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Nutrition and Fetal Aortogenesis

The link between a low birth weight and higher rates of cardiovascular disease in adult life has been confirmed in many epidemiological studies. According to the fetal origin hypothesis, or Barker hypothesis, cardiovascular diseases may be programmed during certain periods of intrauterine life. A disproportionate fetal growth can occur because different tissues have critical periods of growth at different times. In the human aorta the scleroprotein content varies, so an important difference was observed in the aortogenesis. The rates of elastin synthesis increase to a maximum in the perinatal period (late gestation and first weeks). Collagen has a steady increase during intrauterine life reaching a plateau at term.

We studied the association between intrauterine growth retardation (IUGR) and histomorphological changes in the aorta. The project was designed to test the fetal origin hypothesis with regard to the composition of the aortic wall using an established animal model in which IUGR was induced by a low protein (LP) diet (9% casein). The aortae of the rat’s offspring were examined at the ages of 4, 8 and 12 weeks and compared with the aortae of the offspring on an isocaloric normal diet (18% casein). Morphometrically, the offspring from LP-diet fed rats presented variable results: In the 4 and 8 weeks old male and female animals less elastin and more collagen in the LP-diet animals with a statistically significance in the 8 weeks old male subgroups was found. These findings may account for a reduction in the “material” stiffness of the LP-aortae with further changes in vessel dimensions and elasticity.

With the exception of one subgroup (8 weeks old male, isocaloric normal diet), the scleroproteins showed similar ratios in the examined groups, i.e. more collagen than elastin was found. Most results of the scleroprotein analysis can be attributed to the hypothesis of reduced elastin synthesis. A structural replacement of elastin with collagen leads to an increased aortic stiffness which further leads to higher pulse pressure and systolic hypertension in later life. Once established, the elastin deficiency cannot be corrected due to the extremely low turnover of this protein. Our studies showed that the fetal aortogenesis seems to be affected by nutritional factors in a more complex manner.

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References

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