Rolex Oyster Perpetual Datejust Serial Number 045

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16 Jun 2015 - 25 sec - Uploaded by WATCHWORLDReady for a Rolex The Rolex Website is a Leading Reseller of Rolex Watches.Angiogenesis is implicated in pathogenesis of atherosclerosis through recruitment of macrophages. However, the factors that drive macrophage recruitment and arteriogenesis and their mechanistic link to plaque stability are not known. The overall hypothesis of this research proposal is that endoglin - the transforming growth factor-beta receptor that is highly expressed in macrophages - modulates arteriogenesis and plaque stability by promoting activation of endothelial progenitor cells and their transmigration. The two specific aims are designed to test this hypothesis. The first specific aim will test the premise that endoglin promotes a regenerative microenvironment in the plaque by acting on the transforming growth factor-beta/Smad2-Smad3 pathway and promotes macrophage cell proliferation. The second specific aim will investigate the basis for altered Smad2 and Smad3 expression by endoglin and their functional significance in macrophage biology. The proposed studies will provide mechanistic insights into the role of endoglin in atherosclerosis and address the pathophysiology of plaque instability and atherothrombotic stroke. The outcomes of this research proposal will contribute to a better understanding of the pathogenic link between atherosclerotic disease and stroke. PUBLIC HEALTH RELEVANCE: The overall hypothesis of this research proposal is that endoglin - the transforming growth factor-beta receptor that is highly expressed in macrophages - modulates arteriogenesis and plaque stability through activation of endothelial progenitor cells and their transmigration and that this mechanism promotes the stability of vulnerable atherosclerotic lesions. The proposed studies will provide mechanistic insights into the role of endoglin in atherosclerosis and address the pathophysiology of plaque instability and atherothrombotic stroke.The ability of bone morphogenetic protein (BMP)-2 to cause ectopic bone formation has recently been proposed as a mechanism for cyclosporine-induced gingival overgrowth in organ transplantation. In the proposed experiments, we will begin to address the mechanism of BMP-2-induced bone growth in vivo. The first hypothesis to be tested is that BMP-2 is internalized into target cells and induces its effects through non-autocrine mechanism in cells surrounding the target tissue. Both in vitro and in vivo models will be used in this study. The second

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