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Sex steroids block the initiation of atherosclerosis

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Atherosclerosis is the main cause of death in men and women. This so-called ‘‘hardening of the arteries’’ results from advanced atherogenesis, the accumulation and death of subendothelial fat-laden macrophages (vascular plaque). The macrophages are attracted as the result of signals from injured vessels recruiting and activating cells to quell the injury by inflammation. Among the recruited cells are circulating monocytes that may be captured by the formation of neural cell adhesion molecule (nCAM) tethers between the monocytes and vascular endothelium; the tethers are dependent on electrostatic binding between distal segments of apposed nCAM molecules. The capture of monocytes is followed by their entry into the subendothelial area as macrophages, many of which will remain and become the fat-laden foam cells in vascular plaque. Neural cell adhesion molecules are subject to sialylation that blocks their electrostatic binding. We showed that estradiol-induced nCAM sialylases are present in vascular endothelial cells and tested whether sex steroid pretreatment of human vascular endothelium could inhibit the capture of monocytes. Using in vitro techniques, pretreatment of human arterial endothelial cells with estradiol, testosterone, dehydroepiandrosterone and dihydrotestosterone all induced sialylation of endothelial cells and, in a dose–response manner, reduced the capture of monocytes. Steroid hormones are protective against atherogenesis and its sequellae. Sex steroid depletion is associated with atherosclerosis. Based on this knowledge plus our results using sex steroid pretreatment of endothelial cells, we propose that the blockade of the initial step in atherogenesis by sex steroid-induced nCAM sialylation may be crucial to hormonal prevention of atherosclerosis.

Naftolin F, Mehr H, Fadiel A. Sex Steroids Block the Initiation of Atherosclerosis. Reprod Sci. 2016 Dec;23(12):1620-1625.

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