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JOURNAL ARTICLE

Involvement of endothelial nitric oxide synthase in the impaired endothelium-dependent relaxation of cavernous smooth muscle in hypercholesterolemic rabbit

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The present study was designed to evaluate whether functional impairment and/or protein expression of constitutive nitric oxide synthase (cNOS; endothelial NOS [eNOS] and neuronal NOS[nNOS]) was involved in impairment of endothelium-dependent relaxation of cavernous smooth muscle in hypercholesterolemic rabbits. New Zealand White rabbits were randomly divided into control and experimental groups. The control group (n=20) received a regular diet, while the two experimental groups (n=20 for each) were fed a 2% cholesterol diet for 4 and 8 weeks, respectively. We conducted isometric tension studies with endothelium-dependent and endothelium-independent vasodilators with or without preincubation with L-arginine and nonadrenergic, noncholinergic (NANC)-selective electrical field stimulation on isolated strips of corpus cavernosum. Expression of cNOS (eNOS and nNOS) protein was assessed by Western blot analysis. cNOS activities in both cytosolic and particulate fractions were measured by determining the conversion of L-[U-14C] arginine to L-[U-14C] citrulline. Blood levels of cholesterol were significantly higher ($P < 0.01$) in the experimental groups than in the control group. The relaxation responses to endothelium-dependent agents (acetylcholine and adenosine 5'-diphosphate [ADP]) were significantly reduced ($P < 0.05$) in both experimental groups, regardless of their incubation with L-arginine, compared with the control group. However, no differences were found among the three groups in the relaxation response to endothelium-independent agents (papaverine and nitroprusside) and to NANC-selective electrical field stimulation. There was no difference in immunoreactive nNOS from cytosolic and particulate fractions between the cavernous tissues of the control and experimental groups. nNOS protein levels in the particulate fractions were markedly lower than in the cytosolic fractions. The particulate cNOS activity was significantly decreased ($P < 0.05$) in the experimental groups compared with the control group, while the cytosolic cNOS activity in the experimental groups was not different from that found in the control group. Therefore, it is concluded that functional impairment of eNOS, rather than of nNOS, may lead to impairment of cavernous smooth muscle relaxation in response to endothelium-mediated stimuli in hypercholesterolemic rabbits.

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