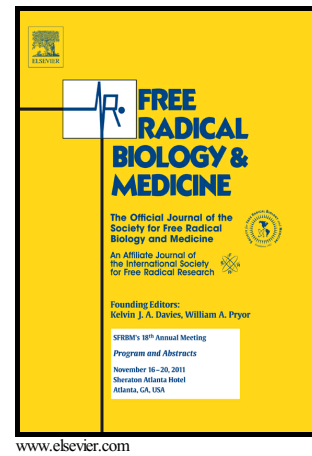


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Anagliptin inhibits neointimal hyperplasia after balloon injury via endothelial cell-specific modulation of SOD-1/RhoA/JNK signaling in the arterial wall

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Abstract

Intimal hyperplasia is one of the major complications after stenting, but the underlying mechanisms remain unclear. Our previous study found that the dipeptidyl peptidase IV (DPP-4) inhibitor, Anagliptin, suppresses intimal hyperplasia after balloon injury. Here, we further investigated the effects of Anagliptin on endothelial cell (EC) migration after balloon injury. The results showed that Anagliptin administration significantly reduced intimal hyperplasia by stimulating the migration of endothelial cells, but had no effect on the medial area after balloon injury. Anagliptin elevated the total plasma activity of SOD by up-regulating the level of SOD-1, but not SOD-2, after balloon injury. Meanwhile, pre-incubation with Anagliptin suppressed the hydrogen peroxide-mediated formation of oxidant species and apoptosis in HUVECs. In vitro pre-incubation with Anagliptin promoted the migration of HUVECs via the SOD-1/RhoA/JNK signaling pathway mediating the

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