P10.06: ABNORMAL VASCULAR PROGRAMMING OF ACID ARACHIDONIC METABOLISM COULD EXPLAIN HYPERTENSION IN RATS EXPOSED IN UTERO TO MATERNAL DIABETES


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Objective: to test whether 1,25(OH)2D3 (active vitamin D) modifies contractility of proximal resistance vessels, dose-dependently.

Methods: Male Wistar rat mesenteric arteries were investigated by wire myography. A group of rats was treated with a prostacyclin analogue: iloprost (iv, 4 ng/kg/ml). Another group received 1,25(OH)2D3 (10 nM) for 30 min incubation. We found an increase of CTP4P2 (however we failed to confirm its up-regulation at the protein level), and a decrease by 50% of the prostacyclin (IP) receptor at messenger and protein levels in aorta of rats exposed to maternal diabetes (DMO) compared to rats from control mothers (CMO). We demonstrated the functional impairment of this down-regulation of the IP receptor in a pharmacological study using a prostacyclin analogue: iloprost (iv, 4 ng/kg/ml). Indeed, we showed that, even before the onset of hypertension, SBP reduction in response to iloprost was attenuated in DMO rats (-10.7% vs -21.3% in CMO, p < 0.05). In parallel, we studied vascular reactivity and myogenic response of carotid and mesenteric arteries of 18-months-old CMO and DMO. At this later stage, we found similar results, i.e. vasodilation in response to Beraprost was reduced in DMO, and myogenic reactivity was increased.

In this study, we clearly demonstrated a fetal programming of the vessels, which could explain the development of hypertension and a re-setting of physiological functions in adult rats exposed to maternal diabetes.