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Effects of ethanol treatment on the neurogenic- and endothelium-dependent relaxation of corpus cavernosum smooth muscle in the mouse

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Abstract:
The relaxation of cavernous smooth muscle is critical for inducing and maintaining a penile erection. The neurogenic- and endothelium-dependent relaxation of corpus cavernosum smooth muscle and the degenerative effect of subacute ethanol treatment on the endothelial cells of corpus cavernosum was investigated in mice. In the cavernous strips contracted with phenylephrine, electrical field stimulation (EFS), acetylcholine and exogenous nitric oxide (NO) induced relaxations in the control group. Ethanol treatment abolished the endothelium-dependent relaxations induced by acetylcholine but failed to alter the relaxation to EFS and NO. L-nitroarginine, a NO synthase inhibitor, reduced relaxations induced by EFS and acetylcholine, but not those induced by NO in control and ethanol-treated mice. L-arginine prevented the response inhibited by L-nitroarginine. ODQ, a guanylyl cyclase inhibitor, inhibited relaxations in response to EFS, NO and acetylcholine in control and ethanol-treated mice. Corpus cavernosum tissues were investigated using electron microscopy and endothelial damage was observed in ethanol-treated mice. These results suggest that ethanol impairs the endothelial function of corpus cavernosum in mouse, and it may lead to erectile dysfunction through a reduced NO release *via* endothelial impairment.

Key words:
corpus cavernosum, ethanol, endothelium, neurogenic-and-endothelium-dependent relaxation, nitric oxide