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Melatonin restores endothelium-dependent relaxation in aortic rings of pancreatectomized rats.

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In rats turned hyperglycemic by a subtotal pancreatectomy, a decreased relaxation response of aortic rings to acetylcholine (ACh) was found; this effect was amplified by preincubation in a high glucose medium (44 mmol/L). The relaxation response to ACh did not occur in endothelium-denuded rings or after the aortic rings were exposed to l-nitro-arginine methyl ester [L-NAME, a nitric oxide (NO) synthase inhibitor]. Incubation with the NO donor sodium nitroprusside (SNP) restored the impaired relaxation response seen in endothelium-denuded or L-NAME-treated aortic rings. Pancreatectomy decreased the vasorelaxation of aortic rings caused by SNP. Only in pancreatectomized rats, incubation in a high glucose medium impaired the relaxation effect of SNP. To assess whether melatonin preincubation reversed the impaired relaxation response to ACh (intact endothelium aortic rings) or to SNP (endothelium-denuded or L-NAME-treated rings) in hyperglycemic rats, cumulative dose-response curves were performed in the presence of 10(-5) mol/L melatonin. Melatonin preincubation did not modify ACh-induced relaxation of aortic rings in a normal glucose concentration but was highly effective in preventing the impairment of relaxation caused by a high glucose solution. Melatonin was also effective in restoring the impaired SNP-induced vasorelaxation seen in endothelium-denuded or L-NAME-treated aortic rings from hyperglycemic rats. **The results further support the improvement by melatonin of the endothelial-mediated relaxation in blood vessels of diabetic rats.**

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