Sex Differences in body fluid and electrolyte homeostasis: sex chromosome complement influences on angiotensin responses

Vivas, Laura
Caeiro, Ximena
Dadam, Florencia

Instituto Ferreyra (INIMEC-CONICET) – Universidad Nacional de Córdoba, Argentina.

Clinical and basic findings indicate that angiotensin II (Ang II) differentially modulates hydroelectrolyte and cardiovascular responses in male and female. But are only the activational and organizational hormonal effects to blame for such differences? Males and females not only differ in their sex (males are born with testes and females with ovaries) but also carry different sex chromosome complements (SCC) and are thus influenced throughout life by different genomes. In this talk, we discuss our recent studies in order to evaluate whether SCC is in part responsible for gender differences previously observed in angiotensin II bradycardic-baroreflex response and sodium depletion-induced sodium appetite and neural activity. To test the hypothesis that XX or XY contributes to the dimorphic Ang II bradycardic-baroreflex response, we used the four core genotypes (FCG) mouse model, in which the effects of gonadal sex (testes or ovaries) and SCC (XX or XY) are dissociated. The results indicate that Ang II bradycardic-baroreflex sexual dimorphic response may be ascribed to differences in sex chromosomes, indicating an XX-SCC facilitatory bradycardic-baroreflex control of heart rate. Furthermore, we evaluated whether genetic differences within the SCC may differentially modulate the known sexually dimorphic sodium appetite as well as basal or induced brain activity due to physiological stimulation of the renin-angiotensin system by furosemide and low-sodium treatment. Our studies demonstrate an organizational hormonal effect on sexually dimorphic induced sodium intake in mice, while at the brain level (subfornical organ and area postrema) we showed a SCC effect in sodium-depleted mice, suggesting a sex chromosome gene participation in the modulation of neural pathways underlying regulatory response to renin-angiotensin stimulation.

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