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Reactive oxygen species and cyclooxygenase 2-derived thromboxane A2 reduce angiotensin II type 2 receptor vasorelaxation in diabetic rat resistance arteries

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Résumé en anglais	Angiotensin II has a key role in the control of resistance artery tone and local blood flow. Angiotensin II possesses 2 main receptors. Although angiotensin II type 1 receptor is well known and is involved in the vasoconstrictor and growth properties of angiotensin II, the role of the angiotensin II type 2 receptor (AT2R) remains much less understood. Although AT2R stimulation induces vasodilatation in normotensive rats, it induces vasoconstriction in pathological conditions involving oxidative stress and cyclooxygenase 2 expression. Thus, we studied the influence of cyclooxygenase 2 on AT2R-dependent tone in diabetes mellitus. Mesenteric resistance arteries were isolated from Zucker diabetic fatty (ZDF) and lean Zucker rats and studied using in vitro using wire myography. In ZDF rats, AT2R-induced dilation was lower than in lean rats (11% versus 21% dilation). Dilation in ZDF rats returned to the control (lean rats) level after acute superoxide reduction (Tempol and apocynin), cyclooxygenase 2 inhibition (NS398), or thromboxane A(2) synthesis inhibition (furegrelate). Cyclooxygenase 2 expression and superoxide production were significantly increased in ZDF rat arteries compared with arteries of lean rats. After chronic treatment with Tempol, AT2R-dependent dilation was equivalent in ZDF and lean rats. Chronic treatment of ZDF rats with NS398 also restored AT2R-dependent dilation to the control (lean rats) level. Plasma thromboxane B(2) (thromboxane A(2) metabolite), initially high in ZDF rats, was decreased by chronic Tempol and by chronic NS398 to the level found in lean Zucker rats. Thus, in type 2 diabetic rats, superoxide and thromboxane A(2) reduced AT2R-induced dilation. These findings are important to take into consideration when choosing vasoactive drugs for diabetic patients.
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