivering response. (*viii*) Increase in metabolic rate three or four fold. (*ix*) Occasional bouts of vasodilation especially on the fingers and the toes due to Lewi's hunting reaction.

When the subject leaves the cold room there may be a fall in the rectal temperature to as low as 35°C. This lowering in core temperature will happen with both vasoconstriction or vasodilation of the skin. It must be due to lack of metabolic heat when cold blood is still being returned to the core from the periphery.

## INITIAL HORMONAL RESPONSES TO COLD

The human thyroid, adrenal cortex and adrenal medulla have been shown to undergo more or less functional fluctuations by acute exposure to a mild cold of 10—15°C for one hour in summer. Among these glands the adrenal, especially the medulla, may be responsible for the physiological responses such as the rise in rectal temperature and changes in heart rate and blood pressure. Because of a long latent period of thyroid hormone action the thyroid may have no direct connection with the initial responses to cold, though thyroxin exerts its potentiating effect on calorigenic action of catecholamines[21]. In the humans a heat debt of 830 cal/Kg was not able to activate pituitary-thyroid axis despite the fact that the tympanic membrane temperatures were significantly lowered. The parameters were TSH, GH, PBI and glucose in the blood[22], In a field study on the effect of cold exposure and muscular activity, the increase in thyroid parameters PBI and cellular concentration of Triiodothyroine, were not regarded as evidence of change in glandular activity. Nor was there any measurable increase in TSH plasma concentration[23].

## HABITUATION TO PAIN AND COLD PRESSOR RESPONSE

A common characteristic of primary manifestation of acclimatization to cold is a reduction of cold pain, cold pressor response and peripheral vasoconstriction. In the studies of Hellstrom[24] the outdoor workers were less troubled by local pains than indoor workers at standard cold exposure. At the same finger skin temperature there was significant lower frequency of pain in the outdoor than in the indoor workers. A significant difference in magnitude of CPR was also found between the groups. These parameters were also the only ones which were substantially changed in the experimentally acclimatized subjects. Convincing evidence of a change in hand blood circulation was not established in these subjects. On the basis that no peripheral mechanism could be postulated, it has been suggested that this adaptation was due to habituation i.e., to some modification in the interpretation of efferent impulses by central nervous system [25,26]. If this were so, increased resistance would not develop if the subjects were distracted from the cold stress whenever they were exposed to it.

This proved to be the case in the subjects who did mental artithmatic test every time the hand was immersed in cold water. They did not develop adaptation to cold[27]. It was felt that habituation observed applies to cold pain and not to cold sensation itself. The phenomena are entirely separate as has been shown by Wolf & Hardy[28]. Adaptation in one hand failed to confer the response in the opposite hand and this applied both to pain and cold pressor response.

## DEVELOPMENT OF CONDITIONED REFLEXES TO COOLING

The conditions under which the effect of cold is felt and the very work done at this time becomes signal stimulus for the processes of chemical thermogenesis in man. The latter is particularly closely related with cortical influences on metabolism. Thus the centres of thermoregulation function reflexly when corresponding receptors are stimulated. There are no isolated subcortical reactions of thermoregulation, because in the process of individual life all regulations of heat exchange is subject to the control of cerebral cortex as a result of the development of numerous extero and interoceptive conditioned reflexes. Thus the process of heat regulation becomes highly specific under conditions of rest and multifarious activity. For example, conditioned reflex mechanism of chemical thermogenesis was domonstrated in the studies of railway workers—freight train conductors—in winter. As the conductor travelled away from the point of departure with its warm room, where he stayed before leaving, this gaseous exchange sharply increased. On the way back the consumption of oxygen decreased, the closer the moment the conductor would find himself in the warm room, although there was no change in the physical conditions of heat loss[29]. Conditioned vascular reflexes to cold were associated with a conditioned increase of oxygen consumption[30].

Due to formation of conditioned reflexes solely under different conditions of human life as in labour and sports activity, it is not possible to explain the whole process of adaptation of human body to cold. This especially requires more intense and more prolonged cooling than used in experiments on acclimatization[31].

## HEAT PRODUCTION BY THE HUMAN BODY

In response to a cold stimulus the body starts shivering. The shivering stimuli originate from the receptors in the skin and the act of shivering depends upon the integrity of the posterior hypothalamus. The function of shivering is to add to heat production. It provides improved protection to the core heat by enlarging the thermogenesis to include the muscle mass. Shivering may be considered from the thermodynamic point of view to be more efficient than voluntary contraction for heat production. In cold shivering periodic spasms are observed involving quick contractions of separate group of muscle fibers associated with a high electrical activity (200—500 $\mu v$) on the electromyograms. The oxygen utilization by the muscle increases by 200 to 400% or more. The average increase in blood flow is 170% and the respiratory quotient as a rule approximates one[32]. In the voluntary contraction only 60 to 70% of the energy consumed appears immediately as heat while in shivering there is an immediate 100% transformation of energy into heat. However it is not very efficient for the maintainance of the body temperature. The heat loss is also increased. Further shivering interferes with skilled movements and sleep in the man[33]. Maximum heat production by shivering has been observed to be 442 cal. per hour after thirty minutes of exposure[38] to 30°F. (wind speed 10 mph). The shivering may be prolonged for a considerable time. Rodahl[35] observed shivering in man for 9 days and

nights. The critical temperature of environment for man at which heat production begins to increase is 28°C, for man immersed in water bath the corresponding temperature is 33°C[36]. There is a large subject to subject variation in the onset of shivering and the time until development of severe continuous shivering. These times are increased by increasing the percentage of oxygen in the inspired gas mixture and on the degree of cold acclimatization of the subject[37]. But individuals are consistent in their response to cold exposure[38]. A linear relation exists between shivering and heat production as shown by the regression equation $Y = aX + b$. It is clear that the slope of the line varies from individual to individual indicating different efficiencies of shivering. The correlation between excess heat production and shivering is highest in subjects who do not exhibit marked cooling of the peripheral tissues. These subjects respond to cold exposure by increased heat production from shivering. In the equation given above $Y$ is the metabolic cold ratio introduced by Hemingway[38]. This is the ratio of oxygen consumption rate in the cold to the oxygen consumption rate under comfortable warm conditions.

The biochemical mechanism of shivering has been explained on the basis of currently accepted concepts of the control of the rate of electron transport and oxygen utilization deduced from studies on isolated mitochondria. When cold exposure induces shivering the greater frequency of muscular contractile activiy causes an increased rate of Adenosine triphosphate (ATP) hydrolysis to ADP and $Pi$ in the muscle. This greater availability of ADP $+Pi$ leads to an elevated rate of electron transport in mitochondria functioning respiratory control. In turn this stepped rate of electron transport provides both an increased generation of ATP for the heat producing hydrolysis of ATP by contractile elements and also a greater direct production of heat by increased activity of electron transport steps not directly involved in ATP synthesis[33].

The changes in shivering thermogenesis have been described on cold acclimatization. Hong[19] has shown an increase in the shivering threshold of the women divers of Korea as compared to the rest of the subjects. He used the critical water temperature as a criteria at which 50% of the subjects shivered. The 50% critical water temperature varied from 31·1°C in males to 28·2°C in the divers. The time of onset of shivering and the time until development of severe continuous shivering, have also been shown to change with cold acclimatization[37]. Ivanov[39] observed thermoregulatory muscle tonus and intensive cold shivering in persons well adapted to intensive repeated cooling (winter swimmers). Cold shivering appeared only after a rather long latent period from the beginning of the cooling. Therefore it can be considered that contractile activity of the muscle in the form of cold shivering and thermoregulatory muscle tonus are of great importance even after acclimatization to cold[39]. Decreased shivering has been shown by Davis[12] & Gupta *et. al.*[4'5]. It has been concluded that a decrease or extinction of shivering reflects important changes resulting from chronic cold exposure[41'42'58].

Hurley *et. al.*[40] & Girling[43] demonstrated a nonshivering thermogenesis in subjects exposed nude at 10°C. Their subjects showed increase in heat production upto 45% above resting levels without shivering. In their further experiments the regression equation showed an elevated metabolic cold ratio at zero electrical activity indicating excess heat production in the absence of shivering and confirmed the previous demonstration of nonshivering thermogenesis in cold exposed subjects. The magnitude of this heat production was 35% above the resting level[38]. The thermoregulatory muscle tonus presents a relative-

ly (5 to 100$\mu v$) in apparently immobile muscles and appears during moderate cooling of the body. The frequency of these feeble mechanical vibrations (25-45 or more/sec.) indicates that during the thermoregulatory tonus fast contractions of separate muscle fibers do occur[44]. The oxygen utilization by the muscle increases by 35%—150% and the average blood flow was 75%. However the respiratory quotient varied considerably[32].

Enough work has been done on nonshivering thermogenesis in laboratory animals especially the rat. The site of nonshivering thermogenesis is either the large muscle mass[45] or the liver and the intestines[46]. As the site of heat production is relatively inside the core the heat production is used to raise the core temperature. The onset of nonshivering thermogenesis appears to be mediated by sympathetic nervous system directed by the CNS, norepinephrine acting as the humoral agent linking the nerve endings to the thermogenic tissue sites[47]. This is supported by the pronounced calorigenic action of norepinephrine in the cold acclimatized rat[48—50]. The biochemical mechanism of nonshivering thermogenesis is either an increased electron transport related to a decreased phosphorylative efficiency or by an increased ATP utilization without a rise in net work yield.

Voluntary heat production as expressed earlier has thermodynamically only 60-70% efficiency. The motor activity of the muscle is an important source of body heat but it is not a particular physiological mechanism of chemical thermoregulation in the cold. In long distance swimmers and also in survivers from ship wreck voluntary exercise is helpful in maintaining body temperature. Pugh *et. al.*[51] have shown that swimmers are able to maintain high skin temperatures by expending 10 *met* of energy—nearly 16°C higher than the water temperature. Whenever the body is in heat, debt voluntary exercise is resorted to even by the Eskimo to meet the heat debt[52]. The metabolic cost of work does not increase if the weight of the winter clothing is accounted for[53].

## BODY CHARACTERISTICS AND EXPOSURE TO COLD

The cooling rate of the human body takes into account the Newton's law of cooling and also the Fouriers law of heat conduction. A combination of the two gives the following equation[54]:

$$\frac{dT}{at} = \frac{1}{W^{\frac{1}{3}}} \times \frac{(T_B - T_E)}{r}$$

This indicates that the cooling rate of the body in the cold environment will be inversely related to the cube root of the body weight. The factor $r$ indicates the insulation of the body which is increased by an increase in the skin fold thickness or by a change in fat composition and conductivity of the skin. Experimental results have shown that fat acted as insulation in all subjects while fat-free body weight has strong positive effect on the total body heat content[55]. As indicated by skin temperatures, men with a fat content equal to 20% of their body weight are approximately 30% better insulated than men with 2% of fat[56]. Besides the fat content there is a variable vascular heat exchange to conserve heat in the cold[57]. One acclimatization to cold heat insulation increases. The resulting increase in the depth of temperature gradients has been demonstrated[58]. From a study of subjects living in tents at 0°C, it was observed that differences between trunk temperatures and extremity temperatures diminish with time in the cold. The extremity temperature tends to rise with the fall of trunk temperature[59].

Results suggestive of increased tissue insulation have been obtained in studies of seasonal acclimatization in Japan and Korea[60,19] and in a study on men exposed to wet cold conditions on an Antarctic Island[16]. Moreover it seems quite probable that the decreased heat production[61] and decreased shivering reported earlier may also be due to increased tissue insulation which would have reduced the requirements for increased heat production in the cold.

## THEORIES OF COLD ACCLIMATIZATION

*Shell and core concept*—This theory visualizes an effective increase in the thickness of relatively cooler peripheral tissues and a relative decrease in the core maintained at the normal body temperature, so that in the cold the body is able to economise heat production in view of the smaller core to be maintained for homeothermy[62-64]. Using a constant skin core ratio the total body heat content at the 7th hour of cold exposure was not altered during cold acclimatization period which lasted for 25 days. Thus changes in body heat content for the cold acclimatization process in man must involve alteration of the relative volumes of the body at shell and core levels. As cold acclimatization was induced, the cyclic rewarming characteristic of hunting reaction occurred at higher finger skin temperature but with reduced amplitude[65].

*Chemical theory of thermoregulation*—The total heat producing capacity of the acclimatized homeotherm is greatly enhanced resulting in a greater ability to maintain homeothermy in extreme cold. The data in man indicates that the acclimatized responses consist of a decreased cold response associated with a raising of the threshold requirements for its elucidation[66].

*Nervous mechanism of cold acclimatization*—The first stage of acclimatization is characterised by the fact that sudden cold action does not elicit marked thermoregulatory reaction, either chemical or physical. It is connected with a orienting reflex—a general nonspecific alarm reaction. The peculiarities of this stage depend upon the intensity of the cold stress and the inherited individual pattern of nervous system.

The second stage of cold acclimatization is characterised by formation of conditioned reflexes to cold where respiratory metabolism is greatly increased and the latent period of thermoregulation is decreased under repeated cooling. During this period strong and inadequate cold stress may call forth inhibition of chemical thermoregulation depending on the typological pattern of neural make up.

The third stage of cold acclimatization is characterised by diminution of cold reaction, adaptation of receptors and greater stability of body temperature under cooling. The decrease of thermoregulatory muscle tonus and of shivering during cooling and the strengthening of the non-shivering thermogenesis and oxidative processes are characteristic of this stage.

The speed of development of these stages depends upon the special or hereditory difference of the individual and the intensity of cooling[67].

Man living in tropics can acclimatize to both heat or cold depending upon the environmental stress. But inhabitants of temperate zone achieve greater part of the possible adaptation to cold. Hence further changes on acclimatization must necessarily be small[68].

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