Myocardin

Myocardin (MYOCD), a key contractility regulator of vascular Smooth Muscle Contractility SMCs, is a critical factor in intracranial aneurysm (IA) progression. Using a multifaceted computational and experimental approach, Zhang et al. determined that depletion of competitive endogenous RNAs (ARHGEF12, FGF12, and ADCY5) enhanced factors that downregulate MYOCD, which induces the conversion of SMCs from differentiated contractile states into dedifferentiated phenotypes that exhibit enhanced proliferation, synthesis of new extracellular matrix, and organization of mural thrombi. These effects may lead to the repair and maintenance of IAs. The study of Zhang et al. presents guidelines for the prediction and validation of the IA regulator MYOCD in competitive endogenous RNA

Serum response factor (SRF)-MYOCD overexpression in small cerebral arteries appears to initiate independently of Abeta a pathogenic pathway mediating arterial hypercontractility and CBF dysregulation, which are associated with Alzheimer's dementia².

Thus, drugs that specifically disrupt SRF-MYOCD interaction in small brain vessels may hold potential to improve brain perfusion and CBF dysregulation in AD³⁾.

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