J. Grad. Sch. Biosp. Sci.,
Hiroshima Univ. (2003), **42** : 28

NEURONAL NETWORKS REGULATING DRINKING BEHAVIOR IN THE EEL

Takao MUKUDA

Division of Morphological Analysis, Department of Functional, Morphological and Regulatory Science, Tottori University, Yonago 683-8503

Drinking behavior is essential for almost all vertebrates to maintain body fluid homeostasis. However, the mechanisms of control of drinking behavior are not clear, even in mammals. Using Japanese eel *Anguilla japonica* as a model system, probably more suitable than mammals, present study was performed to clarify the neuronal networks regulating drinking behavior. At the first place, since brain atlas of the eel is not published yet, a comprehensive brain atlas ranging from the olfactory bulb to the medulla oblongata (MO) was constructed based on both transverse and sagittal sections stained with Klüver-Barréra and Bodian methods. Based on the brain atlas, identification and electrophysiological characterization of the nuclei associated with regulation of drinking behavior was carried out. After injecting Evans blue (EB) intraperitoneally, many neurons in the magnocellular preoptic nucleus (PM) and anterior tuberal nucleus (NAT) in the diencephalons, and the area postrema (AP) in the MO were stained by the dye in the systemic circulation. This result indicates that these neurons distribute outside the blood-brain barrier (BBB). On the other hand, neurons in the spinooccipital motor nucleus (NSO) and glossopharyngeal vagal motor complex (GVC) in the MO were retrogradely labeled following EB injection into the oral, pharyngeal and esophageal muscles, which are associated with the drinking behavior. Almost all EB-positive neurons in these nuclei were also stained by an antibody raised against to choline acetyltransferase (ChAT), a rate limiting enzyme for acetylcholine (ACh) synthesis, suggesting that the NSO and GVC innervate the 'drinking-associated muscles' directly through ACh. The neuronal activity of the isolated GVC, which innervates the upper esophageal sphincter (UES) muscle directly, was inhibited by addition of adrenaline, dopamine or noradrenaline. Moreover, several neurons of the AP and commissural nucleus of Cajal (NCC) projecting to the GVC were stained by an antibody raised against to tyrosine hydroxylase, a rate limiting enzyme for L-dopa synthesis, suggesting that these neurons innervate the GVC and use catecholamines as a neurotransmitter. These results suggest that the PM, NAT and AP may receive dipsogens and antidipsogens produced in the systemic circulation, and that the thirst information integrated in the brain may finally transmit to the GVC through catecholamines to relax the UES.