Normal serum sodium levels are between approximately 135 and 145 mEq/liter (135 - 145 mmol/L).

Hyponatremia is low sodium concentration in the blood.

Changes in body water/sodium balance are tightly controlled by the central nervous system (CNS) to avoid abnormal cardiovascular function and the development of pathological states. Every time there is a disturbance in extracellular sodium concentration or body sodium content, there is also a change in extracellular fluid volume and, depending on its magnitude, this can be associated with an adjustment in arterial blood pressure (BP). The process of sensory integration takes place in different nuclei, with diverse phenotypes and at different levels of the CNS. To control those several changes, the CNS receives continuous input about the status of extracellular fluid osmolarity, sodium concentration, sense of taste, fluid volume, and BP.

Sodium appetite constitutes an important homeostatic behavior involved in seeking out and acquiring sodium from the environment. Under normal circumstances, the average daily intake of sodium in animals exceeds what is actually needed; however, when they are challenged by environmental (e.g., increased ambient temperature), physiological (e.g., exercise, pregnancy and lactation), or pathophysiological (e.g., emesis, diarrhea, adrenal, or kidney insufficiency) conditions, endocrine and autonomic mechanisms primarily target the kidney, to influence the rate of water and sodium loss, and the vasculature, to maintain arterial BP. Afterward, a behavioral mechanism such as sodium appetite is the means by which sodium loss to the environment is ultimately restored (Geerling and Loewy 2008). It is important to note that in humans, salt appetite is permanently enhanced after perinatal sodium loss (Crystal and Berstein 1995, 1998; Leshem 2009), but putative sodium loss in adults due to, for example, hemorrhage, dehydration, or breastfeeding, does not increase salt appetite significantly; thus, the existence of sodium appetite as a result of sodium loss in adult humans remains controversial (Bertino et al. 1982; Beauchamp et al. 1983, 1987; Leshem 2009). This review will focus on evidence from our laboratory for neurophysiological mechanisms that regulate sodium balance. Specifically, it tries to answer how the brain elicits sodium appetite in response to hyponatremia/hypovolemia associated with sodium depletion, which areas are activated after sodium depletion, how the brain controls the inhibition of this behavior once the deficit is compensated (satiety phase), and what role brain neurochemical groups have for endocrine responses. We close the chapter by analyzing the effects of gonadal hormones and sex chromosome complement (SCC) on sodium appetite and cardiovascular function, respectively [1)].

Hypovolemia is characterized by sodium (salt) depletion and thus differs from dehydration, which is defined as excessive loss of body water.

1)
Vivas L, Godino A, Dalmasso C, Caeiro XE, Macchione AF, Cambiasso MJ. Neurochemical Circuits Subserving Fluid Balance and Baroreflex: A Role for Serotonin, Oxytocin, and Gonadal Steroids. In: De Luca LA Jr, Menani JV, Johnson AK, editors. Neurobiology of Body Fluid Homeostasis: Transduction and Integration. Boca Raton (FL): CRC Press/Taylor & Francis; 2014. Chapter 9. PubMed PMID: 24829993.