

THE INFLUENCE OF MATERNAL DIABETES ON THE EXPRESSION OF INFLAMMATORY MARKERS IN THE OFFSPRING

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Background and aims: Previous studies from our group have shown that maternal diabetes is an important cause of glomerular hypertrophy in the offspring (DO) and that hypertension linked to an impairment in the vascular endothelium-dependent relaxation is early detected. The present study was designed to evaluate the expression of cytokines/chemokines in plasma and renal tissue from DO and the possible effect of L-arginine administration on those parameters.

Methods: Diabetes mellitus was induced in Wistar female rats with a single dose of streptozotocyn, 50mg/kg, IP. Diabetic dams were caged overnight with a male. After birth, each litter was left with the mother for 28 days. Some of the offspring received a 2% L-Arg solution in drinking water (groups DA and CA, controls+ L-arg). Tumor necrosis factor-alpha (TNF- α), interleukin (IL) -6 and IL-1b were measured by real time PCR in the kidney from 2 and 6 month-old rats.

Results: Serum expression of TNF- α , interleukin (IL)-1b, IL-2, IL-17, interferon (IFN)- γ , macrophage inflammatory protein (MIP)-1 and leptin enhanced in DO group and L-arg prevented this increase. TNF- α , interleukin (IL) -6 and IL-1b (measured by real time PCR) in the kidney from DO, 6 month-old, were also increased but returned to control values in DA.

Conclusions: Renal and serum inflammatory pathways seem to be early activated in DO, contributing or being the triggering factor for the development of hypertension and renal injury in this model. Our results suggest that these inflammatory pathways can be attenuated by L-arginine.

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