Low birth weight and hypertension: Do sex and age matter?

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Date: 13/03/20 – 12:30h
Place: Salón de Actos CIPF

Abstract: Essential hypertension is a complex condition of unknown pathogeneses. Recent advances in the field of developmental origins of increased blood pressure add another layer of complexity. Complications during pregnancy that impair fetal growth and contribute to the developmental origins of increased blood pressure in the offspring are varied and include preeclampsia, diabetes, maternal obesity, parental smoking, maternal stress, alcohol consumption, age and poor perinatal nutrition. Our laboratory uses a model of placental insufficiency in the rat that results in intrauterine growth restriction and an increase in blood pressure in male, but not female growth-restricted offspring in young adulthood. The etiology of increased blood pressure in male growth-restricted offspring is multifactorial and involves a role for the renal nerves, oxidative stress, endothelin and enhanced sensitivity to angiotensin II. However, protective compensatory mechanisms are observed in female growth-restricted offspring that remain normotensive. These protective mechanisms include an increase in renal antioxidant activity and a shift towards the intrarenal ACE2/Ang-(1-7) arm of the renin angiotensin system. Yet, female growth-restricted offspring do not remain protected against increased blood pressure in later life. Blood pressure is elevated in female growth-restricted by 12 months of age in association with cessation of estrous cyclicity that occurs 6 months prior to control counterparts indicative of early reproductive senescence. A shift in the hormonal milieu in addition the renal nerves and the renin angiotensin system are contributory mediators of increased cardiovascular risk that develops with age in female growth-restricted offspring. Thus, placental insufficiency programs sex- and age-specific cardiovascular risk in growth-restricted offspring.