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Titel des Beitrags:
EMAP-II downregulation contributes to the beneficial effects of rapamycin after vascular injury.

Abstract:
AIMS: Neointima formation after vascular injury is strongly associated with inflammation. Rapamycin inhibits human neointima formation and reduces expression of the proinflammatory cytokine endothelial-monocyte activating peptide II (EMAP-II) in vitro. Here we investigated the interplay between EMAP-II and rapamycin after vascular injury in vivo. METHODS AND RESULTS: In a mouse model of vascular injury, mice were either not treated, given everolimus, a rapamycin derivate, or subjected to simultaneous challenge with everolimus and EMAP-II. EMAP-II expression was measured in coronary artery smooth muscle cells (CASMC) and monocytic cells in vitro and in patients after percutaneous coronary intervention (PCI). After vascular injury, rapamycin reduced neointima formation and adventitial thickening. Immunohistochemistry revealed reduced EMAP-II protein expression and suppressed recruitment of inflammatory cells. Simultaneous challenge with EMAP-II counteracted these effects of rapamycin. Expression of EMAP-II and its inhibition by rapamycin was confirmed in CASMC and monocytic cells. In patients, EMAP-II upregulation was confined to PCI of distal coronary artery segments and profoundly suppressed by oral rapamycin treatment. CONCLUSION: These data...
suggest important yet unrecognized roles of EMAP-II and adventitial inflammation in neointima formation: Through inhibition of EMAP-II, rapamycin reduces the recruitment of inflammatory cells to the adventitia and supports an early and bland healing.