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Effects of Body Fluid Shift on Sodium/Water Balance Homeostasis by Vasopressin System, Using a Novel Genetically Modified Rat

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Summary

Sodium and water balance in the body is maintained within a certain range by various hormonal and neuronal regulations. In this study, we aimed to elucidate how the central body fluid homeostasis, which is mainly regulated by arginine vasopressin (AVP) that is known as a posterior pituitary hormone is driven by fluid shift into the peritoneal cavity, using AVP-eGFP transgenic rats.

Adult male AVP-eGFP transgenic rats were intraperitoneally administered with 3% hypertonic saline, polyethylene glycol and 0.9% saline as control. AVP-eGFP fluorescent intensities, Fos protein expression were measured quantitatively at 0, 3, 6, 12, and 24 hours after administration. AVP mRNA, AVP hnRNA, eGFP mRNA, and CRF mRNA in the hypothalamus, plasma AVP and corticosterone levels were also measured at 3 and 6 hours in each group.

The results showed that the polyethylene glycol-treated group showed a significant increase in AVP-eGFP in the hypothalamus, not only magnocellular division but also parvocellular division of the paraventricular nucleus (PVN). In particular, the parvocellular PVN neurons were activated for a long time after polyethylene glycol treatment. Furthermore, we also found an increase in AVP hnRNA, eGFP mRNA and CRF mRNA after polyethylene glycol administration. We also confirmed that not only plasma AVP but also corticosterone level was markedly increased after polyethylene glycol administration.

These results suggest that prolonged fluid shift into the abdominal cavity activates AVP-eGFP neurons in the magnocellular division of the PVN caused an increase in plasma AVP concentration, and that AVP synthesis as well CRF is simultaneously enhanced in the parvocellular division of the PVN. Fluid shift into the abdominal cavity without plasma osmotic changes activated the hypothalamus-pituitary-adrenal (HPA) axis by activating the AVP neurons of the parvocellular division of the PVN. In general, this response is a physiological response to chronic stress, but it remains to be clarified why chronic fluid shift into the abdominal cavity activates the HPA axis and what physiological role it plays in fluid regulation.