The paradoxical effects of statins on the cancer cells and endothelial cells; the impact of concentrations

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Abstract

Beside their lipid lowering functions, statins impose some additional effects known as the pleotropic effects. In endothelial progenitor cells (EPO) and cancer cells statins have been shown to impose the opposite effects on the cell cycle, senescence and apoptosis. The most significant reason may arise from bias in selecting the applied doses in cancer cells compared to those used in EPO cells; in EPOs it was evident that lower (nanomolar) concentrations of statins impose anti-senescence, anti-apoptotic, and angiogenic effects; however, higher doses (micromolar) imposed the adverse effects. In cancer cells, most studies used high (micromolar) doses of statins showing statins-induced apoptosis. Some studies indicated that even at low concentrations (nanomolar) statins induced senescence or imposed cytostatic but not cytotoxic effects. Regardless of the concentrations, statins in cancer cells induce apoptosis or cell cycle arrest, anti-proliferation effects, and cause senescence. However, their effects on EPOs depend on the concentrations.

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