PHYSIOLOGICAL ADAPTATION

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Edited by

C. LADD PROSSER

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Introduction

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THE interaction between living organisms and their environment is an old but very important problem for biologists. Organisms respond to environmental change in different ways according to the time during which the environmental change persists and according to the magnitude of the stress. In living organisms those alterations which favor survival in a changed environment are said to be adaptive. Similar adaptive variations of organisms may be genetically determined or they may be environmentally induced. This symposium deals largely with adaptive physiological changes in individual organisms and clones, and with the ways in which environmental change can bring about adaptive alterations in living organisms.

Animal physiologists have been mainly concerned with adaptive variation in the intact organism or in organ systems; they have paid most attention to variations induced by physical factors in the environment—temperature, light, oxygen, salinity. Microbial physiologists, on the other hand, have been most concerned with enzymatic changes induced by varying the nutrients in the environment, and have used the adaptive enzyme changes as a means of studying protein synthesis. Plant physiologists have been less concerned with environmentally-induced variations in individual plants but more with analysis of the wealth of genetic variation as shown by strains adapted to a variety of environments.

The aim of this symposium is to combine the different viewpoints of animal, microbial and plant physiologists. We are here not just to present experimental data but to learn to what extent the approach with one kind of organism can be applied to others, to learn whether certain basic patterns of response are common and whether others are peculiar to particular organisms. Our ultimate goal is to understand at the molecular level how environmental factors bring about adaptive changes in animals, plants and microorganisms. Also, as one aim of the Society of General Physiologists, we are trying to break across the intellectual barriers which separate physiologists according to the kind of organism on which they work.

An Approach to the Organ and Cellular Physiology of Adaptation to Temperature in Fish and Small Mammals

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LIVING SYSTEMS are continually reacting to changes in the environment. In some instances the changes are called 'stimuli' and the reactions of the living system, 'responses.' In other instances, where typically the reaction of the system is slow compared with the change in the environment, and where the changes in the system persist for appreciable periods after the environmental change, the system is said to have 'adapted' to the changed environment. Each specific characteristic such as blood pressure, heart rate etc. thus modified may be called an 'adaptate' (1).

Many different manifestations of the ability of a living system to modify its properties in accord with changes in the environment have been studied. These differ most obviously in the time course characteristic of each. Starting with the process called accommodation, in which the threshold of an excitable tissue rises when the strength of a stimulus applied to it rises gradually, one has an "adaptive' process measured typically in fractions of a second. Next, one has the decline in the response of a sense organ during the application of a constant stimulus, which is an 'adaptive' process measured in whole seconds or minutes. Slower still are the changes produced in whole organisms as a consequence of exposure to a change in environment. These are included in the term 'acclimation.' The change in the lethal temperature of an organism, which can often be produced by changing the temperature at which the organism is continuously living, is a typical example of an acclimation. The properties of single individuals are concerned here and the time course of the changes in the case of fish or small mammals is of the order of days—much longer than for adaptation of a sense organ or for accommodation but still small by comparison with the life span of the organism. Finally, there is adaptation in the classical evolutionary sense—a process involving large numbers of generations of a species and with a time course measured perhaps in millions of years. To the degree that these several adaptive processes reflect, for example, similar changes in the colloidal structure of protoplasm or its proteins, they are no doubt inherently the same. Yet the very great differences in the time courses characteristic of each imply that there are actually very fundamental differences in the mechanisms which are operative in the different instances.

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This symposium is to be concerned primarily with adaptive changes included under the term 'acclimation' as it was used above. In this first paper attention will be fixed on certain examples of acclimation to temperature which have been studied in some detail in fish and small mammals. Dr. Prosser, who has so ably organized this discussion, has suggested that I should refer particularly to the work of the several groups in Canada who, as it happens, have been especially concerned with fish and small mammals. This I will therefore do, although it will be necessary, in order to provide a context, to refer to much other work as well. We shall attempt here, as the other participants will attempt in their presentations, to discover in the actions and properties of the *parts* of organisms something about the ultimate, fundamental mechanisms which produce the gross manifestations observable in the whole animal as a consequence of acclimation.

A relatively large literature dealing with the reactions of organisms to the ambient temperature has developed recently as evidenced by the fact that in the last eight years six reviews on the physiology of heat and cold have appeared in *Annual Review of Physiology* (2–7). Much of this research is relevant in considerations of acclimation. It is indicative too of the interest in this area of investigation that reviews relating to it by Prosser (8) and Bullock (9) have appeared recently along wth the monograph by Precht *et al.* (10) and others by Adolph (11), and by Burton and Edholm (12). The June 1957 issue of the *Revue Canadienne de Biologie* is given over completely to papers dealing with effects of cold (Hart, 13; Sellers, 14; DesMarias, 15; Rossiter and Nichols, 16; Page, 17; Brown, 18; and Burton, 19) and there have been five conferences devoted specially to the reactions observable in man (20). A very useful discussion of acclimation for anyone wishing a general and inclusive approach is that given by Heilbrunn (21).

The present consideration will deal first with various processes related to the gross tolerance of animals to heat and to cold and then with the effects of temperature on two aspects of animal behavior, namely, temperature selection and general locomotor activity.

LETHAL TEMPERATURE

High Lethal Temperature in Mammals. That species differ in the ability to survive extremes of heat and cold has been appreciated ever since the present conceptions of heat and cold arose and the first thermometers were made (22). Examples of data obtained more recently on lethal temperatures in a few mammals are shown in table 1.

In this series man, cat and dog can withstand markedly higher environmental temperatures than the guinea pig, rabbit, rat and mouse, so that there are clearly very considerable differences among these species.

When the rectal temperatures at death are examined, however, it appears that

TABLE 1. LETHAL TEMPERATURES (°C)*

SPECIES	ENVIRONMENT (DRY)	RECTAL	SPECIES	ENVIRONMENT (DRY)	RECTAL
Man	59.4 (23)	43 (24)	Rabbit	41.7 (23)	43.4 (23)
Cat	56 (23)	43.5 (23)	Rat	38.6 (23)	42.5 (23)
Dog	56 (23)	41.7 (23)	Mouse	37.2 (23)	43.3 (24)
Guinea pig	43.9 (23)	42.8 (23)			

* Temperature tolerated indefinitely by half the animals exposed. Source in parentheses.

the species differences are rather small. An important generalization is therefore immediately suggested. It is that the real differences between the species are differences in ability to keep the body temperature from rising to lethal limits. The differences in tolerated environmental temperatures reflect, therefore, differences in efficiency of body cooling.

It is now well established for man that the ability to withstand high temperatures, which for the sake of precision may be considered to be temperatures above the thermally neutral temperature, is determined in part by previous experience at high temperatures. In a word, acclimation to high temperatures occurs. This acclimation is manifested for example by the maintenance of lower body temperatures, lower pulse rates and readier sweating under standard exposures, conditions and activities of heat stress (11). It is of interest however that 'mere residence in a hot climate falls far short of acclimatizing men for work in heat' (4). Evidently some degree of muscular activity above basal levels plays a significant role in triggering the adaptive response to the stressor. It is necessary to 'exercise' the regulatory machinery if acclimation is to occur.

While there is a large literature dealing with the responses including acclimation of the human to heat, data concerning the smaller mammals are scanty as Hart (13) notes.

There is, of course, in homeotherms a definite nervous center anatomically situated close to the vasomotor center in the hypothalamus which exerts primary control over the body temperature. It has become abundantly clear, however, that the various parts of an animal's body may differ greatly in temperature, and along with this finding it has been established that there are local responses taking part in temperature regulation which occur independently of the thermoregulating center. Some of these responses may involve specific temperature receptors but some may not. It is also clear that many organisms have characteristics of *behavior,* which enable them to survive high environmental temperatures. Cats lick their fur thus wetting it and undoubtedly obtaining evaporative cooling in this way. Desert rodents withdraw from the heat of the ground surface in the desert to burrows (25). Stinson and I (26) have observed in the deer mouse, and Smith and I (27) have observed in the lemming, that voluntary locomotor activity is greatly decreased at high temperatures thus decreasing the heat production and so reducing the heat load of the individual. In a warm environment too, mice

minimize the use of such heat-conserving devices as the huddle (28) and the compact, thick-walled, well-built nest (29).

Perusal of the literature dealing with acute responses to heat (for example, the *Annual Review of Physiology*) indicates that a great variety of different processes and properties have been examined experimentally. Undoubtedly many of the quantities which are being measured will undergo changes in acclimation. Considerable attention has been directed to the possibility that the water content of protoplasm or the nature of its fat content might be factors determining its lethal temperature (21). These considerations may have significance in relation to any one type of cell. In a multicellular organism, however, it is already evident from the variety of processes studied, that a great many different kinds of cells are involved in the response to heat. These cells have quite different parts to play in the overall regulation of temperature and they are not all at the same temperature. Altogether therefore it is difficult to believe that any single characteristic, such as the kind or quantity of fat or the water content of all of the cells collectively in a multicellular organism, could be of great significance in relation to heat tolerance. As a matter of fact, Adolph (11) makes the point that dehydration, even though it may occur typically in animals exposed to high temperatures, is not a necessary consequence of this, and that acclimation to high temperature does not mean also acclimation to lowered body water. In fact acclimation to dehydration has not been demonstrated, nor does dehydration assist in the tolerance to high temperature. There would be, I think, a general tendency for workers in this area to ascribe the tolerance limit ultimately to some particular few cells, probably in the central nervous system (24, 30). Not the whole central nervous system certainly, because it has been shown that normal responses to electrical stimuli can be evoked in the cortex of a cat when the cortical temperature is raised by radiant heating to 50°C, which temperature is probably 5°C higher than the lethal colonic temperature of this animal (31).

In general, then, high temperatures are survived by mammals provided the animals are able to keep the body temperature below a lethal limit. I have not seen evidence that this lethal body temperature can be raised by acclimation and the improved tolerance of high environmental temperatures, which has been recorded as a consequence of exposure to elevated temperatures, appears to be due to an increase in the efficiency with which the body temperature can be kept below the lethal limit. There are five major factors involved in this: *1*) heat produced in the organism; *2*) heat absorbed from the environment; *3*) transfer of heat to the periphery; *4*) evaporative cooling arrangements; *5*) ability to drop the body temperature below 37° when the environment is cold, so that the periphery of the body may then be used as a heat sink for short periods when the environment is hot.

If we assume for the present that the individual adaptates, seen as a consequence of acclimation as measured by the gross ability of the organism to survive an

elevated environmental temperature, assist in producing the increased tolerance of heat, then it could be expected that each of these individual adaptates will be of significance in connection with one or several of these major factors. Some indications of this are now appearing.

As far as heat production is concerned, Bazett (32) was satisfied that acclimation at high temperatures in humans results in a reduction of the amount of heat produced, that is at high temperatures the basal metabolism is reduced. This is difficult to establish as he pointed out, because total heat production includes that from muscular exercise and an individual transferred to a hot environment typically lowers his total metabolism by reducing voluntary movement to a minimum. The same thing is seen in small mammals where, as indicated above in experiments with deer mice and lemming, we see almost complete cessation of locomotor activity at air temperatures of 30°C and above. The reality of a drop in heat production as a consequence of exposure to heat is made more certain by three separate reports (33–35) that white residents in the tropics have a lower basal metabolism than they do when resident in temperate regions; by the measurements (36) made on a woman normally resident in the temperate zone on a journey to the tropics; by the observation (37, 38) that in women (but not in men) exposure to heat for a few hours results in a drop of as much as 15% in the basal metabolism; by the finding (39) that exposure of rats to unusually high temperatures for several days causes an apparent depression of the activity of the thyroid gland. When the temperature of the tissues is elevated cellular damage occurs as indicated by the leakage of certain components into the circulation of isolated perfused organs (40).

That radiation from the environment can cause appreciable increases of the heat load in man has now been well established. It is shown by Adolph (11) and by Burton and Edholm (12) that clothing, by providing an insulating layer, can significantly reduce the heat increment received from the environment. The functional significance of the fur of an animal in this connection has recently been strikingly demonstrated for the camel by Schmidt-Nielsen et al. (41). In hot desert climates this animal has a wool insulation varying in thickness from 30 mm up. Temperatures as high as 70°C have been recorded at its outer surface and when the fur was removed from several animals an increased heat load was indicated by an increase in their water consumption of more than 50%. There is also a report (42) that skin texture in man is important in determining the heating of the skin surface by thermal radiation.

The fact that when an organism is exposed to a heat stress it increases the flow of heat to the periphery by increasing blood flow there, and especially to the skin and superficial tissues (43), is attested to by a great variety of experimental evidence. It is of interest to record here that this operation of the circulation as part of the heat regulatory mechanism shows acclimation (44). After 4 days in a hot environment the blood flow through the fingers of a man was 10–25%

above that seen on the first day. When now exposed to cold the flow remained high. It required several days of exposure to cold to restore the original pre-heat level. There are undoubtedly other examples. Dill, writing in 1938 (45), remarks that the heat regulatory apparatus certainly improves with training. In other words it acclimates.

Many of the existing investigations of body temperature regulation in relation to heat stress deal with aspects of evaporative cooling. It is known in general that this takes place in two ways, one from the surface of the body generally, the other from the nasopharyngeal passages and mouth. The latter is associated with the characteristic shallow high frequency (10 times normal resting) respiratory movement called panting (23, 46, 47). In some animals such as the dog, the panting is almost completely responsible for evaporative cooling. In man evaporation from the body surface in general is the important source of cooling. In the cow (4–8) both are appreciable though the movements of panting do not reach such a high frequency relatively as in the dog. In still other animals like the rat and mouse, evaporative cooling is hardly significant under any circumstances unless an external source of water is available with which the animal may wet its fur (23). Human children brought up in warm areas of the world develop more sweat glands than do those brought up in temperate or cold areas (49). Men acclimated to heat sweat more readily and more profusely than do nonacclimated individuals (50). As a consequence of acclimation too, sweat produced contains less chloride than does the sweat of a nonacclimated person (45). While the exact nature of the stimulus to sweating is not known, nor the manner in which the nervous connections and nerve centers involved in its control operate, integration of it with the other provisions for heat regulation is conceded to reside within the central nervous system.

Indications that hormonal readjustments are required as part of acclimation to heat are also appearing. The activity of the thyroid gland in the rat appears to be depressed by exposure of the animal to heat (39). Seasonal differences in the response to thyroxine have been reported (51), the hormone having less effect during the warm season than in the cold. There are also seasonal differences in the plasma antidiuretic activity (52) produced in man by a standard heat stimulus—it is three times higher in autumn—and there is a seasonal change in urinary output of 17-ketosteroids (53), the output being lower in summer than in winter. Selye's compendium on 'Stress' (54) contains a few other references.

It has recently been reported (41) that the camel uses a relatively variable body temperature in contending with the problem of heat regulation it faces in the desert. Apparently during the night when heat can be lost to the environment with little use of evaporative cooling, the body temperature may be allowed to fall to approximately 34°C. Then during the day when environmetal temperatures rises and the radiant heat adds to the heat load, the body temperature is

allowed to rise to 40 °C. It is thus unnecessary for the animal during the day to lose to the surroundings all of the heat it gains by production and by radiation, and so it conserves the water which would otherwise be required to do this by evaporative cooling.

Low Lethal Temperature in Mammals. Turning now to the question of acclimation to cold, one finds relatively a wealth of data concerning small mammals. Survival ability at low temperatures certainly shows major variations. At −35 °C for example survival times as given in table 2 have been observed. Lethal colonic temperatures do not differ so widely. The values in table 3 have been obtained. The exact significance of these colonic temperatures is in doubt however since Andjus (61) first demonstrated that a certain proportion of rats cooled to body temperatures of 0–2 °C in a special way could be resusciated after heart and breathing had been stopped for three quarters of an hour. Subsequent improvements in technique have made possible recoveries of 75 to 100% and similar cooling has been brought about in hamsters, dogs (see 62, 63 for references) and monkeys (64). Quite evidently animals can survive after cooling to temperatures which heretofore have been considered lethal. Refinements in the criteria to be used in defining lethal temperatures are required here.

While some mammals at least can be cooled for a short while, far below their normal body temperature for the species and yet recover normal function, there is no suggestion as yet that normal function is still observed at temperatures more than a few degrees below normal body temperatures, except in hibernators (14). The latter do appear to have fundamental characteristics in respect to resistance to cold which distinguish them from the common type of homeotherms. This is illustrated for example by the fact that nerve conduction in the hibernating mammal persists at temperatures very appreciably lower than those which permit normal function in the common homeotherm (65). Similarly the heart of a hibernating mammal survives lower temperatures than does the heart of a nonhibernating species (66). Thus the primary emphasis in relation to survival

TABLE 2. SURVIVAL TIMES AT − 35°C

SPECIES	HOURS	REF.	SPECIES	HOURS	REF.
Mouse	0.4	(55)	Chicken	3.3–16.0	(55)
Canary	0.6	(55)	Pigeon	22.0–78.0	(55)
Rat	0.75–2.0	(55)	Lemming	circa. 100	(56)
Rabbit	3.53.5 −6.5	(55)	Eskimo dog	indefinitely	(57)

TABLE 3. COLONIC TEMPERATURE (°C) AT DEATH

SPECIES	TEMP.	REF.	SPECIES	TEMP.	REF.
Marmot	0– 5	(58)	Cat	14–16	(58)
Ground squirrel	2– 4	(59)	Rabbit	4–14	(60)
Hamster	2– 4	(59)	Dog	18–20	(58)
Rabbit	3.5 −6.5	(55)	Man	24–26	(58)

in the cold is on the ability to maintain the temperature of the body at the normal value. This depends upon an exact balance between the rate of heat gain, which is primarily from metabolism, and the rate of heat loss, which depends on the insulation. The organism produces heat which it loses to the environment through an insulating layer (skin, fat, fur etc.) whose properties as an insulator are to a degree under the control of the organism. Because the effective amount of insulation can be varied (by varying blood flow to periphery, surface temperature, extending limbs etc.) there is a range of temperature, the thermoneutral range, in which the body temperature may remain fixed at a constant minimum heat production by variation of the effective insulation. As the external temperature is lowered the insulation finally attains a maximum value (at the critical temperature) and at lower temperatures still, maintenance of the body temperature requires increased heat production.

Figure I (13) illustrates the general situation as it was set forth originally in the work of Scholander, Hock, Walters, Johnson and Irving (57). In this figure the metabolism of an animal is plotted against the temperature of the environment. The body temperature is T at which temperature the metabolism is A. Initially, as the environmental temperature is decreased the metabolism remains constant, the increasing tendency to lose heat being compensated for by an increase in the effective insulation of the animal. At B the limit to the increase in the insulation is reached, and if the environmental temperature is lowered below this critical value, the body temperature can be maintained only by an increased rate of metabolism which then in the warm-acclimated animal rises along the line BC. At C the maximum rate of heat production is reached and so temperature L_1 represents the lowest temperature tolerated by this individual. It is to be noted that line CB extrapolated to a metabolic rate of zero cuts the temperature axis at the body temperature T, that is, no metabolism, obviously, is necessary at environmental temperature T in order to keep body at temperature T.

If the maximum insulation which the organism is able to bring into operation is increased, let us say as a consequence of acclimation to cold, then the body tem-
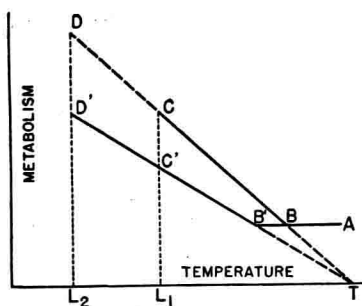

FIG. I. Metabolic vs. insulative acclimatization. L_1 and L_2 represent low temperature limits of animals acclimatized to warm and cold climates. Extension of limits from L_1 to L_2 may be carried out through insulative or metabolic adjustment. In insulative acclimatization there is lowering of the critical temperature from B to B^1 and change in slope from BC to BC^1D^1, so that the energy expenditure is not greater at $L_2(D^1)$ than at $L_1(C)$. In metabolic acclimatization there is extension of the curve BC to BCD with greater energy expenditure at $L_2(D)$ than at $L_1(C)$. From Hart (13).

perature can be successfully maintained without an increase of the metabolic rate down to an environmental temperature of B^1 instead of only to B. As the temperature of the environment is further lowered, the metabolism rises again, this time along line $D^1C^1B^1$ which in turn when extrapolated to zero metabolism intersects the temperature axis at the body temperature T.

In this example the maximum metabolic rate of the warm-acclimated animal is taken to be the same as the maximum metabolic rate of the cold-acclimated one. However, because of the greater insulation in the cold-acclimated animal it is able to tolerate a lower environmental temperature, L_2, than does the warm-acclimated animal. Alternatively, acclimation to cold could lower the minimum temperature tolerated without a change in insulation by raising the maximum metabolic rate which is possible, that is, by extending the line BC to D. This latter adjustment, metabolic acclimation, does not involve a change in the slope of the line relating metabolism and temperature nor does it affect the critical temperature. When insulative acclimation occurs, both the slope and the critical temperature are lowered. The increase in heat production as measured by the metabolism necessary to maintain the body temperature for a given drop in the external temperature (i.e. the slope of such lines as BC and B^1C^1 in fig. 1) provides a measure of the average effective insulation. The smaller the metabolism increase required for a given drop in temperature, the better the insulation.

It is not necessary here to review the impressive body of data which can now be usefully considered from this viewpoint for this has been done recently by Hart (13) who with the Scholander-Irving group has been one of the principal investigators in this field. Suffice it to say that no conspicuous species differences in ability to produce heat for the maintenance of body temperature at low environmental temperatures have been recognized. Specifically it has been found that the metabolic rate of arctic animals is no higher than that of temperate ones. On the other hand it is clear that there are large differences in effective insulations between species and that these are quite sufficient to account, for example, for the ability of the arctic species to survive (57). This situation contrasts with that reported for fish where arctic forms typically have a higher rate of oxygen consumption relatively than species from moderate climates (129).

It is now well established that subjection of many homeotherms to moderately low temperatures (say 0° to 10°C) lowers the low temperature survival limit (12). This point is illustrated in figure 2 (13) which shows the survival times of several species at various temperatures. The temperature at which the organisms had been living prior to the experiment on survival time is indicated for each curve. Acclimation to low temperatures undoubtedly occurs in the two species of deer mice, in rats, and in mice, and the limiting temperature may change by as much as 10° to 20°C. The extension of the low temperature limits is made possible by the development of an ability to produce heat at higher rates and for
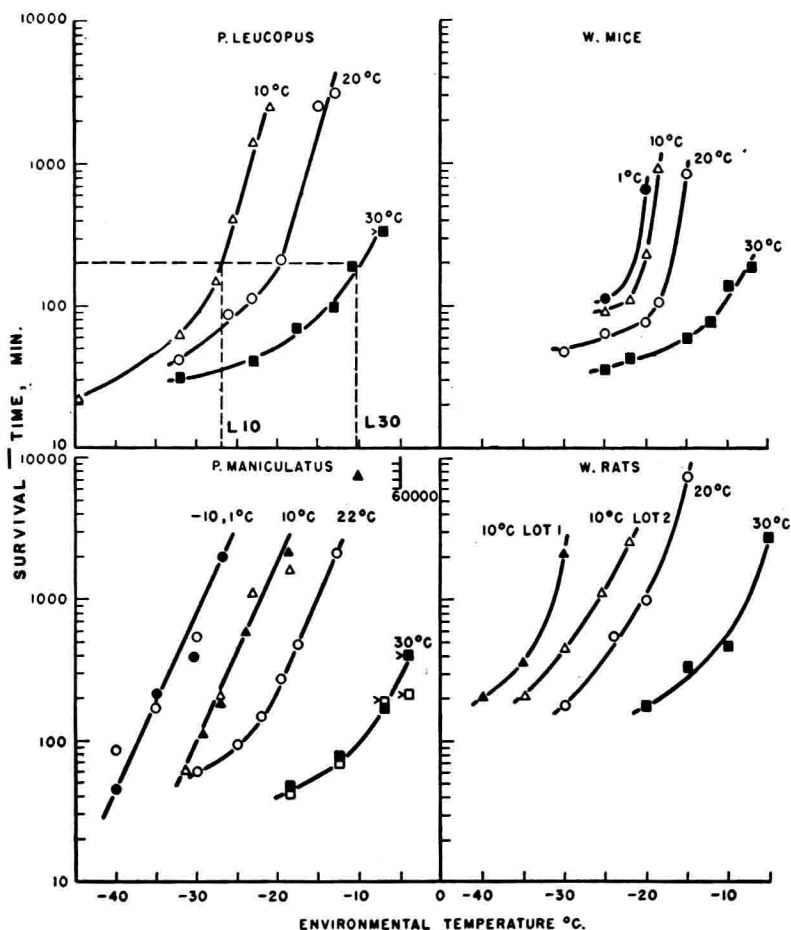
FIG. 2. Survival time in relation to environmental temperature of mice and rats acclimated to various temperatures. L_{10} and L_{30} are low temperature limits of deer mice acclimated to 10° and 30° respectively for a survival time of 200 minutes. From Hart (13).

longer periods. There are no instances thus far where exposure to *cold* increases the *maximum* insulation. There is on the contrary in many instances an actual decrease of the effective insulation during acclimation to cold owing to the increase in peripheral circulation required to prevent cold damage in the periphery.

Hart draws attention to the literature showing that low temperature tolerance undergoes a seasonal variation, the greater tolerance occurring during the winter. This might be expected from the lowering of the environmental temperature in winter. The nature of the adjustment with the seasons (acclimatization) is not the same however as that which takes place in response to an exposure to cold.

The data in figure 3 (13), which show the relation of oxygen uptake to temperature, provide two and perhaps three examples where the insulation is clearly greater in winter than in summer. Thus, while exposure to cold does not increase the *maximum* insulation, this *maximum* does change with the seasons in a number of instances. It is evident that these seasonal effects cannot be accounted for by the temperature changes associated with the change of season, and the pertinent factor in this connection, possibly the changing day-length, has yet to be demonstrated.

For our present purpose however, it is sufficient to know that both heat production and effective insulation are subject to change by acclimation or acclimatization. To what degree can these gross changes be traced down to organs and cells?

The most obvious immediate response of man or animal to cold is muscular activity, usually of that special kind known as shivering. It results in the production of heat which contributes appreciably to the maintenance of body temperature (see for example 67, 68). It is to be noted, however, that in lemming and rabbit (69) and in mice (70) the heat produced in the muscle exercise of locomotion is not a satisfactory substitute for that produced during shivering. Apparently the movement of the limbs and body lowers the effective insulation sufficiently to dispose of practically all the extra heat being produced. The loss is relatively less in mice acclimated to low temperatures than in animals acclimated to high temperatures.

Rats acclimated to 30°C shiver when first transferred to 6°C (71, 72). Over a period of 4 weeks, however, the shivering decreases and disappears so that
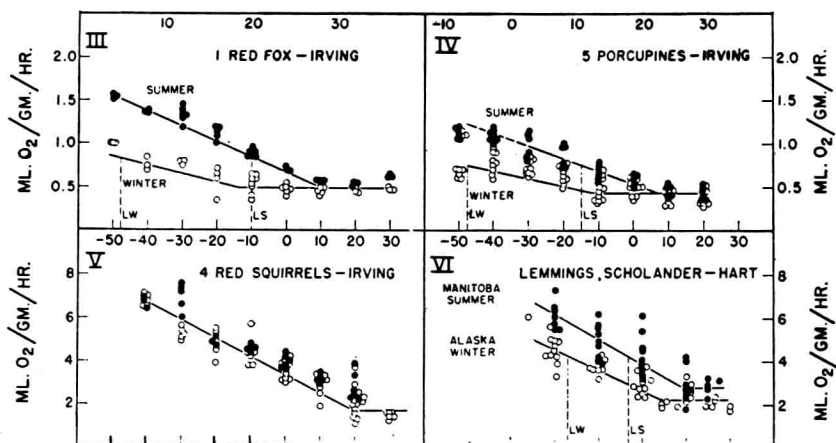

FIG. 3. Temperature-metabolism curves for mammals acclimatized to summer or laboratory (•) and winter (○) temperatures. Broken vertical lines for fox, porcupine and lemming show extension of winter temperature limits (*LW*) below summer limits (*LS*) for same elevation of metabolic rate (insulative acclimatization). From Hart (13).