

Thalidomide inhibits early atherogenesis in apoE-deficient mice.

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Inflammation is present in all stages of atherosclerosis, from fatty streaks to rupture of mature plaques. Tumour necrosis factor (TNF)-alpha is expressed in atherosclerotic lesions but its role in atherogenesis has not been defined. To clarify the role of this cytokine, we administered thalidomide, a compound known to inhibit TNF-alpha production, to homozygous apolipoprotein E-deficient (apoE^{-/-}) mice in order to examine the effect of thalidomide on the development of early atherosclerotic lesions. Twelve apoE^{-/-} mice were randomized to receive either sustained-release thalidomide or placebo pellets implanted subcutaneously, and the amount of atherosclerosis was quantified six weeks later. Thalidomide was well tolerated and did not result in any changes in body weight. Mice treated with thalidomide had significantly smaller mean (7986 +/- 5189 vs 19607 +/- 10353 microns², p = 0.05) and maximum (15800 [12777-23675] vs 37169 [28000-41351] microns², p = 0.03) lesion sizes than those treated with placebo. Thus, thalidomide is capable of inhibiting the early development of atherosclerosis, presumably by inhibition of TNF-alpha secretion.

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