AB035. Vasohibin-1 as a potential therapeutic target for erectile dysfunction

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Abstract: Induction of neovascularization is a promising strategy to treat erectile dysfunction (ED). Here we for the first time report the unexpected role of vasohibin-1, which is previously defined as an anti-angiogenic factor, in the restoration of erectile function by enhancing cavernous (penile) angiogenesis in an animal model of diabetic ED. The cavernous expression of vasohibin-1 was down-regulated in diabetic mice and in patients with diabetic ED; vasohibin-1 was mainly expressed in endothelial cells. Vasohibin-1 knockout mice revealed decrease in cavernous endothelial and pericyte content compared with wild type mice, which resulted in deterioration of erectile function. Local delivery of vasohibin-1 peptide into the corpus cavernosum of diabetic mice induced significant restoration of erectile function, which reached up to 85% of control values at the concentration of 4 μg/20 μL. Vasohibin-1 significantly increased cavernous endothelial cell content and induced eNOS phosphorylation (Ser1177). Vasohibin-1 decreased extravasation of oxidized-LDL by restoring pericytes and endothelial cell-cell junction proteins in the diabetic mice. By using proteome profiler array kit, we found that the induction of angiogenic factors, including angiopoietin-1, vascular endothelial growth factor, and basic fibroblasts growth factor, mainly in fibroblasts is a major molecular mechanism responsible for vasohibin-1-mediated cavernous angiogenesis and subsequent restoration of erectile function. Our findings suggest that vasohibin-1 is proangiogenic in diabetic penis and is a promising therapeutic target for ED from vascular causes.

Keywords: Erectile dysfunction (ED); diabetes mellitus; endothelial dysfunction; therapeutic angiogenesis; vasohibin-1

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