The contribution of $\alpha_1B$-adrenoceptor subtype in the renal vasculature of fructose-fed Sprague–Dawley rats

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Abstract

Purpose Fructose feeding induces a moderate increase in blood pressure, insulin resistance, and hyperinsulinemia. This study investigated the role of $\alpha_1B$-adrenoceptor subtype in the control of renal hemodynamic responses to exogenously administered angiotensin II (Ang II) and a set of adrenergic agonists in a model of high fructose-fed rats.

Methods Sprague–Dawley rats were fed for 8 weeks with 20% fructose in drinking water (FFR). The renal cortical vasoconstriction to noradrenaline (NA), phenylephrine (PE), methoxamine (ME) and Ang II in the presence and absence of chloroethyldiamidine (CEC) ($\alpha_1B$-adrenoceptor antagonist) was determined. Data, mean ± SEM or SD were subjected to ANOVA with significance at $p < .05$.

Results FFR showed significant increase in the systolic blood pressure, plasma glucose, and insulin levels when compared to control. FFR expressed reduced renal cortical vascular sensitivity to NA, PE, ME, and Ang II. Furthermore, renal cortical vasoconstriction response to NA, PE, ME, and Ang II was blunted in the presence of CEC in control. While in FFR, renal cortical vasoconstriction to NA, PE, and ME was enhanced by CEC. Renal cortical vasoconstriction to Ang II in FFR was reduced in the presence of CEC.

Conclusions In the presence of a hyperinsulinemic state resulting from chronic and high fructose feeding, an attenuated AT1 and $\alpha_1B$-adrenoceptors response to Ang II and adrenergic stimuli respectively, is expected. In addition, $\alpha_1B$-adrenoceptor is the functional subtype that mediates renal cortical vasoconstriction in control rats, while high fructose feeding did influence the functionality of $\alpha_1B$-adrenoceptor in mediating the renal cortical hemodynamic changes.

Keywords Fructose · Sprague–Dawley rats · Noradrenaline · Hemodynamics · Chloroethyldiamidine

Introduction

Metabolic syndrome is a pathophysiological entity characterized by insulin resistance, hyperinsulinemia, dyslipidemia, hypertension, and obesity [1]. The risk for developing diabetes type 2, cardiovascular disease, and renal disease is increased with increasing manifestations of the various components of the syndrome within any individual. The precise mechanisms behind the development of hypertension in this model have not yet been clearly defined though it has been proposed to be secondary to hyperinsulinemia [2]. In addition, it has been demonstrated that an increase in sympathetic activity could account for the hypertension induced by fructose feeding [3]. Renal $\alpha_1$-adrenoceptors play important role in the regulation of renal hemodynamic and tubular functions [4]. Pharmacological