POSTALIMENTARY HYPERTHERMIA: A ROLE FOR GASTROINTESTINAL BUT NOT FOR CALORIC SIGNALS

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Fasting causes suppression of metabolic rate (MR) and core temperature (T_c). In contrast, food intake induces immediate elevation of MR and T_c (postalimentary hyperthermia, or thermic effect of food, TEF). These are not simply changes due to altered energy reserves, instead they are regulatory changes since, *e.g*., acute cold exposure can increase MR even in the state of severe fasting hypometabolism (Székely *et al.*, 1997). The question regarding the nature of information channels for such regulatory changes has not been resolved. In the present studies MR and T_c of Wistar rats were measured (by diaferometer and thermocouples, respectively) at thermoneutrality following a 48-h food withdrawal or in connection of re-feeding. Spontaneous re-feeding for 3-h with rat-chow or saccharine-sweetened $CaCO_3$ was followed by MR and T_c measurements. In other cases, during MR and T_c measurements, artificial re-feeding was performed: a) through a preimplanted gastric cannula either calorie-rich (FWG) or calorie-free (HD) substance was injected (water in controls), b) through a preimplanted jugular cannula either 4 ml 40% glucose or 2.5 ml 20% fat emulsion (Intralipid) (or 0.9% NaCl in controls) was infused within 2-h. All implantations were performed under intraperitoneal ketamine + xylazine (78 + 13 mg/kg) anesthesia 3-7 days before fasting, and all operated animals were given a narcotic overdose after finishing the measurements. Fasting caused suppression of MR and T_c. Spontaneous re-feeding was followed by reversal of this suppression both in chow- and $CaCO_3$-fed rats; not the composition but the volume of ingested substance seemed to be important. Both FWG and HD injections elicited elevations in MR and T_c, although the dynamics were different for the two substances (the rise commenced earlier in case FWG was given). Neither glucose, nor Intralipid infusion modified low MR and T_c values of fasting rats. It is concluded that not caloric signals, neither oro-facial neural impulses, rather gastrointestinal signals (most likely due to stretch, nutrients, gastrointestinal hormones) may be responsible for the postalimentary rise in MR and T_c, and probably similar (or inverse) gastrointestinal signals may be detectible in the background of the fasting-induced hypometabolism and hypothermia.

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