THE ACCLIMATIZATION OF WHITE RATS TO THE CHRONIC INTERMITTENT EFFECT OF UNFAVORABLE TEMPERATURES

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In the many investigations devoted to the experimental study of the problem of acclimatization laboratory animals are usually subjected to constant high or low temperatures for a more or less prolonged period. However, such an experimental scheme creating a pattern of acclimatization in different climatic zones has little in common with the effect of an unfavorable microclimate on man in those conditions. Intermittent thermal action and adaptation to it by animals has been studied only in short-term trials with predators and primates [1]. The intermittent action of cooling has been studied with rats, but only for 4 weeks [10].

This work presents some findings on the effect of chronic (up to 6 months) intermittent thermal and cooling action on white rats.

EXPERIMENTAL METHODS

For 5-6 h per day 20 rats were placed in a chamber in which the temperature was automatically maintained at 38-40°. The experiment lasted 2, 4, or 6 months after which the experimental animals and an equal number of controls were killed by rapid decapitation. A similar plan was used in a second experiment in which the experimental rats were placed in the chamber at a temperature of 5-11° (usually 7-8°). To exclude the possibility of mutual heating by the animals huddling together, the chamber was divided into cells in each of which 1 or 2 rats were placed.

Thermistor detecting elements of the TEMP-4 apparatus were used to measure the rectal temperature (at a depth of 2 cm) and the skin temperature of the tail. During each experiment the rats were weighed twice a month. The kidneys, adrenals, spleen, liver, and lungs of the slaughtered animals were also weighed; in the last two organs the total lipid content was estimated from the weight lost by the dry tissue during ether extraction in a Soxhlet apparatus.

RESULTS

During single thermal action, the rectal temperature of the rats at the start of the experimental period increased daily compared with the temperature measured before they were placed in the chamber. During the first 10 days the increase was on average for the group 1.1°, one day it reached 2.4°. The rats in the chamber lay motionless with eyes closed and with a sharp increase in the rate of respiration. According to findings in the literature [4], the continuous action of the same temperature, 38-39°, resulted in the rats dying of hyperthermia in the first few days if they had not first been adapted to the continuous action of a temperature of 35° for 7 weeks.

In our experiment regular repeated heating led to clear acclimatization of the rats. Thus, during the 6th month of the experiment in the same group of animals the mean rectal temperature after 24 days of daily thermal exposure was the same as before exposure. On average, there was no change in temperature in 6, an increase, but not more than 1°, in 12, and a decrease of 0.4-2.4° in 6. In the first 2 months of the experiment no such decrease in temperature was observed.

The change in temperature caused by each thermal exposure of the tail skin in rodents with a specialized zone of heat irradiation is quite different during the same period [1, 17]. In the initial period the increase in skin temperature was on average for the group on different days from 0.4 to 4.7°, and on average for 10 days 2.6°. By the end of the experiment the average increase in temperature during 24 days increased to 4.8° ($P < 0.05$), while 18 times the variation observed in the group was more than 4° (up to 7.7°). Thus, adaptation of rats to intermittent heating was shown by the increased reaction of the skin temperature. It may be assumed that the increase in heat irradiation associated with this also was a most important cause of stabilizing the rectal temperature.

In control rats placed in a similar chamber but not subjected to heating, the rectal temperature in the same 5 h fell on average by 1-1.1°. Consequently, the fact that in the heat chamber the temperature of the acclimatized rats was on average practically unchanged can be regarded as a latent effect of heating, although less marked than before acclimatization when the same action caused an increase in body temperature. Cases of a reduction in temperature as a result of thermal exposure are of special interest. A similar reaction, which we consider to be the result of hypercompensation for heating by increased heat irradiation, has been described in nonacclimatized rats [8]; in the latter however it was never observed with an environmental temperature above 32°. Our data show that a considerably higher environmental temperature may cause such a paradoxical reaction, but only after adaptation to its intermittent action.

Rats placed in a cold chamber maintained their activity. Each exposure at the start of the experiment led to a reduction in rectal temperature of, on average for the group, of 0.8-2.9°, and, on average for the 10-day observation period, of 2.1°, which differs significantly ($P < 0.001$) from the reduction in temperature observed during the same time in control rats in an uncooled chamber, 1.1° on average. After only 2 weeks however the reduction in the rectal temperature of the trial rats was no greater than in the controls, never exceeding 1.5°, on average 1° in both groups. Thus, the temperature in the chamber rapidly ceased to have any more pronounced cooling effect than simple transfer of the animals from the group cages of the animal house to the laboratory at 19-20°. The state of adaptation to the intermittent action of cold is also maintained longer.

It can be assumed from the literature that the chief mechanism of this acclimatization is associated with adaptation of the heat irradiation processes. The reduction in heat irradiation as a reaction to each action of the cold in acclimatized rats was however maintained. The temperature of the tail skin fell after exposure by, on average for the group, 4.7-8.3°, mean 6.5°; $P < 0.001$, whereas in control rats in a chamber at room temperature it even increased somewhat. At 15-30 min after the rats were removed from the cold chamber the skin temperature was 4-7° higher than before they were placed in the chamber which evidently reflects the reactive hyperemia which alternates with the stimulation of vascular spasms caused by the cold.

Periodic cooling did not cause the destructive changes described [11] during the continuous action of the same temperatures. Neither the heating nor the cooling action in our trials produced the regular change in the weight curves of experimental rats compared with the corresponding controls which is usually observed [5, 12, 13] during continuous acclimatization. The weight coefficients of the lungs, liver, kidneys, and spleen did not change either, although during the continuous action of cold an increase in the weight of the liver and kidneys has been described [11].

The relative weight of the adrenals after thermal action for 2 months was greater than in control rats by 110 and after 4 months by 70%; after 6 months it had fallen by 45%. After cold action for corresponding periods values varied by only +21, +38, and −4%. Thus, the initial increase in weight of the adrenals in both cases varied with a more or less marked fall or at any rate by a return to the control level. It can be assumed that these variations are associated with the participation of the adrenal cortex in the adaptation reaction of the organism. More pronounced changes were caused by heating, to which the rats were less adapted than to moderate cooling, and which, judging by the behavior of the animals, produced more pronounced stress. On the other hand, the hormones of the medullary layer of the adrenals play a very important role in the chemical thermoregulation of animals during adaptation to cold [6, 7, 9, 15, 16]. During the continuous action of a temperature about 0° [17] and during continuous or periodic action of a temperature of 6° for 4 weeks [10] hyperplasia of the adrenals was noted in the rats.

The thermoregulatory heat production stimulated by catecholamines is brought about by increased mobilization and oxidation of the free fatty acids [9, 14, 15]. Considering the special role of the liver and lungs in the thermogenic oxidation of fats, changes may be expected in the lipid content of those organs during cooling or heating. However, findings on this question in the literature, relating to conditions of continuous acclimatization, are scanty and contradictory [2, 3, 19, 20], but we have not found in the literature any data on the effect of chronic intermittent cold or heat action on fat metabolism.

In our trials we detected no consistent change in the total lipid content of the lungs during cold action. The relative lipid content of the liver was after 2 months 13.3, after 4 months 10.4, and after 6 months 10.9% compared with 14.6, 12, and 11,2% in animals of the corresponding control groups (difference significant only after 4 months; $P < 0.01$).

As a result of thermal action lasting 2 months, the lipid content of the lungs increase to 14.1% compared with 10.4% in the control ($P < 0.01$). After 4 months this difference practically disappeared (the lipid content of the lungs was 13.9 and 12.4% respectively; $P > 0.05$). After 6 months the lipid content of the lungs was no different from that of the control. In the liver, on the other hand, the most pronounced fall in lipid content was observed after 6 months of thermal action; in the experimental rats it was 8.7 and in the controls 12.7%, or recalculated on the whole organ 236 and 449 mg respectively ($P < 0.005$).

Thus, intermittent temperature action, especially heat, despite developing acclimatization, but possibly also in association with it, causes distinct variations in lipid metabolism.

SUMMARY

Experiments on albino rats regularly exposed over a period of 6 months to temperatures from 38 to 40°C for 5-6 h daily or from 7 to 8°C for 5-6 h every other day showed the process of acclimatization to such intermittent temperature conditions and certain reactions accompanying this process.

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