Unraveling Novel Signaling Mechanisms Contributing to Neurohumoral Activation in Cardiovascular Diseases

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The long-term goal in our laboratory is to elucidate central neuronal mechanisms contributing to elevated neurohumoral activation in cardiovascular diseases, including hypertension, heart failure and the metabolic syndrome. We have established a multidisciplinary approach that enables us to study precise intrinsic (ion channels, neuronal structure, intracellular signaling mechanisms) and extrinsic (synaptic organization and function, structural/functional plasticity) mechanisms contributing to altered neuronal function in a variety of animal models of cardiovascular disease. This not only includes neurons, but also neuronal bidirectional communication with other key cell types in the brain, including astrocytes and microglial cells. We recently unraveled a novel signaling mechanism within the hypothalamic paraventricular nucleus that contributes to the generation of multimodal homeostatic responses (Son et al., Neuron, 2013). This involves activity-dependent dendritic release of vasopressin, which acts as an interpopulation signaling modality to recruit presympathetic neurons in response to challenges to homeostasis. I will present in my talk recent advances we have obtained regarding this novel hypothalamic communication modality, as well as evidence supporting a contribution of this mechanism to neurohumoral activation in hypertension and heart failure.

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