

# Human Physiological Adaptations to the Arctic Climate

JUHANI LEPPÄLUOTO<sup>1</sup> and JUHANI HASSI<sup>2</sup>

(Received 28 June 1990; accepted in revised form 25 March 1991)

**ABSTRACT.** This review deals with thermal, metabolic and hormonal responses of various human populations to natural or experimental acclimation. Modern people react to cold with shivering, increased metabolism and cutaneous vasoconstriction (metabolic response). Native people, such as Australian aborigines, Eskimos, arctic Indians and Lapps, who were regularly exposed to cold in their natural habitat, have been reported to exhibit less pronounced shivering during experimental cold exposure and experience a greater fall in body temperature (hypometabolic and hypothermic type of adaptation). Australian aborigines and traditional Korean divers have been shown to have low body heat conductivity (insulative type of adaptation). Modern Caucasians intensively exposed to prolonged cold may also develop hypothermic and insulative types of adaptation. Exposure to cold climate increases blood pressure, which may be a factor contributing to the greater mortality due to cardiovascular diseases and stroke observed in the winter. The secretion of the pineal hormone melatonin, which is believed to inhibit the secretion of a pituitary luteinizing hormone, is elevated during winter and decreased in summer. This leads to the higher conception rate observed during spring and summer.

**Key words:** acclimatization, birth rate, blood pressure, body temperature, cold exposure, conceptions, hormones, light, metabolic rate, mortality, native people, season

**RÉSUMÉ.** Cet article se penche sur les réponses thermales, métaboliques et hormonales de diverses populations humaines à l'acclimatation naturelle ou expérimentale. Les peuples modernes réagissent au froid par des frissons, une augmentation du métabolisme et une vasoconstriction cutanée (réponse métabolique). Les peuples plus primitifs, comme les aborigènes d'Australie, les Eskimos, les Indiens de l'Arctique et les Lapons, qui sont exposés au froid sur une base régulière dans leur habitat naturel, auraient démontré qu'ils frissonnaient moins, au cours d'expositions expérimentales au froid, et qu'ils manifestaient une plus grande baisse de la température du corps (type d'adaptation hypométabolique et hypothermique). On a aussi démontré que les aborigènes d'Australie et les plongeurs coréens traditionnels ont une faible conductivité thermique corporelle (type d'adaptation isolante). Il est aussi possible aux caucasoïdes contemporains exposés assez longtemps à un froid rigoureux de développer un type d'adaptation hypométabolique et hypothermique. L'exposition à un climat froid augmente la pression sanguine, ce qui peut être un facteur contribuant au plus grand nombre de décès observés en hiver, dus à des maladies cardio-vasculaires et à des attaques cardiaques. La sécrétion de mélatonine, l'hormone pinéale qui empêcherait la sécrétion de l'hormone lutéinisante hypophysaire, s'accroît au cours de l'hiver et diminue en été. Ce fait est responsable de l'augmentation du taux de conception observé au printemps et en été.

**Mots clés:** acclimatation, taux de natalité, pression sanguine, température du corps, exposition au froid, conception, hormones, lumière, taux de métabolisme, mortalité, autochtones, saison

Traduit pour le journal par Nésida Loyer.

## INTRODUCTION

Primitive man, without knowledge of the use of fire, insulative clothing or housing, had to live in areas with a constant environmental temperature close to his thermo-neutral point. This point is approximately 27-28°C in naked modern man at rest (Erikson *et al.*, 1956) and this was evidently true of primitive man as well. There are areas close to the equator in which the mean annual temperature varies between 25 and 27°C and the difference between the warmest and coldest month is as low as 1°C. This suggests that the birthplace of primitive humans was most probably near the equator. The mean temperature of the coldest month in polar areas exceeds -20°C and is intolerable to unclothed man. Hence the humans who settled in cold weather regions had to develop clothing and housing. There is also scientific evidence that humans have adapted physiologically to cold. The purpose of this paper is to review thermoregulatory, metabolic and cardiovascular adaptation in human populations living in areas with long periods of cold and darkness.

## PHYSIOLOGICAL ADAPTATION TO COLD

In homeothermic humans, various physiological mechanisms operate to maintain a constant body temperature. This means that in a cold environment heat production has to be increased and heat losses decreased. Heat production can be increased by voluntarily increasing

motor activity and by involuntary shivering, which lead to an increased metabolic rate. It can also be increased by increasing the secretion of thermogenic hormones such as thyroid and adrenal hormones (Jansky, 1973; Leblanc, 1966) and by increasing the formation and mobilization of brown fat (Smith and Horwitz, 1969), carbohydrates and other fat, but the role of this non-shivering thermogenesis in adult humans is not clearly established. Heat losses can be limited by behavioral or technical protection and by cutaneous vasoconstriction. In arctic animals, the skin undergoes morphological changes during cold seasons, but no such insulative changes in human skin have been reported.

## Body Temperature and Metabolic Rate

In the 1950s and '60s, there was a great deal of interest in the thermoregulation of several original populations around the world and a reproducible field method was developed in which body temperatures, metabolic rate and other parameters could be followed during sleep (Scholander *et al.*, 1958a). The subjects of these studies slept lightly clad overnight in a bag with their heads in boxes, which enabled collecting the expiratory respiratory gases. The ambient temperature varied between 0-6°C and the insulation of the bag was approximately 2 clo (clothing unit; 1 clo keeps a resting man comfortable at room temperature), ensuring a moderately intensive cold exposure. However, these exposures were carried out in less controlled field conditions and the

<sup>1</sup>Department of Physiology, University of Oulu, Kajaanintie 52 A, SF-90220 Oulu, Finland

<sup>2</sup>Regional Institute of Occupational Health, Oulu, Finland

©The Arctic Institute of North America

comparisons of the physiological responses among different populations may therefore be difficult.

This all-night cold test was performed on unacclimatized or cold-exposed Norwegians (Caucasians), Australian aborigines, Eskimos, Indians and Lapps (Scholander *et al.*, 1958b; Irving *et al.*, 1960; Lange Andersen *et al.*, 1960; Hammel *et al.*, 1964; Hart *et al.*, 1962). In these conditions unacclimatized Caucasians shivered, did not sleep well, clearly increased their metabolic rate and showed a decrease in skin temperature. Norwegian men acclimatized for six weeks to outdoor life close to the freezing point had a higher metabolic rate and skin temperature and they slept better than their unacclimatized partners in this test. On the other hand, Australian aborigines from the central parts of the continent, who during that time lived almost unclothed outdoors, slept well and had a clearly lower metabolic rate and foot temperature during the all-night cold test than the white controls (Scholander *et al.*, 1958b:Fig. 7). The central Australian aborigines demonstrated a hypometabolic and hypothermic response to the standardized cold test, which was evidently due to their continuous exposure to cool nights in their natural habitat. The aborigines also had lower body heat conductivity values than the white controls and lower fat percentage as well (Hammel, 1964). The low heat conductivity was thus based on the effective constriction of skin blood vessels in cold. This hypometabolic and insulative type of adaptation has some advantages, since the lowering of the metabolic rate observed in the aborigines decreases the energy expenditure by 20% during the night, which may be important in a land where food is not abundant (Hammel, 1964).

Later, native populations living in cold climates were also studied using the same methodology. Responses of nomadic Lapps closely resembled those of the central Australian aborigines. During the all-night cold test they slept undisturbed, maintained an almost unchanged metabolic rate and a decreased rectal temperature but had warmer skin temperatures than their Caucasian controls (Lange Andersen *et al.*, 1960). Responses of Canadian Indians and Eskimos differed. They did not sleep well, increased their metabolism and had a decreased rectal temperature but warmer skin temperature than the white controls (Hart *et al.*, 1960; Irving *et al.*, 1960). Metabolic rate and rectal temperature of Alacaluf Indians from Tierra del Fuego did not change during the all-night cold test (Hammel *et al.*, 1960).

Due to the great variability in the thermoregulatory responses of these populations, it is difficult to make any generalizations about human metabolic and thermal adaptability to cold. When compared to unacclimatized Caucasians, the native groups most frequently showed a slight lowering of their rectal temperature in the all-night cold test. It appears that the type of cold adaptation in primitive people is determined by the degree of cold stress that they experience in their habitat and by the protection from the environmental cold. Eskimos, although living in the coldest environment, have developed better protective clothing, and hence show less adaptation to cold than, for example, the Alacaluf Indians.

Traditional Korean women divers, who were subjected to severe cold stress during their work periods in water, have also been studied for their adaptability to cold (Kang *et al.*, 1963). In summer the water temperature was over 22°C, but in winter *ca.* 10°C. Their basal metabolic rate showed marked seasonal variations, being 30% higher in winter than in

summer (Kang *et al.*, 1963). They were also shown to shiver at a water temperature of 28.2°C, but non-diving women shivered at 29.9°C (Kang *et al.*, 1963). The divers had greater body insulation than the non-divers and had a similar type of insulative adaptation as observed in the Australian aborigines. It is of great interest that this adaptation gradually disappeared after the onset of wet-suit diving (Park *et al.*, 1983). This was shown to be due to the fact that body temperature stayed at a higher level during wet-suit dives than during traditional ones, especially in winter. Thus the cold stimulus leading to metabolic response and insulative adaptation no longer exists in Korean divers (Park *et al.*, 1983).

In several of the above-mentioned studies, unacclimatized Caucasians were exposed to the all-night cold test as controls. As discussed earlier, they exhibited a hypermetabolic response, which contributed to the maintenance of a constant rectal temperature (Scholander *et al.*, 1958a). Following acclimation, other investigators have observed changes in metabolic and thermal responses of Caucasians. Members of an Antarctic base had, at the beginning of their residence, a decreased rectal temperature during a cold air test (hypothermic response), but after wintering on the base, the rectal temperature was better defended (Budd, 1962). Cold-exposed Norwegians also exhibited a metabolic response when tested in the all-night cold test (Scholander *et al.*, 1958a).

In contrast, however, others have observed hypometabolic and hypothermic responses after various acclimatization processes (Leblanc, 1956; Bruck *et al.*, 1976). Radomski and Boutelier (1982) exposed two groups of subjects to a cold air test (60 min at 10°C). One group had been pre-adapted by 9 daily immersions in cold water; these subjects showed a significant decrease in rectal temperature (hypothermic response) and an unchanged metabolic response in the cold air test. The other group was not pre-adapted and had a significant metabolic response with unchanged rectal temperature. Young *et al.* (1986) studied the effects of a cold air test (90 min at 5°C) before and after a series of 24 daily cold water immersions. After the acclimation program they found an increase in plasma norepinephrine levels and a decrease in mean skin temperature, indicating that their chronic cold water acclimation was more of an insulative type. We had the opportunity to test the three members of the Finnish expedition to the North Pole in 1984. The subjects skied about 500 miles and slept in a tent and were thus exposed to very intense cold, mostly around -30°C for two months. In our standard cold air test (120 min at 10°C) they showed a reduced rectal temperature and metabolic rate (Hassi and Korhonen, 1987).

The responses of rectal and skin temperature and metabolic rate to the all-night cold exposure test in some native populations are summarized in Table 1. For this summary each response was compared with that of non-acclimatized Caucasians serving as controls. Body temperature and metabolic responses of Australian aborigines clearly differed from the controls. They also had lower body heat conductance values (which Eskimos did not have [Hammel, 1964]) and represent the insulative type of cold adaptation. Later studies have shown that traditional Korean divers and Caucasians exposed to cold water for a long time (Park *et al.*, 1983; Young *et al.*, 1986) develop that adaptation too, which evidently is the most effective type of adaptation to a cold environment. Changes in rectal temperature and metabolic rate in other populations were variable, but all had increased skin tem-

TABLE 1. Summary of temperature and metabolic responses during the all-night cold test in selected populations compared to nonacclimatized Caucasians

Population	Core temperature	Skin temperature	Metabolism
Australian aborigines	decreased	decreased	decreased
Nomadic Lapps	decreased	increased	decreased
Eskimos	no change	increased	no change
Cold-exposed Norwegians	no change	increased	increased

perature. Perhaps in the conditions such as those experienced by Eskimos and Lapps, increased skin temperature is particularly necessary in order to maintain the functionality of hands and feet.

Table 2 summarizes temperature and metabolic responses to the experimental cold air test after various acclimatization programs. The cold air test usually lasted 1-2 h and all showed hypothermic (core) and hypometabolic adaptation. No values for body heat conductance were given, but the decreased skin temperature response (Young *et al.*, 1986) suggests that their acclimation (24 daily cold water immersions) was insulative.

Based on these findings about the physiological adaptation of various native groups to cold, we may conclude that primitive humans living almost naked in low latitudes with limited food supply had a hypothermic, hypometabolic or insulative type of cold response. The decrease in environmental temperature was never life threatening and the hypometabolic response saved energy, i.e., food.

Involuntary or voluntary exploitation of colder areas set several demands on primitive humans. In the first place, they had to develop clothing and housing, and second, their bodies had to adapt to cold. This adaptation may have depended on technical insulation against the cold, food supply and the duration of the cold exposure. It is notable that the various forms of cold responses — hypothermic, hypometabolic, metabolic and insulative — still exist in present populations. Skreslet and Aarefjord (1968) have suggested that the different types of cold adaptation are not mutually exclusive but rather different stages in the development of complete cold adaptation. This concept has also been presented in a recent review (Young, 1988). The insulative type of cold adaptation, observed in Australian aborigines, traditional Korean divers and Caucasians exposed chronically to cold water (see above), is evidently one of the characteristics of complete cold adaptation.

TABLE 2. Summary of temperature and metabolic responses to the cold air test in selected cold acclimatization programs compared to pre-exposure or control group

Reference	Core temperature	Skin temperature	Metabolism
Bruck <i>et al.</i> , 1976	decreased		decreased
Radomski and Boutelier, 1982	decreased	no change	decreased
Young <i>et al.</i> , 1986	decreased	decreased	decreased
Hassi and Korhonen, 1987	decreased		decreased

### Non-shivering Thermogenesis

**Brown fat:** Non-shivering thermogenesis is regulated by sympathetic nerves and thermogenetic hormones. The main energy source of non-shivering thermogenesis is brown fat, especially after birth, and other fat and carbohydrate stores of the body (Smith and Horwitz, 1969). The sympathetic nerves and adrenal medullary hormones, catecholamines, control the mobilization of energy from brown fat and other energy stores. The contribution of non-shivering thermogenesis can be estimated by infusions of noradrenaline (a catecholamine and a transmitter substance of sympathetic nerves). It has been shown that noradrenaline infusions raise the metabolic rate about 10% in traditional Korean divers during winter or in acclimatized members of Antarctic bases (Kang *et al.*, 1963; Budd and Warhaft, 1966). This is believed to represent the proportion of non-shivering thermogenesis of the energy production in cold-adapted humans. In a cold climate the amount of brown fat appears to be related to the duration of the cold exposure. Autopsy studies in Finland have shown that outdoor workers have more brown fat around their central blood vessels than indoor workers in winter (Hassi, 1977; Huttunen *et al.*, 1981). It has also been reported that the thickness of subcutaneous fat in long-distance swimmers is about double that of the general population (Pugh and Edholm, 1955). Thus the development of brown or subcutaneous fat in adults exposed to chronic cold may be regarded as a separate type of cold adaptation.

**Thermogenic hormones:** Several hormones are required for optimal thermogenesis. It is well known that thyroidectomized and adrenalectomized animals do not tolerate cold (Leblanc, 1966; Jansky, 1973), but the role of hormones in adult human cold adaptation is less known. During a single exposure to cold (cold air 120 min at 10°C) there are no changes in the serum levels of thyroid hormones, cortisol, adrenaline or pituitary thyrotropin, indicating that a short-time cold air stimulus is not enough to stimulate the secretion of these hormones in humans (Young *et al.*, 1986; Leppäluoto *et al.*, 1988). On the other hand, serum noradrenaline levels clearly increase in response to this short-time cold stimulus. Increased serum noradrenaline originates from sympathetic nerve endings and is related to vasoconstriction and the release of energy substrates such as fats and carbohydrates.

The results concerning the secretion of catecholamines during chronic cold exposures are controversial. Unchanged (Budd and Warhaft, 1970) or decreased (Mager and Robinson, 1969; Radomski and Boutelier, 1982) secretions of urinary noradrenaline and unchanged (Mager and Robinson, 1970; Budd and Warhaft, 1970) or increased (Radomski and Boutelier, 1982) secretions of urinary adrenaline have been observed after various acclimatization methods. In a recent study cold acclimation led to an increase in plasma noradrenaline levels in a cold air test (Young *et al.*, 1986). It would be useful to know how plasma noradrenaline levels are related to the metabolic type of adaptation, but at present no firm conclusions can be drawn about the association of noradrenaline secretion to metabolic acclimation.

Although thyroid hormones and the pituitary thyroid-stimulating hormone thyrotropin are not secreted during a short cold exposure in humans (Leppäluoto *et al.*, 1988), their secretions and degradation appear to increase in cold seasons. Studies from Japan show that serum thyrotropin levels in

hypothyroid patients increase in winter (Konno and Morikawa, 1982). During a five-month Antarctic expedition low serum-free triiodothyronine levels and an exaggerated thyrotropin response to hypothalamic thyrotropin-releasing hormone (TRH) were observed (Reed *et al.*, 1988). These authors showed that the degradation of thyroid hormones gradually accelerated in the cold environment. This indicates greater utilization of thyroid hormones at the cellular level and greater heat production, a well-known thermogenic effect of thyroid hormones. There are also indirect findings showing that more thyroid hormones are needed in winter. Hypothyroid patients under constant substitution of thyroid hormones have greater thyrotropin responses to TRH in winter than in summer (Konno and Morikawa, 1982:Fig. 2).

Circulating levels of the major glucocorticoid hormone cortisol are increased in winter (Reinberg *et al.*, 1978; Touitou *et al.*, 1983). Whether this increase is associated with any harmful process in humans is unknown. Since high levels of cortisol have been found to attenuate the immune system (MacMurray *et al.*, 1983), the latter authors have suggested that the high cortisol levels in winter are associated with the spreading of viral respiratory epidemics occurring most frequently in winter in the northern hemisphere.

The changes in some parameters of non-shivering thermogenesis are given in Table 3. Long-term cold exposure (winter) appears to decrease circulating levels of thyroid hormones in humans. This is, however, counterbalanced by the increased secretion of pituitary thyrotropin. The role of thyroid hormones in non-shivering thermogenesis in these conditions is not known; their low levels may even indicate the presence of a calorogenic effect. Increased responses of cortisol, adrenaline and noradrenaline to long-term cold exposure have been reported in most, but not all, studies. Finally, there are anthropological and biochemical data indicating that brown or subcutaneous fat grows in response to severe cold exposures. The summarized data show that in response to long-term cold exposure the secretion of thermogenic hormones increases and energy stores grow. It is not clearly known how this non-shivering thermogenesis is related to thermic or metabolic adaptation to cold.

### Circulatory Responses to Cold

The main circulatory response to cold is a vasoconstriction of the peripheral circulation that is mediated by sympathetic autonomic nerves and circulating catecholamines. For example, skin blood flow in thermoneutral conditions is about  $0.3 \text{ L} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$  but decreases during severe cold exposure to  $0.05 \text{ L} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$  (Rowell, 1977). The purpose of this cold-

induced vasoconstriction is to decrease heat conductivity from the body to the environment. Conductivity appears to depend on the thickness of the skin and subcutaneous fat and, to a greater extent, on the organization of skin and muscle vessels. The mechanisms resulting in decreased heat conductivity during cold adaptation have been shown to be the more efficient countercurrent heat exchange (Hong, 1973) or redistribution of the central blood flow to peripheral vessels (Carlson *et al.*, 1953). It is also possible that circulating noradrenaline potentiates the vasoconstriction and thus leads to lower heat conductivity, since the plasma noradrenaline levels in a standard cold air test were higher after repeated cold water immersions than before them (Young *et al.*, 1986).

The cold-induced peripheral vasoconstriction leads to increased peripheral vascular resistance and also to increased blood pressure. The results from our laboratory show that during a 2 h whole-body cold-exposure test the systolic and diastolic blood pressure were increased and the heart rate decreased (Leppäluoto *et al.*, 1988). Blood pressure increases also when only hands or feet are exposed to cold. This is a so-called cold-pressure test, which has been used in the diagnosis of high blood pressure (Hines and Brown, 1932).

Cold-induced vasoconstriction is a definite advantage in human cold adaptation and has increased our capacity to tolerate low environmental temperature. Other circulatory responses may not be as advantageous. In several European countries annual mortality appears to be linked to seasons, so that the highest mortality rates occur in winter (Näyhä, 1981). The most distinct seasonal variations can be seen in the diseases of the cardiovascular and respiratory systems. Mortality rates of myocardial infarction and stroke in particular are 15-30% higher in winter than in summer (Näyhä, 1981). The relation of increased blood pressure to these diseases has been investigated in many studies. In the northern hemisphere, blood pressure is higher in winter than summer (Rose, 1961; Brennan *et al.*, 1982; Leppäluoto *et al.*, 1985; Näyhä, 1985). The difference in the systolic blood pressure was about 7 mm Hg (millimetres of mercury) in England (Brennan *et al.*, 1982) but may be as high as 20 mm Hg in northern Finland in some outdoor workers (Leppäluoto *et al.*, 1985).

The data collected by Näyhä (1985) is presented in Table 4 and demonstrate a maximal seasonal difference in systolic blood pressure of 22 mm Hg between November and July and 21 mm Hg between April and July. His study group con-

TABLE 3. Summary of changes in non-shivering thermogenesis during short- and long-term cold exposures

Parameter	Short-term	Long-term
Thyroid hormones	no change	decreased
Pituitary thyrotropin	no change	increased
Adrenal cortisol	no change	increased
Noradrenaline	increased	increased or variable
Adrenaline	no change	increased or variable
Brown fat		increased
Subcutaneous fat		increased

TABLE 4. Seasonal variation in blood pressure and mortality to cardiovascular diseases according to Näyhä (1981,1985)

Month	Systolic blood pressure (mm Hg)	Diastolic blood pressure (mm Hg)	Mortality (% of annual mean)
January	158	91	106
February	161	91	106
March	158	88	101
April	169	94	98
May	160	90	97
June	161	93	97
July	148	88	94
August	156	90	95
September	154	89	95
October	154	96	100
November	170	95	100
December	163	90	107

sisted of 801 healthy males of ages 50 years and over invited to participate in a medical examination in a rural area of central Finland. It should be noted that the houses in Finland have a central heating system, keeping the room temperature around 20°C throughout the year. These subjects were mostly farmers who daily spent some hours outdoors in winter. A mid-winter drop (between December and April) in systolic blood pressure found in these subjects was attributed to the fact that during cold periods these people frequently remained indoors.

The reason for increased blood pressure in winter may be the cold-induced increase in the peripheral resistance, but in Finland other factors, such as nutrition, have to be taken into account. The diet in Finland has a lower polyunsaturated/saturated fatty acid ratio in winter than in summer and it has been found in a dietary intervention study that increasing the polyunsaturated/saturated fatty acid ratio for a period of six weeks lowers blood pressure (Puska *et al.*, 1983).

Increased blood pressure has been shown to be a risk factor for ischemic heart disease (angina pectoris) and stroke (Kannel *et al.*, 1970; Keys, 1970). In Finland, the mortality related to cardiovascular and cerebrovascular diseases is clearly higher in winter and especially so in older age groups (Näyhä, 1981). When the mortality caused by cardiovascular diseases is compared with the monthly variations in blood pressure (Table 4), it can be seen that the highest blood pressure levels occur in November and the highest mortality in ischemic heart disease in December. It is thus possible that the cold-induced increase in blood pressure in winter is one of the causative factors for the increased mortality caused by vascular diseases in winter. It may take a cold period of 1-2 months before the mortality rates are affected.

Monthly mortality rates are also available from 18th-century Sweden-Finland (Wargentin, 1767). These data show that mortality began to increase in winter and was approximately 50% higher in late spring than in autumn. The mortality rates in Finland from 1961 to 1980 show a peak in January-February, the coldest months (Näyhä, 1981). It is most probable that the spring mortality peak in the 18th century was mainly caused by a shortage of food, since the previous crop was used up and the soil offered nutrition only after 2-3 months. At present the maximum annual mortality occurs in the winter and one of the causal mechanisms underlying this phenomenon may be insufficient adaptation to cold, especially insufficient circulatory adaptation, as discussed above. Näyhä (1984) has estimated that the excess mortality due to winter in Finland is today as high as 2-5% of the total mortality. These figures are quite similar in the rest of Europe, indicating that the cold season is a considerable death harvester still in our time.

#### ENVIRONMENTAL LUMINOSITY

In addition to great seasonal differences in the environmental temperature, there are also great differences in the luminosity (the visible radiation of solar irradiance) between winter and summer in the polar areas. At latitude 70°N, which represents the northernmost areas of the Nordic countries, the sun stays above the horizon for about two months in the summer and does not rise above the horizon for two months in the winter. In low latitudes the luminosity follows heat irradiance from the sun and both are at a maximum around

the summer equinox. In arctic and related areas the reflection of the sunlight from snow (albedo) increases luminosity, so that, for example, in Finland the highest levels are observed between March and May (Helle *et al.*, 1986).

#### Seasonal Luminosity and Vitamin D

Sunlight has several important biological effects. Its effects on the synthesis of vitamin D and on the timing of the reproductive functions have been necessary for species to adapt to high latitudes. Vitamin D deficiency, or rickets, does not occur in the tropics but was earlier a nuisance in northern areas. The problem has disappeared in modern societies due to vitamin D supplementation. Before this, rickets, tetanus and osteomalacia were noted to be at their worst in the late spring, and this seasonal incidence was attributed to the long absence of sunlight during the winter months (McCance and Widdowson, 1943).

#### Seasonal Luminosity and Reproduction

It has been suggested that the 10% increase in the conception rate of humans in spring and early summer is associated with the increase in environmental light (Timonen *et al.*, 1964). Table 5 shows a summary of the data of Timonen *et al.* (1964) contrasted with environmental luminosity, measured by a solarimetry directed downwards. Maximum luminosity occurred in April and that of conceptions two months later. Birth registers from other Nordic countries show that the conception rate is 5-10% above the average between June and July (Rönnike, 1980). Data from Greenland and the Faroe Islands show more fluctuation, which is most probably related to fishing periods and the absence of males at certain times of year.

In addition to an increased conception rate in spring, recent studies from northern Finland have shown that the serum concentrations of estradiol, an ovarian hormone, were higher in spring than in winter and the development of ovarian follicles and ovum was more disturbed in winter than in spring (Ronkainen *et al.*, 1985; Kauppila *et al.*, 1987). These findings strongly support the concept that in high latitudes the conception rate is higher in spring than in winter.

In several species, especially in certain rodents and to a lesser degree also in humans, reproduction seems to depend on the pineal hormone melatonin (Vaughan, 1984). It has been shown that the decreased luminosity in winter is related to the increase in the secretion of melatonin, which has a

TABLE 5. Seasonal variation in conceptions and luminosity in Finland according to Timonen *et al.* (1964) and Helle *et al.* (1986)

Month	Conception rate (% of annual mean)	Luminosity (monthly mean) ( $W \cdot m^{-2}$ )
January	92	110
February	93	120
March	96	190
April	100	220
May	102	140
June	108	135
July	105	135
August	106	120
September	104	105
October	101	105
November	97	100
December	97	100

suppressing effect on reproduction (an anti-gonadotropic effect). We have measured serum melatonin and pituitary gonadotropic hormone levels in winter and in summer in healthy females living around 65°N (Kivelä *et al.*, 1988:Fig. 1). The nocturnal melatonin levels in the first part of the menstruation cycle were significantly higher in winter than in summer and the levels of serum luteinizing hormone (LH) at ovulation higher in summer than in winter. Since ovulation is dependent on the elevated levels of serum LH, we have hypothesized that the higher melatonin levels in human females during winter may partly prevent the LH release at ovulation, thereby explaining the observed decreases in conceptions during the dark season. The significance of seasonal breeding is that offspring are born during the most favorable season. In the case of primitive humans in high latitudes, spring and summer certainly offered the best starting points for newborns.

Arctic and subarctic climates typically have great variations in temperature and luminosity between various seasons. The populations of those areas are therefore continuously exposed to changing stimuli of cold, darkness and light. The present technical development in clothing, housing, transportation and food supply has made the stimuli less challenging but still capable of inducing several detectable physiological responses. We know from previous studies how a response adapts to a cold stimulus, e.g., adaptation of body or skin temperature to cold air. However, we know less about other effects that may modify or even explain the response we are measuring. Thus new areas of research concerning physiological adaptations to the arctic climate should include simultaneous measurements of neural, visceral and humoral responses to distinctly defined stimuli.

## REFERENCES

- BRENNAN, P.J., GREENBERG, G., MIALL, W.E., and THOMPSON, S.G. 1982. Seasonal variation in arterial blood pressure. *British Medical Journal* 285:919-923.
- BRUCK, K., BAUM, E., and SCHWENNICK, H.P. 1976. Cold-adaptive modifications in man induced by repeated short-term cold exposures and during a ten-day and night cold exposure. *Pfluegers Archiv* 363:125-133.
- BUDD, G.M. 1962. Acclimatization to cold in Antarctica as shown by rectal temperature response to a standard cold stress. *Nature* 193:886.
- BUDD, G.M., and WARHAFT, N. 1966. Cardiovascular and metabolic responses to noradrenaline in man, before and after acclimatization to cold in Antarctica. *Journal of Physiology (London)* 186:233-242.
- BUDD, G.M., and WARHAFT, N. 1970. Urinary excretion of adrenal steroids, catecholamines and electrolytes in man, before and after acclimatization to cold in Antarctica. *Journal of Physiology (London)* 210:799-806.
- CARLSON, L.D., BURNS, H.L., HOLMES, T.H., and WEBB, P.P. 1953. Adaptive changes during exposure to cold. *Journal of Applied Physiology* 5:672-676.
- ERIKSON, H., KROG, J., ANDERSEN, K.L., and SCHOLANDER, P.F. 1956. Critical temperature in naked man. *Acta Physiologica Scandinavica* 37:35-39.
- HAMMEL, H.T. 1964. Terrestrial animals in cold: Recent studies of primitive man. *Handbook of Physiology*. Washington, D.C.: American Physiological Society. 413-433.
- HART, J.S., SABEAN, H.B., HILDES, J.A., DEPOCAS, F., HAMMEL, H.T., LANGE ANDERSEN, K., IRVING, L., and FOY, G. 1960. Thermal and metabolic responses of coastal Eskimos during a cold night. *Journal of Applied Physiology* 17:953-960.
- HASSI, J. 1977. The brown adipose tissue in man. Structural and functional aspects in relation to age. *Acta Universitatis Ouluensis, Serie D, Medica* 21:1-92.
- HASSI, J., and KORHONEN, I. 1987. The Huurre Expedition to the North Pole at 1984. Technical Research Centre of Finland, Research Note 685:44-52.
- HELLE, T., KEMPPAINEN, J., LIIKANEN, A., and ASPI, J. 1986. *Lapin tutkimusseuran vuosikirja* 27:23-29. English summary.
- HINES, E.A., and BROWN, G.E. 1932. A standard stimulus for measuring vasomotor reactions: Its application in the study of hypertension. *Proceedings of Staff Meeting of the Mayo Clinic* 7:332-335.
- HONG, S.K. 1973. Pattern of cold adaptation in women divers of Korea (Ama). *Federation Proceedings* 32:1614-1622.
- HUTTUNEN, P., HIRVONEN, J., and KINNULA, V. 1981. The occurrence of brown adipose tissue in outdoor workers. *European Journal of Applied Physiology* 46:339-345.
- IRVING, G.L., ANDERSEN, K.L., BOLSTAD, A., ELSNER, R., HILDES, J.A., LÖYNING, Y., NELMS, J.D., PEYTON, L.J., and WHALEY, R.A. 1960. Metabolism and temperature of arctic Indian men during a cold night. *Journal of Applied Physiology* 15:635-644.
- JANSKY, L. 1973. Nonshivering thermogenesis and its thermoregulatory significance. *Biological Reviews* 48:85-132.
- KANG, B.S., SONG, S.H., SUH, C.S., and HONG, S.K. 1963. Changes in body temperature and basal metabolic rate of Ama. *Journal of Applied Physiology* 18:483-488.
- KANNEL, S.B., WOLF, P.A., VESTER, J., and McNAMARA, P. 1970. Epidemiological assessment of the role of blood pressure in stroke. *JAMA* 214:189-198.
- KAUPPILA, A., PAKARINEN, A., KIRKINEN, P., and MÄKILÄ, U. 1987. The effect of season on the circulating concentrations of anterior pituitary, ovarian and adrenal cortex hormones and hormone binding proteins in the subarctic area: Evidence of increased activity of the pituitary-ovarian axis in spring. *Gynaecological Endocrinology* 1:137-150.
- KEYS, A. 1970. Coronary heart disease in seven countries. *Circulation* 41/42 (suppl. 1):1-211.
- KIVELÄ, A., KAUPPILA, A., YLÖSTALO, P., VAKKURI, O., and LEPPÄLUOTO, J. 1988. Seasonal, menstrual and circadian secretions of melatonin, prolactin and gonadotropins in women. *Acta Physiologica Scandinavica* 132:321-327.
- KONNO, N., and MORIKAWA, K. 1982. Seasonal variations of serum thyrotropin concentration and thyrotropin response to thyrotropin releasing hormone in patients with primary hypothyroidism on constant replacement dosage of thyroxine. *Journal of Clinical Endocrinology and Metabolism* 54:1118-1123.
- LANGE ANDERSEN, K., LÖYNING, Y., NELMS, J.D., WILSON, O., FOX, R.H., and BOLSTAD, A. 1960. Metabolic and thermal response to a moderate cold exposure in nomadic Lapps. *Journal of Applied Physiology* 15:649-653.
- LEBLANC, J. 1956. Evidence and meaning of acclimation to cold in man. *Journal of Applied Physiology* 9:395-398.
- \_\_\_\_\_. 1966. Effects of reserpine on increased sensitivity to noradrenaline of cold adapted rats. *American Journal of Physiology* 204:520-522.
- LEPPÄLUOTO, J. 1984. Cold as a disabling factor in northern countries. *Arctic Medical Research* 37:10-12.
- LEPPÄLUOTO, J., HASSI, J., and PÄÄKKÖNEN, R. 1985. Seasonal variations of blood pressure, basal metabolism, and skin temperature in outdoor workers in northern Finland. In: Fortune, R., ed. *Circumpolar Health* 84. Seattle: University of Washington Press. 70-73.
- LEPPÄLUOTO, J., KORHONEN, I., HUTTUNEN, P., and HASSI, J. 1988. Serum levels of thyroid and adrenal hormones, testosterone, TSH, LH, GH and prolactin in men after a 2-h stay in cold room. *Acta Physiologica Scandinavica* 132:543-548.
- MACMURRAY, J.P., BARKER, J.P., ARMSTRONG, J.D., BOZETTI, L.P., and KUHN, I.N. 1983. Circannual changes in immune functions. *Life Sciences* 32:2363-2370.
- MAGER, M., and ROBINSON, S.M. 1969. Substrate mobilization and utilization in fasting men during cold exposure. *Bulletin of New Jersey Academy of Sciences, Symposium Issue*: 26-30.
- MCCANCE, R.A., and WIDDOWSON, E.M. 1943. Seasonal and annual changes in the calcium metabolism of man. *Journal of Physiology (London)* 102:42-49.
- NÄYHÄ, S. 1981. Short and medium-term variations in mortality in Finland. *Scandinavian Journal of Social Medicine, Supplementum* 21:1-79.
- \_\_\_\_\_. 1984. The cold season and deaths in Finland. *Arctic Medical Research* 37:20-24.
- \_\_\_\_\_. 1985. Adjustment of blood pressure data by season. *Scandinavian Journal of Primary Health Care* 3:99-105.
- PARK, Y.S., RENNIE, D.W., LEE, I.S., PARK, Y.D., PAIK, K.S., KANG, D.H., SUH, D.J., LEE, S.H., HONG, S.Y., and HONG, S.K. 1983. Time course of deacclimatization to cold water immersion in Korean women divers. *Journal of Applied Physiology* 54:1708-1716.
- PUGH, L.G.C., and EDHOLM, O.G. 1955. Physiology of channel swimmers. *Lancet* 2:761-768.

- PUSKA, P., NISSINEN, A., VARTIAINEN, E., DOUGHERTY, R., MUTANEN, M., IACONO, J.M., KORHONEN, H.J., PIETINEN, P., LEINO, U., MOISIO, S., and HUTTUNEN, J. 1983. Controlled, randomized trial of the effect of dietary fat on blood pressure. *Lancet* 1:1-5.
- RADOMSKI, M.W., and BOUTELIER, C. 1982. Hormone response of normal and intermittent cold-preadapted humans to continuous cold. *Journal of Applied Physiology* 53:610-616.
- REED, H.L., FERREIRO, J.A., SHAKIR, K.M.M., BURMAN, K.D., and O'BRIAN, J.T. 1988. Increased metabolism of serum triiodothyronine after prolonged residence in Antarctica. *American Journal of Physiology* 254:E733-E739.
- REINBERG, A., LAGOGUEY, M., CESSALIN, F., LEGRAND, J.C., DELASSALLE, A., ANTREASSIA, A., and LABOBUEY, A. 1978. Circadian and circannual rhythms in plasma hormones and other variables of five healthy young human males. *Acta Endocrinology* 88:417-427.
- RONKAINEN, H., KAUPPILA, A., PAKARINEN, A., and KIRKINEN, P. 1985. Physical exercise-induced changes and season-associated differences in pituitary-ovarian function of runners and joggers. *Journal of Clinical Endocrinology and Metabolism* 60:416-421.
- RÖNNIKE, F. 1980. Årtidsfordeling for menneskets undfangelse i landene omkring Norskehave — med spesielt henblik på Nord — Norge. *Tidskrift for Norske Laegeforening* 101:151-155.
- ROSE, G. 1961. Seasonal variation in blood pressure in man. *Nature* 189:235-239.
- ROWELL, L.B. 1977. Reflex control of cutaneous vasculature. *Journal of Investigations in Dermatology* 69:154-166.
- SCHOLANDER, P.F., HAMMEL, H.T., LANGE ANDERSEN, K., and LÖYNING, Y. 1958a. Metabolic acclimation to cold in man. *Journal of Applied Physiology* 12:1-8.
- SCHOLANDER, P.F., HAMMEL, H.T., HART, J.S., LE MESSURIER, D.H., and STEEN, J. 1958b. Cold adaptation in Australian aborigines. *Journal of Applied Physiology* 13:211-218.
- SKRESLET, S., and AAREFJORD, F. 1968. Acclimatization to cold in man induced by frequent scuba diving in cold water. *Journal of Applied Physiology* 27:177-181.
- SMITH, R.E., and HORWITZ, B.A. 1969. Brown fat and thermogenesis. *Physiological Reviews* 49:330-425.
- TIMONEN, S., FRANZAS, B., and WICHMANN, K. 1964. Photosensitivity of the human pituitary. *Annales Chirurgiae et Gynaecologiae Fenniae* 53:165-172.
- TOUITOU, Y., SULON, J., BOGDAN, A., REINBERG, A., SODOYEZ, J., and DEMEY-PONSARI, E. 1983. Adrenocortical hormones, ageing and mental condition: Seasonal and circadian rhythms of plasma 18-hydroxy- and 11-deoxycorticosterone, total and free cortisol and urinary corticosteroids. *Journal of Endocrinology* 96:53-64.
- VAUGHAN, G.M. 1984. Melatonin in humans. *Pineal Research Reviews* 2:141-201.
- WARGENTIN, P. 1767. Uti hvilka månader flera människör årligen födas och dö i Sverige. *Kongliga Vetenskapsacademiens Handlingar* 4:249-258.
- YOUNG, A.J. 1988. Human adaptations in cold. In: Pandolf, K.G., Sawka, M.N., and Gonzales, R.R., eds. *Human performance physiology and environmental medicine at terrestrial extremes*. Indianapolis: Benchmark. 401-435.
- YOUNG, A.J., MUZA, S.R., SAWKA, M.N., GONZALEZ, R.G., and PANDOLF, K.B. 1986. Human thermoregulatory responses to cold air are altered by repeated cold water immersion. *Journal of Applied Physiology* 60:1542-1548.