Brain nitric oxide can induce frequent urination via brain glutamatergic receptors in rats

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Recently, we reported that centrally administered SIN-1, a donor of nitric oxide (NO), induced frequent urination in rats. In the present study, therefore, we investigated central mechanisms how brain NO induced frequent urination focusing on glutamatergic receptors in urethane-anesthetized (0.8 g/kg, ip) male Wistar rats. A catheter was inserted into the bladder to perform cystometrograms (CMG). CMG was started 2 h after the surgery and 1 h after the start, SIN-1 (250 μg/rat) was intracerebroventricularly (icv) administered. Effects of icv pretreated carboxy-PTIO (PTIO, NO scavenger, 750 μg/rat), MK-801 [N-methyl-D-aspartate (NMDA) receptor antagonist, 10 or 30 nmol/rat] or DNQX (AMPA receptor antagonist, 3 nmol/rat) on the SIN-1-induced responses were also investigated. SIN-1 dose-dependently shortened intercontraction intervals (ICI). The SIN-1-induced ICI shortening was significantly attenuated by central pretreatment with PTIO or MK-801, respectively. On the other hand, DNQX showed no significant effect on the SIN-1-induced ICI shortening. These results suggest that brain nitrergic pathway can directly regulate the micturition reflex via brain NMDA receptors in rats.