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Research Report

Cellular plasticity in the supraoptic and paraventricular nuclei after prolonged dehydration in the desert rodent *Meriones shawi*: Vasopressin and GFAP immunohistochemical study

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ABSTRACT

Supraoptic (SON) and paraventricular (PVN) nuclei are part of the hypothalamic–neurohypophysial system, they constitute the main source for vasopressin and they represent also obvious examples of activity-dependent neuroglial plasticity. Certain physiological conditions such as dehydration are accompanied by a structural remodeling of the neurons, their synaptic inputs and their surrounding glia. In the present work, an adult *Meriones shawi* (a rodent adapted to desert life) is used as an animal model. Using GFAP and vasopressin expressions as indicators successively of astrocytes and neuronal activations, the effect of a prolonged episode of water deprivation on the SON and PVN, hypothalamus nuclei were examined. We studied the immunoreactivity of GFAP and vasopressin in various hydration states (total deprivation of drinking water for 1 and 2 months compared to hydrated animals). Prolonged dehydration produces an important decrease of GFAP immunoreactivity in both SON and PVN after 1 and 2 months of water restriction. This decrease is accompanied by increased vasopressin immunoreactivity following the same periods of water deprivation. These findings may explain a real communication between vasopressin neurons and their surrounding astrocytes, thus the retraction of astrocytes and their processes is accompanied by an enhancement of vasopressin neuron density and their projecting fibers in response to this osmotic stress situation. Furthermore, these data could open further investigations concerning the possible involvement of the communication between astrocytes and vasopressin neurons in both PVN and SON in the regulation of *Meriones* hydrous balance and resistance to dehydration.

1. Introduction

Mammals living in the desert can survive for long periods without free water by obtaining preformed water from food and metabolic water (Degen, 1997). For this, they have developed homeostatic mechanisms and numerous adaptations. Among animals needing the development of these mechanisms, the desert rodent, *Meriones shawi* is characterized

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Abbreviations: SON, supraoptic nucleus; PVN, paraventricular nucleus; AVP, vasopressin; GFAP, glial fibrillary acidic protein; MNCs, magnocellular neurosecretory cells; OC, optic chiasm; 3V, third ventricle

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by its resistance to long periods of thirst and has a particular ability to support prolonged dehydration for periods going until several months (Elgot et al., 2009; Laalaoui et al., 2001). Dehydration is known to be a major factor affecting the activation of several endocrine systems, including the hypothalamic–neurohypophysial system (Ciosek et al., 1993; Hatton, 1997).

The osmotic control of vasopressin (AVP) secretion plays a key role in systemic osmoregulation since this peptide stimulates water reabsorption by the kidney. AVP (also known as antidiuretic hormone) is synthesized in the soma of hypothalamic magnocellular neurosecretory cells (MNCs) located in SON and PVN. After water deprivation, the axons of MNCs project to the neurohypophysis, where Ca2+-dependent exocytosis in their nerve terminals causes the release of AVP in blood circulation (Bicknell, 1988). Moreover, expression of the immediate-early gene c-*fos* has been extensively used as a marker of neural activation, particularly in neuroendocrine systems. Previous study have shown that water restriction for 6 days results in increased Fos expression in AVP magnocellular and parvocellular neurons (Wotus et al., 2007). In rat, water deprivation or salt loading increases AVP mRNA levels in the SON and the PVN and the release of AVP into the blood, causing a decrease of diuresis (Meister et al., 1990; Carter and Murphy, 1991). Recent data indicate that drinking-induced decreases in glucocorticoids in dehydrated rats involve multiple factors including reduction in magnocellular release of AVP and reduction in parvocellular neuronal activity (Arnhold et al., 2007). Furthermore, drinking after a period of water deprivation causes a rapid decrease in plasma AVP in rats (Stricker and Hoffman, 2005).

Otherwise, the SON and PVN nuclei constitute obvious examples of activity-dependent neuroglial plasticity, in which certain physiological conditions are accompanied by structural changes of the neurons, their projections and their surrounding astroglial cells (Theodosis and Poulain, 1993; Piet et al., 2004) showing that glia is intimately involved in magnocellular cells functions. In the hypothalamus, astrocytes, besides supporting metabolic and scaffolding functions, play a prominent role in the modulation of neuronal communication (Piet et al., 2004). The SON responds to a variety of stimuli such as lactation, parturition or dehydration (Hatton, 1997). Numerous ultrastructural studies have shown that there is a significant reduction of astrocytic processes that normally separate MNCs, their dendrites and synapses (Hatton, 1990, 1997; Salm et al., 1998; Theodosis and Poulain, 1993). This finding is accompanied by an increase of MNC excitability and release of AVP (Hatton, 1990, 1997; Theodosis and Poulain, 1993). Previous immunohistochemical studies have determined that GFAP immunoreactivity is significantly decreased during the period of dehydration (Landry et al., 1994). These decreases are further accompanied by a decreased SON astrocyte surface density (Hawrylak et al., 1999).

The aim of the present study is to analyze by immunohistochemical procedure, using the AVP and GFAP antibodies, the effect of prolonged water deprivation on SON and PVN of a desert rodent *M. Shawi* characterized by its resistance to severe thirst, and to understand the contribution of the interaction between the neuronal system (vasopressin neurons) and glial system (astrocytes) in these nuclei on the resistance to prolonged dehydration developed by this rodent.

2. Results

2.1. *Effect of dehydration on GFAP expression in SON*

The GFAP immunoreactivity in the SON was significantly affected by dehydration states. A visual and a statistical comparison between the controls versus dehydrated groups, show that GFAP immunoreactivity was decreased following 1 and 2 months of water deprivation.

In the control *Meriones*, the GFAP immunoreactivity of the SON appeared very dense, homogeneous, and spread throughout the nucleus (Fig. 1A). The labeling was observed covering the optic chiasm (OC) and the astrocytic processes formed a remarkable network around the neurons of the SON (Fig. 1Aa).

After 1 month of water deprivation the SON showed a marked decrease of immunolabeling in all SON areas except the ventral side close to the extremity of OC (Figs. 1B and Bb). In contrast, we note no change in GFAP immunoreactivity in the OC.

Reduction in GFAP immunoreactivity in SON becomes more obvious after 2 months of dehydration and a few reactive astrocytes could be seen in SON (Figs. 1C and Cc); however, the GFAP immunoreactivity in OC remains unaltered following 2 months of water deprivation.

Compared to controls, quantification of GFAP immunoreactive labeling in SON showed a statistically significant reduction in both water-deprived groups and also between treated groups. ($^{*}p=0.029$ versus C, $^{\#}p=0.029$ versus D1). There were no significant changes in GFAP immunoreactive labeling in OC between controls and water-deprived groups, and also between treated groups ($p>0.05$) (Fig. 1G).

2.2. *Effect of dehydration on AVP expression in SON*

The appearance of AVP immunoreactivity in the SON was also significantly affected by dehydration states. A comparison between the controls versus dehydrated groups, show that AVP immunoreactivity was increased in 1 and 2 month water-deprived groups. The anti-vasopressin immunohistochemistry of the SON in controls (Fig. 1D) showed a slight immunolabeling. Moreover, the projecting fibers coming from the SON and reaching the PVN are discreet (Fig. 1Dd). In the water-deprived group for 1 month (Fig. 1E), a significant increase of AVP immunoreactivity was observed; this augmentation concerns both the number of immunoreactive neurons and the density of fibers innervating the PVN (Figs. 1E and Ee). Following 2 months of water deprivation (Fig. 1F), a marked increase in both AVP neurons and fibers was seen; the fibers formed a bidirectional network connecting both SON and PVN. We note also that cell sizes of AVP magnocellular neurons of *Meriones* were significantly increased in response to a dehydration state (Fig. 1Ff).

Compared to controls, quantification of AVP immunoreactive labeling in SON showed a statistically significant