Vascular Actions of Nitric Oxide as Affected by Exposure to Alcohol.

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Abstract
Vasodilator substances liberated from endothelial cells, mainly nitric oxide (NO), play important roles in physiologically regulating blood flow and blood pressure and preventing pathological vascular damage. Impairment of these actions promotes the genesis of cardiovascular diseases such as hypertension, cerebral and cardiac hypoperfusion, impaired vasodilatation and atherosclerosis. Low concentrations of alcohol induce increased release of NO from the endothelium due to activation and expression of NO synthase (NOS). In contrast, administration of high concentrations of alcohol or its chronic ingestion impairs endothelial functions in association with reduced NO bioavailability. The endogenous NOS inhibitor asymmetric dimethylarginine may participate in decreased synthesis of NO. Chronic alcohol intake also impairs penile erectile function possibly by interfering with endothelial, but not nitrergic nerve, function. This review article summarizes the vascular actions of NO derived from endothelial and neuronal NOS as affected by alcohol, other than wine, and acetaldehyde in healthy individuals, human materials and various experimental animals.

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