Cold-adapted (CA) rats, as compared with non-adapted (NA) ones, have been reported to give enhanced metabolic response to acute cold exposure (Székely et al., 1994). This may be due to altered function of thermosensors (Székely & Mercer, 1999), to altered central processing of thermoregulatory information, or to altered metabolic responsiveness of tissues. In the present studies the possibility of cold adaptation-induced regulatory changes was checked. Wistar rats were adapted to a room of 3-5°C or 22-25°C (CA or NA group, respectively). Temperatures of the colon ($T_c$), tail skin ($T_s$), and metabolic rate (MR) of CA vs. NA rats were analyzed at 25 vs. 30°C (thermoneutrality) and after exposure to moderate (15 vs. 21°C) or to intense (5 vs. 15°C) cold. Temperatures were measured by thermocouples, MR by diathermometer, while the rats stayed semirestrained in a metabolic chamber. Resting MR at thermoneutrality is higher (8.10 ± 0.32 W/kg), $T_c$ is lower (37.84 ± 0.05°C) in CA rats (as compared with 6.05 ± 0.23 W/kg and 38.33 ± 0.09°C, respectively, in NA rats); the high MR is counterbalanced by earlier onset of cutaneous heat loss. Acute cold exposure causes an immediate MR-rise, without initial $T_c$-fall in CA rats; this MR rise exceeds the actual need („overshoot”) and results in „paradoxical” elevation of $T_c$ (persisting as long as the cold exposure); upon re-warming both MR and $T_c$ fall. Comparable cold exposure in NA rats (e.g. similar MR rise in % in NA rats exposed to 15°C and CA rats exposed to 5°C, or similar MR level in NA and CA rats at 15°C) always causes $T_c$-decline and slow MR-rise, in sharp contrast to CA rats. Stepwise cooling of CA rats causes stepwise rise in $T_c$, suggesting that the overshoot MR- and $T_c$-levels are not due to the rate of cooling, rather to the actual severity of cold which alters the regulated levels of MR and $T_c$. It is concluded that oversensitive peripheral cold sensors, while stimulated, sustain a too high metabolic tone and a paradoxically high $T_c$. The high metabolic activity of tissues (e.g. brown fat) and the enhanced responsiveness of tissues to thermogenic (e.g. noradrenaline) stimuli contribute to the overshoot phenomenon. This phenomenon can still be explained basically by regulatory changes: increased cold sensitivity plus greater central regulatory responses (altered central processing of thermoregulatory information), resulting in greater outgoing signal to the peripheral heat producing/conserving mechanisms.


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