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JOURNAL ARTICLE

Evidence for a role of intracellular-calcium release in nitric oxide-stimulated relaxation of the rabbit corpus cavernosum

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Erection is mediated by relaxation of the smooth muscle elements within the sinusoids of the corpus cavernosum. Although cavernosal relaxation can be mediated by a variety of mechanisms including purinergic stimulation, prostoglandins, and beta-adrenergic stimulation the major mechanism involves the stimulated release of nitric oxide (NO) and subsequent relaxation of the corporal smooth muscle. Experimentally, NO can be released both by direct stimulation of NO-containing nerves (using field stimulation) and indirectly via cholinergic stimulation of NO release from the endothelium (using bethanechol). Preliminary studies have indicated that NO release and/or NO-stimulated relaxation of corporal smooth muscle is an active process involving both an increase in cytosolic calcium and an increase in metabolic energy utilization. Ryanodine is a pharmacological agent that can inhibit calcium-stimulated calcium release from the sarcoplasmic reticulum. The results of the current study demonstrated that ryanodine inhibited both field-stimulated relaxation and bethanechol-stimulated relaxation but did not affect relaxation induced by adenosine triphosphate (ATP) or nitroprusside. These studies strongly support the hypothesis that NO-stimulated relaxation is mediated, in part, by calcium release from the sarcoplasmic reticulum through ryanodine-sensitive channels.