Habituation of thermal sensations, skin temperatures, and norepinephrine in men exposed to cold air

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Leppäluoto, J., I. Korhonen, and J. Hassi. Habituation of thermal sensations, skin temperatures, and norepinephrine in men exposed to cold air. J Appl Physiol 90: 1211–1218, 2001.—We studied habituation processes by exposing six healthy men to cold air (2 h in a 10°C room) daily for 11 days. During the repeated cold exposures, the general cold sensations and those of hand and foot became habituated so that they were already significantly less intense after the first exposure and remained habituated to the end of the experiment. The decreases in skin temperatures and increases in systolic blood pressure became habituated after four to six exposures, but their habituations occurred only at a few time points during the 120-min cold exposure and vanished by the end of the exposures. Serum thyroid-stimulating hormone, total thyroxine and triiodothyronine, norepinephrine, epinephrine, cortisol, and total proteins were measured before and after the 120-min cold exposure on days 0, 5, and 10. The increase in norepinephrine response became reduced on days 5 and 10 and that of proteins on day 10, suggesting that the sympathetic nervous system became habituated and hemoconcentration became attenuated. Thus repeated cold-air exposures lead to habituations of cold sensation and norepinephrine response and to attenuation of hemoconcentration, which provide certain benefits to those humans who have to stay and work in cold environments.

body temperature; cold adaptation; cold sensations; epinephrine; hemoconcentration; norepinephrine; thyroid hormones; thyroid-stimulating hormone

WHOLE BODY COLD-AIR EXPOSURE in laboratory conditions has been used previously in several studies on human cold adaptation. It has been observed that the decrease in deep body temperature was greater after cold-air exposures than before them (3, 16, 18), but decreases in mean skin temperature remained unchanged (3). Cold-induced stimulation of the metabolic rate has also been found to be attenuated after repeated cold exposures (3, 11, 16). Only a few studies have reported peripheral skin temperatures or cold sensations during repeated cold-air exposures. Nighttime toe and arch temperatures were found to be higher after 14 days of daily cold-air exposures than before (18), but no differences were found in face, finger, or forearm temperatures (16, 18). Subjective sensations of cold diminished after 3 days in the cold room (7), and discomfort caused by cold-air exposure, as well as shivering, started later or at 0.5°C lower body temperature after four to seven cold-air exposures (2). Thus repeated cold-air exposures in laboratory conditions appear to lead to adaptive changes such as central hypothermia, decreased metabolic response, delayed onset of shivering, and reduced cold sensations. These findings bear some correlation to those seen in cold-exposed Australian aborigines or Lapps (for reviews see Refs. 19, 35).

The role of hormones regulating metabolism and energy production in cold adaptation has been little studied in humans. After a single cold-air exposure, serum thyroid hormone, thyroid-stimulating hormone (TSH), epinephrine, and cortisol levels remain unchanged (20), but serum norepinephrine levels increase, and serum prolactin and growth hormone levels decrease (20, 23, 24, 26, 34). When a 30-min cold-air exposure was repeated 80 times during 2 mo, serum thyroxine, TSH, and epinephrine levels remained unchanged, but serum free triiodothyronine decreased, and the plasma norepinephrine increase was attenuated (11). In other studies, serum total triiodothyronine decreased (32) and serum norepinephrine response increased after repeated cold-water immersions (36). The decrease in serum-free triiodothyronine occurs also under natural winter conditions, e.g., on an Antarctic base (30) or in northern Finland (21). Although serum triiodothyronine levels decreased during long-term cold exposures (11, 21, 30, 32), the production and tissue availability of triiodothyronine have been shown to increase (30). High-serum norepinephrine and triiodothyronine production in response to cold exposures is useful, because both hormones are known to increase metabolic rate.

The cold stimuli used in previous studies dealing with the adaptation of physiological responses to cold have varied considerably as to time and intensity, e.g., the heat losses have been 6–13 kJ/kg body wt when cold-air exposures have been used (2, 10, 11, 18), but in cold-water immersions they have been 21–26 kJ/kg (1, 27, 36), assuming that the water temperature at the end of the immersion. The durations of the experimental cold exposures re-
ported in the literature have also varied much, from 4 (2) to 80 (11) days.

Physiological processes by which an individual adapts to his or her environment are habituation, acclimatization, or acclimation (35). Habituation involves the diminution of normal responses or sensations to repeated stimuli and may thus protect the individual from possibly harmful effects of cold exposures, for example. Extensive epidemiological and experimental studies show that cold-induced increases in blood pressure, norepinephrine secretion, and hemococoncentration are risk factors that are believed to explain partly the excess mortality due to cardiovascular diseases in winter (4, 6, 17, 25). Also, decreases in skin temperatures and unpleasant cold sensations lead to poor safety and decreased efficiency in cold work (9, 15, 28).

Previous information about the time course of the development of these habituation processes during repeated cold-air exposures in humans is scanty (see above). Therefore, we sought answers to the following questions: Do the thermal sensations, body temperatures, blood pressure, and hormonal responses become habituated to the repeated cold-air exposures? Can the different time courses of the habituation processes explain the underlying mechanisms? Can the habituation to cold air under laboratory conditions be used to improve physical performance in the cold? As a stimulus, we used a daily 2-h exposure to cold air for 11 days.

METHODS

Subjects. Six healthy male Caucasian volunteers gave their informed consent for the study. Mean (± SE) age, weight, height, and body fat (as measured from the skinfolds) were 20.5 ± 0.2 yr, 66 ± 3 kg, 174 ± 2 cm, and 17 ± 1%, respectively. The experiments were carried out between June and August at the Department of Physiology with outdoor temperatures between 8 and 22°C. The subjects were familiarized with the experimental procedures (cold chamber, insertion of thermodes, and blood sampling) before the tests. The experimental protocol was accepted by the Ethics Review Board of the Medical Faculty, University of Oulu.

Experimental protocol. Our present study consisted of a daily 2-h cold exposure repeated on 11 successive days. The subjects were dressed in shorts during the cold exposures. Physical training, sauna baths, tobacco, and alcohol intake were not allowed during the study and 2 days before it. The subjects woke up at 6 AM, had a light breakfast, and arrived at the Department of Physiology at 8 AM. On the study days, the following recordings were performed. Eighteen skin thermodes and one rectal thermode (Yellow Springs Instruments) were put in place, and the subjects were taken to a room with a temperature of 27–28°C (range) for 30 min. Body temperatures were recorded continuously by a Hewlett-Packard data logger 9000/216. In Figs. 1–3, temperatures are shown at time points 0, 30, 60, and 120 min. A physician recorded blood pressure by a sphygmomanometer and recorded heart rate every 15 min. At 25 min in 27–28°C, oxygen consumption was measured by Morgan Oxylog. Afterward a venous blood sample was taken on study days 1, 5, and 10. At 30 min, the subjects were taken to a cold chamber (Vötsch) with a preset temperature of 10°C, air velocity <0.2 m/s, and air humidity of 2–4 g/m². The actual air temperature was recorded by a dry bulb globe thermometer in the cold chamber. The mean of the measurements was 9.78 ± 0.30°C at the beginning of the cold exposure and 10.08 ± 0.15°C at 120 min. The difference was 0.31 ± 0.20°C and did not differ significantly among the experimental days. The subjects sat on netted chairs in the cold chamber for 120 min, and oxygen consumption, blood pressure, and subjective cold sensations were recorded every 10–30 min, starting from the time point of 5 min before the cold-air exposure (~5 min). They were asked about shivering and their cold sensations in the following locations, general, hand, feet, and face, according to the following validated scale: 1 = cold, 2 = cool, 3 = slightly cool, 4 = neutral, 5 = slightly warm, 6 = warm, and 7 = hot (33). Tissue conductance, heat debt, and mean skin temperature were calculated from the equations given (1). Another blood sample was taken at 120 min immediately after the subjects left the cold chamber.

Assays. Blood samples were centrifuged, and 20 μl of 1 M NaOH per milliliter were added for the HPLC measurement of epinephrine and norepinephrine with interassay coefficient of variation <5% (5). Serum total triiodothyronine, thyroxine, and cortisol were measured by using radioimmunoassay kits from Farmos (Turku, Finland), and TSH was measured by using a kit from Corning. Serum free fatty acids (FFAs) were measured by autoanalyzer (Technicon), and total proteins were measured by a biuret method. The analyses were performed according to the instructions provided by the manufacturers mentioned above. Each analyte was assayed in one assay with an interassay coefficient of variation <8%.

Statistical analysis. The arithmetic mean ± SE was calculated for all data. The effects of cold exposures on the variables temperature, sensation, and hormone measures were analyzed separately through a two-way analysis of variance for repeated measures (BMD P2V) as day of exposure (days 0–10) and time of exposure in minutes as factors 1 and 2, respectively. Comparisons of each exposure time against the respective day 0 value for exposure days 1–10 were carried out with the method using contrast of trials (deltas). A statistically significant reduction in this test was regarded as an indication of the presence of habituation.

RESULTS

Rectal and skin temperatures, cold sensations, metabolic rates, and blood pressures. Table 1 shows the results of the repeated-measures analysis of variance performed on body temperature (10 locations) and thermal sensations (4 locations) during the 11-day experiment. Significant differences in time factor 1 (day) were detected in mean skin, forearm, and chest temperatures and in all of the thermal sensations, suggesting that these variables have become habituated as demonstrated below. As the cold exposure resulted in great decreases in skin temperatures and subjective thermal sensations (units) on every experimental day, the differences in the variables for time factor 2 (minute) were always highly significant. There was a significant interaction of forearm and thigh temperatures, indicating that the decreases in the temperatures were delayed on days 5–8.

The mean preexposure rectal temperature on day 0 was 37.1 ± 0.11 (SE) °C and decreased on average by
temperatures and thermal sensations

Table 1. Differences of time factors in various body temperatures and thermal sensations

<table>
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<tr>
<th>Variable</th>
<th>Time Factor</th>
<th>df</th>
<th>F</th>
<th>Time Factor</th>
<th>df</th>
<th>F</th>
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<td>4.20</td>
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<td>4.20</td>
<td>601§</td>
<td>40/200</td>
<td>2.0†</td>
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<td>2.1*</td>
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<td>125§</td>
<td>40/200</td>
<td>0.4</td>
</tr>
<tr>
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<td>Day</td>
<td>10,50</td>
<td>2.0</td>
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<tr>
<td>Cheek</td>
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Data were analyzed by repeated-measures two-way analysis of variance. Time factor day, differences between days 0 and 10 at each exposure time point; time factor minute, differences between exposure lengths from 0 (or –5) to 120 (or 115) min; df, degrees of freedom. Significant difference: *P < 0.05, †P < 0.01, ‡P < 0.001, and §P < 0.0001.

0.5°C during the 120-min cold exposure (Fig. 1). The decrease was significant between time points 0 and 120 min on day 0 (significance not shown). During the next 10 daily exposures to cold air, the decrease in the rectal temperatures were similar, and hence no habituation process in deep body temperature was observed. The mean skin temperature decreased during the 120 min from 33.1 ± 0.16 to 23.4 ± 0.19°C on day 0 (Fig. 1), with the decrease from the preexposure levels being significant already after 10 min (significance not shown). When the cold-air exposure was repeated, the mean skin temperature at time points 60 and 120 min became similarly warmer, i.e., ~0.8°C on day 5 compared with day 0. Forearm skin temperature at time points 60 and 120 min became similarly warmer, i.e., 1.5–2.0°C on days 1–10; 5–6; and 8; at 65 min on days 3–5, 7, and 9–10; and at 115 min on days 4–10. Thus there was a clearly perceptible habituation in the thermal sensations beginning already after the first cold-air exposure and lasting in most cases to the end of the 11-day experiment.

The subjects were asked about shivering every 5 min during the cold exposure. Subjective shivering started at 15 min on day 0, and the time it started did not change during the experiments (data not shown).

Metabolic rate increased significantly at the 30–120 min time points from the preexposure levels (Fig. 3). During the 11-day experiment, the increases in metabolic rate were similar, and no habituation was seen. Decreases in heart beat were also similar during the experiments.

Systolic blood pressure readings were 122 ± 3 mmHg at 0 min on day 0 and increased significantly after 5 min in response to cold exposure, reaching the maximum of 137 ± 4 mmHg at 60 min (Fig. 3). The increase was significantly reduced only at 60 min on days 4 and 6, indicating that systolic blood pressure also became very transiently habituated. On day 0, the diastolic blood pressure (Fig. 3) increased significantly at the end of the cold exposure, and no habituation was seen during the 11-day experiment.

We had heart rate recordings available only on days 0, 1, 5, and 10 at time points 0, 30, 60, and 120 min. On day 0, the heart rate was 78 ± 6 beats/min before the cold-air exposure and decreased to 64 ± 4 beats/min at 120 min (P < 0.01), and these decreases in heart rates were similar during days 1, 5, and 10 (data not shown).

Serum hormones, proteins, and other constituents. Serum hormones were measured before the cold exposure and after the 120-min cold exposure on study days 0, 5, and 10 (Table 2). No significant changes in the levels of serum total triiodothyronine, thyroxine, TSH, cortisol, and epinephrine in response to the 120-min cold exposure were observed. On the other hand, serum norepinephrine increased significantly from 473 ± 73 to 1,329 ± 154 pg/ml on day 0, but the increase was significantly reduced on days 5 and 10. Serum FFAs increased significantly in response to cold-air exposure on days 0 (P < 0.01), 5, and 10 (P < 0.05 for both days), and the increase on day 10 was significantly smaller than that on day 0. Serum total proteins tended to increase in response to cold-air exposure on days 0 and 5, but on day 10 there was no increase (P < 0.05). Thus
serum norepinephrine, FFA, and total proteins became habituated to repeated cold-air stimuli: norepinephrine and FFA on days 5 and 10 and serum proteins on day 10.

**DISCUSSION**

To understand better the interplay between physiological mechanisms in cold adaptation, we exposed six lightly clad male subjects to cold air for 120 min daily for 11 successive days and recorded the responses in body temperatures, thermal sensations, blood pressure, and metabolic rate every day and those of catecholamines, thyroid hormones, TSH, and serum proteins on days 0, 5, and 10. The responses to the first cold exposure were in line with several previous studies in which healthy subjects had been exposed to cold air (12, 20, 29).

When the cold-air exposures were repeated daily, thermal sensations became habituated first. Hand, foot, and general thermal sensations already became warmer after the first exposure at some time points (35 and 115 min) and that of the face after the second exposure. This early habituation in thermal sensations lasted throughout the 11-day cold exposure period and was almost complete with regard to face thermal sensations. In another experimental setting in which air temperature was decreased by 0.35 or 0.5°C/min for 40–60 min, it was observed that thermal sensations moved from very cold to cold or thermal discomfort was alleviated after the third daily exposure (2). Our exposure time was longer (120 min), and hence it is possible that we already observed significant changes after the first exposure. Increased skin temperatures after long-term or repeated cold exposures evidently reflect greater heat transfer from core to shell, because the cutaneous vasoconstrictor response to cold had become less pronounced because of habituation.

We observed great changes in the forearm skin temperature, which was 1.5–2.0°C warmer at the time points 60 and 120 min on days 5–8 than on day 0. Mean skin temperature sporadically increased on day 5 at time points 30–120 min. In earlier studies, long-term cold-air exposures resulted in increased skin temperatures in toe, arch, and calf, but no changes were seen in chest, hand, forearm, or finger (16, 18). Different experimental settings explain the differences in the results.

Adaptive changes in cold sensations and skin temperatures have been regarded as forms of habituation that belong to the nondeclarative type of memory (14).
After the first cold stimulus, the successive ones evidently lead to synaptic depression in neural connections between hippocampal and cortical areas, which could be reflected in reduced cold sensations and skin temperatures in a manner that we observed in this study. It has been shown that different responses, e.g., cold-induced pain and an increase in blood pressure, became habituated at different rates (8). In the present study, we also observed that cold sensations became habituated first and lasted the longest. The unpleasant nature of the cold sensation and its relation to limbic functions may explain its strong habituation.

We encountered in the present study an unexpected time course of cold habituation. The significantly increased responses to cold air in mean skin temperatures vanished after day 5 and those in the forearm after day 8. It should be noted that the significant changes in thermal sensations lasted to the end of the exposure period. Why the habituation in skin temperatures, but not in cold sensations, vanished after days 5–8 is not known, but it may be related to the low intensity of the cold-air stimulus used. This is a useful piece of information for when experiments utilizing long-term cold-air exposures are planned.

We did not find any significant changes in the responses of the rectal temperature among the exposure days. This is in agreement with a previous study in which subjects were exposed to 30-min cold exposure 80 times and no significant changes in rectal temperature were seen (11). On the other hand, significant decreases in the responses of rectal temperature to cold have been observed, but the exposure times have been longer: 7.5 h (16) or 24 h (18) vs. 2 h in our study. In the present study, the metabolic rate increased by ~40% after the cold-air exposure on the first day (day 0), and the increase was similar throughout the whole experiment. In some previous studies, repeated cold-air exposures have led to decreased metabolic responses after acclimation (3, 11, 16), but the durations of cold acclimation in those studies were longer.

Adrenal medullary and thyroid hormones and brown adipose tissue are crucial in maintaining body temperature in experimental animals, but their role in humans is less well known. A single, whole body cold-air exposure leads to increased plasma norepinephrine levels, but epinephrine levels remain unchanged (11, 20, 29, 34), suggesting an activation of the sympathetic nervous system. We observed in this study that, when the cold exposure was repeated, the significantly increased norepinephrine response on day 0 disappeared on days 5 and 10. In earlier studies, habituation of norepinephrine secretion has been found to occur after
an 8-day stay in a cold room with a temperature of 15.6°C (23) or after 80 cold-air exposures (11). In a study in which cold-water immersions were used, the serum norepinephrine response to the cold-air test did not become habituated but rather increased significantly after the acclimation (36). The heat loss in the latter study was 26 kJ/kg and only 10 kJ/kg in our present study, explaining the difference in the norepinephrine responses. We emphasize that the observed habituation of the norepinephrine secretion could also explain the thermoregulatory changes. The decreases in circulating norepinephrine levels, as well as in the activity of sympathetic innervation to vasoconstrictor arteries, lead to vasodilatation or inhibition of vasoconstriction. This evidently leads to changes in the periphery, such as increased skin temperature, as seen in this study. The serum FFA response was also attenuated, so that the response became less significant. This finding supports the well-known association between norepinephrine and serum fatty acids.

The increase in the number of blood cells and in the concentration of serum proteins after the cold exposure is well documented (4, 17). The increase is due to hemoconcentration caused by cold-induced peripheral vasoconstriction that leads to extravasation of plasma water (4, 17). We observed hemoconcentration on days 0 and 5 from the increases in serum proteins, but not on day 10. It is possible that the serum proteins no longer become concentrated in response to repeated cold-air stimuli, because vasoconstrictions had become reduced. Whether other mechanisms are involved is not known at the moment.

Thyroid hormones are also necessary in the adaptation to cold. We measured serum levels of total triiodothyronine, thyroxine, and TSH at the beginning of our cold exposures and during days 5 and 10. There were no changes in total thyroid hormone and TSH levels in response to cold in our present study, in agreement with a recent study in which 80 cold-air exposures were executed (11). Thus there is no firm evidence that a cold-air stimulus would trigger a neuroendocrine reflex by stimulating the release of TSH in humans, either in acute or subacute experiments. Instead, another mechanism appears to exist by which cold exposures would lead to altered thyroid hormone levels. As discussed above, the concentrations of total thyroid hormones remained stable during repeated cold-air exposures, but free triiodothyronine decreased (11, 31). It was recently shown that an intense cold acclimation, i.e., immersion of lower limbs in icy water for up to 1 h twice a day for 1 mo, decreased serum total triiodothyronine in the cold-air test performed after the acclima-
and 

10

had heart rate recordings available from only at time point 60 min but no significant changes in mental cold stimuli (7, 11). We observed a significant pressure and heart rate responses to repeated exposures. Studies have demonstrated a gradual decrease in blood pressure increased significantly during the first 5 min diastolic blood pressure and, by a reflex mechanism, in cold exposures were experienced as very cold, they did not respond to a single cold exposure (20). In our present study, serum cortisol levels did not change not to the reduction in the activity of the sympathetic nervous system. Thus cold-air exposures in laboratory conditions that may explain their immediate and persistent habituation. The development of habituation in skin temperatures (forearm and mean skin), systolic blood pressure, and norepinephrine responses after four to five daily exposures relates to a common process, probably to the reduction in the activity of the sympathetic nervous system.

Habituations of cold sensation, norepinephrine response, hemoconcentration, and, in lesser amounts, skin temperature and systolic blood pressure provide certain benefits to those humans who have to stay and work in cold environments.

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REFERENCES


