Vessels and Endothelium

12.21 Fructose, Endothelial Function and Vascular Reactivity in Isolated Rat Aorta

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Introduction. High fructose diet (HFD) in normal rats provides a good model of metabolic syndrome, because it induces hyperinsulinaemia, hypertriglyceridaemia and hypertension. The purpose of this study was to assess whether the increase in blood pressure is early associated with increased vascular reactivity and endothelial dysfunction in this model.

Methods. Male Wistar rats (n=16, weight 250 g) were divided into two equal groups. One of the groups was fed standard diet and served as the control group, whereas the other group was fed a HFD for 4 weeks. Systolic blood pressure was estimated by tail-cuff. At the end of the study thoracic rat aorta rings were placed in organ baths filled with warmed and oxygenated Krebs-Henseleit solution. Endothelial function was assessed by pre-contraction with phenylephrine (10⁻⁷ M) after which concentration-relaxation curves to acetylcholine (10⁻⁹–10⁻⁵ M) were obtained. In order to obtain Urotensin-II (U-II) vascular responsiveness, all rings were contracted with 10⁻⁷ M U-II. The same experiment was repeated in the rings with inhibited NOS and COX enzymes by application of L-NNA (0.36 mM) and indomethacine (9.8 microM), respectively. Responses were measured using an isometric mechano-electrical transducer and contraction was expressed as a percentage of 60 mM KCl pre-contracted aortic rings values. Analysis was performed with one-way ANOVA, followed by Bonferroni post-test.

Results. HFD rats showed higher (p<0.01) systolic blood pressure levels. There was no significant difference in concentration relaxation curves to acetylcholine. Contractile response to U-II was higher (p<0.05) in the control (27.7 ± 5.3 %) compared to fructose-fed group (14.4 ± 2.1 %). Similarly, response was higher (p<0.01) in the control (51.6 ± 9.1%) compared to fructose-fed group (25.5 ± 3.6%) in experiments with inhibited NOS and COX enzymes.

Conclusions. The results of this study demonstrate that in the early stage of this animal model of metabolic syndrome vascular reactivity to U-II and endothelial function evaluated 'ex vivo' are not affected, though blood pressure is significantly increased. This observation would exclude that these mechanisms might be involved in the pathogenesis of the increase in blood pressure observed in this model. Moreover, these results indirectly support the hypothesis suggesting an higher adrenergic tone induced by hyperinsulinaemia as a possible mechanism inducing the increase in blood pressure, although alternative hypotheses cannot be excluded.