ENHANCED RESPONSIVENESS TO CENTRAL PROSTAGLANDIN E OR NEUROPEPTIDE Y IN COLD-ADAPTED RATS

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In contrast to non-adapted (NA) rats, cold-adapted (CA) rats give "overshoot" rise in metabolic rate (MR) upon acute cold exposure (Székely *et al*., 2001). This might be explained by an enhanced sensitivity of peripheral temperature (cold) sensors (Székely & Mercer, 1999), possibly by high tissue thermogenesis and increased tissue responsiveness to thermogenic stimuli, but might also be explained by altered central processing of thermoregulatory information. Now this last possibility was analyzed and the effects of non-thermal influences on central regulatory functions were studied. Wistar rats were accustomed to handling and body weight measurements. They were adapted to a room of 22-25 or 3-5°C (NA or CA groups) temperature, with lights on between 6.00-18.00 h. Under intraperitoneal ketamine + xylazine (78 + 13 mg/kg) anesthesia guide-cannula was implanted into a lateral cerebral ventricle (i.c.v.). One week later the rats were semi-restrained, placed in a metabolic chamber and 100 ng prostaglandin E_1 (PGE, Sigma) was injected i.c.v. at thermoneutrality (25°C for CA, 30°C for NA rats). Metabolic rate (MR) and colonic temperature (T_c) were measured by diaferometer and thermocouples, respectively. Similar measurements were done after i.c.v. injection of 10 µg neuropeptide Y (NPY, Bachem) at cool (15°C for CA, 20°C for NA) or thermoneutral temperatures. In other cases, cumulative body weight changes, as indicators of food intake (FI) were measured for 3-h following i.c.v. injection of 2 µg NPY at 9.00 at the adaptation temperature (or in some CA rats at 22-25°C), while the rats stayed in their home-cages where chow and water were freely available. After the experiments the rats were given a narcotic overdose. Resting MR was higher, T_c was lower in CA than in NA rats. Both MR and T_c rose following PGE (but not saline) injections; the rises were significantly greater in CA rats. In cool environments NPY induced slightly more pronounced T_c-fall in CA than in NA rats. NPY induced FI in both groups, but the response was significantly greater in the CA group. 0.9% NaCl injections were without effect on FI. The actual environmental temperature at which the food was offered did not influence the FI response of CA rats to NPY. It is concluded that CA rats, similarly to their "overshoot" thermoregulatory response to peripheral cold, are hyper-responsive to direct central stimuli, without any change in peripheral thermal signals. Identical PGE amounts evoke greater thermoregulatory responses in CA animals. This is not due to increased thermogenic capacity of peripheral tissues, since similar phenomenon can be shown for NPY in FI regulation. Apparently, central regulatory responsiveness is generally enhanced in CA rats.

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Supported by OTKA T026511.

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