Neuroadaptive events in the offspring induced by maternal voluntary ingestion of hypertonic sodium solution: brain neurochemical system programming

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Osmoregulatory mechanisms can be vulnerable to electrolyte and/or endocrine environmental changes during the perinatal period, differentially programming the developing offspring and affecting them even in adulthood. The aim of our study was to evaluate whether the perinatal availability of hypertonic sodium solution [PM-Na] from the pregnancy until one month old of the offspring may induce a differential programming of osmoregulatory mechanisms in adult offspring. We have studied the perinatal manipulation [PM] effects on adult water and sodium intake, brain cell activity and gene expression changes induced by different osmotic challenges: acute sodium depletion (ASD) and systemic sodium overload (SSO).

After ASD we observed an altered pattern of fluid intake in PM-Na adults. At brain level, ASD induced an increased number of activated cells in subfornical organ (SFO) and vasopressinergic neurons of supraoptic nucleus (SON) and a decreased number of them in the nucleus of the solitary tract (NTS) in PM-Na rats.

After SSO, our results showed an increased water intake in PM-Na animals and a decreased neuronal activity in the SFO and NTS compared to control groups. Besides, real-time PCR studies showed a diminished mRNA expression of vasopressin in SON and AT1a receptor in SFO, respect to animals without PM.

These data suggest that perinatal sodium availability induces alterations on fluid intake and brain activity after osmotic challenges in the adulthood indicating a neuroadaptive response in the reestablishment profile of fluid balance. Thus, these data provide evidence of a long term effect on fluid drinking and on brain activity/gene expression along nuclei and systems (vasopressinergic/angiotensinergic) previously involved in hydroelectrolyte balance regulation, suggesting that sodium availability during perinatal period may modulate the osmosensitive mechanism and/or brain neurochemical circuits.