Early life stress-programmed risk for obesity-induced hypertension

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Individuals exposed to adverse childhood experiences and early life stress (ELS) display higher risk for chronic disease. As such, ELS is an emerging independent risk factor for obesity and hypertension, components of the cardiometabolic syndrome affecting more than 50% of the adult US population. While a link between ELS and chronic disease is established, causative mechanisms remain unknown. We found that Maternal Separation (MatSep), a model of ELS, augments the circulating levels of the stress-induced glucocorticoids (GCs) when male and female C57BL/6 mice are fed a high fat (HF) diet. HF-fed male and female MatSep exhibit exaggerated obesity associated with elevated biomarkers of cardiometabolic disease and hypertension compared to control mice. We obtained similar findings in a rat model, with a normalization of the body weight and glucose intolerance after a postnatal treatment with a corticosterone synthase inhibitor. It is well-accepted that GCs are a robust stimulus of adipocyte differentiation and metabolism. Exposure to MatSep increased the production of adipocyte-derived factors intimately associated with the increased risk to develop obesity-induced hypertension, such as angiotensinogen and leptin. First, angiotensinogen is the only known precursor of the vasoactive peptide angiotensin II. Second, leptin is known to enhance the sympato-mediated responses during the progression of obesity induced-hypertension and has been directly associated with metabolic disease in patients with a positive history of ELS. We found that obese MatSep mice show reduced spontaneous baroreflex sensitivity and increased magnitude of blood pressure reduction in response to a ganglion blockade, suggesting that exacerbated blood pressure occurs most likely through a sympato-mediated, cardiovascular-driven response. Taken together, we hypothesize that ELS enhances obesity-induced hypertension in rodents though GC-dependent mechanisms.

Biography
Analia S Loria earned her PhD degree at the University of Murcia, Spain. She developed models to study the origins of adult disease in rodents for the last decade using a unique model called maternal separation (MatSep) to mimic the effects of postnatal chronic behavioral stress exposure or early life stress (ELS). As an Assistant Professor, she has focused on studying the impact of the psychological stress superimposed to energy-dense (high fat) food consumption on the blood pressure regulation.

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