Significance of osmotic stimulation of the intestines in diet-induced thermogenesis

Toshimasa Osaka and Akiko Kobayashi
National Institute of Health and Nutrition, Shinjuku 162-8636, Japan.

Summary

Diet-induced thermogenesis (DIT) is the increase in whole body energy expenditure following food ingestion. We studied the signals by which the ingested nutrients stimulate the energy expenditure in urethan-anesthetized rats. Infusion of 5-20% glucose, 1.8-3.6% NaCl, 20% methylglucose, 20% fructose, or 5-10% solutions of various amino acids (10 ml/kg) into the duodenum induced dose-dependent thermogenesis. In contrast, infusion of 0.9% NaCl, distilled water, or safflower oil had no effect on the metabolic rate. Infusion of 7.2% urea induced a small and transient increase in the metabolic rate. These results suggested that the thermogenesis was caused mainly by changes in osmolality rather than by a specific action of the different solute molecules. The respiratory exchange ratio increased after the infusion of glucose, fructose, glycine, or serine, did not change after the infusion of NaCl, methylglucose, safflower oil, or distilled water, and decreased after infusion of arginine. Therefore, there was no relationship between substrate utilization and the occurrence of thermogenesis. Intestinal infusion of 3.6% NaCl elevated the plasma osmolality, with a plateau increase of ~20 mosmol/kg. However, intravenous infusion of the same amount of NaCl induced a significantly smaller thermogenic response, although it elevated the plasma osmolality with a time course and magnitude similar to those obtained after the intestinal infusion. Infusion of NaCl into the hepatic portal vein or the peritoneal cavity also produced a significantly small thermogenic response. These results suggested an intestinal or mesenteric location for osmoreceptors. To test for possible stimulation of intestinal osmoreceptors after intake of a normal meal, we measured the osmolality of the intestinal contents. The osmolality of the duodeno-jejunal contents was 600-800 mosmol/kg, whereas the plasma osmolality was 306 ± 1 mosmol/kg, which suggests that the intestinal osmoreceptors are stimulated after meals and are involved in diet-induced thermogenesis. In conclusion, the present study demonstrates that the intestinal osmotic pressure, but not specific properties of nutrients, is critically involved in the initiation of DIT.