Effect of cerebrospinal fluid hyperosmolality on sodium depletion-induced salt appetite in rats

Akira Takamata, Taketoshi Morimoto, and Hiroshi Nose*
Department of Physiology, Kyoto Prefectural University of Medicine, Kamigyo-ku, Kyoto, 602, Japan.
* Present address: Department of Sports Medicine, Shinshu University School of Medicine, Matsumoto, 390, Japan.

Summary

We examined the effect of cerebrospinal fluid (CSF) hyperosmolality on sodium depletion-induced salt appetite in rats. Sodium depletion was produced by two injections of furosemide (5mg/rat; i.p.), 2 h apart. Following the 2nd furosemide injection rats were infused either isotonic artificial CSF (IaCSF; [Na] = 150 mM) or hypertonic artificial CSF (HaCSF; [Na] = 500 mM) at a rate of 36 μl / h. One hour after the onset of infusion, access to water and 0.3 M NaCl solution was provided. Na content reduced by 1.3 - 1.4 meq, which is corresponding to the Na content of more than 15 % of total extracellular fluid Na, and water by 12-13 ml before the access was provided. Furosemide treatment induced salt appetite and thirst, the intake of 0.3 M NaCl increased about 4 times more and water intake about 2.5 times more than sodium repleted control rats during 8 h access period, which were injected with saline instead of furosemide. Intracerebroventricular infusion of HaCSF significantly attenuate 0.3 M NaCl intake in sodium depleted rats, the 0.3 M NaCl intake for 8 h in HaCSF infused rats reduced to only 32 % of IaCSF infused rats, which was not different from control rats. Water intake was not significantly influenced by the infusion of HaCSF. The Na excretion into the urine increased in HaCSF infused rats compared to IaCSF infused rats. The rats infused with IaCSF recovered almost 100 % of total Na loss induced by the furosemide treatment, while the recovery ratio of Na during the 8 h access period in HaCSF infused rats was only 20 % of total Na loss. These results indicate that increased CSF osmolality inhibits salt appetite and also induces natriuresis, which results in a large Na deficit during the 8 h access period.