Rosuvastatin: is it a promising agent for melanoma chemoprevention?

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Introduction

Evidence from preclinical and clinical studies indicate that statin use may be associated with lower incidence of melanoma. Inhibition of HMG-CoA reductase decreases synthesis not only of cholesterol but also other intermediates of mevalonate pathway that are involved in protein prenylation. Considering the role of prenylated proteins in regulation of cellular processes connected with melanomagenesis, we studied anticancer activity of rosuvastatin against human melanoma cell lines *in vitro*.

Materials and methods

In our study melanoma cell lines (A375 and WM1552C) established from a primary superficial spreading melanoma were used. Cells were treated with rosuvastatin at concentrations ranged from 0,01-10μg/ml for 24-72h. To asses *in vitro* cytotoxicity of rosuvastatin MTT assay and real time cell growth analysis (xCELLigence system) were performed. We also analyzed influence of rosuvastatin on apoptosis induction and cancer cell cycle progression.

Results

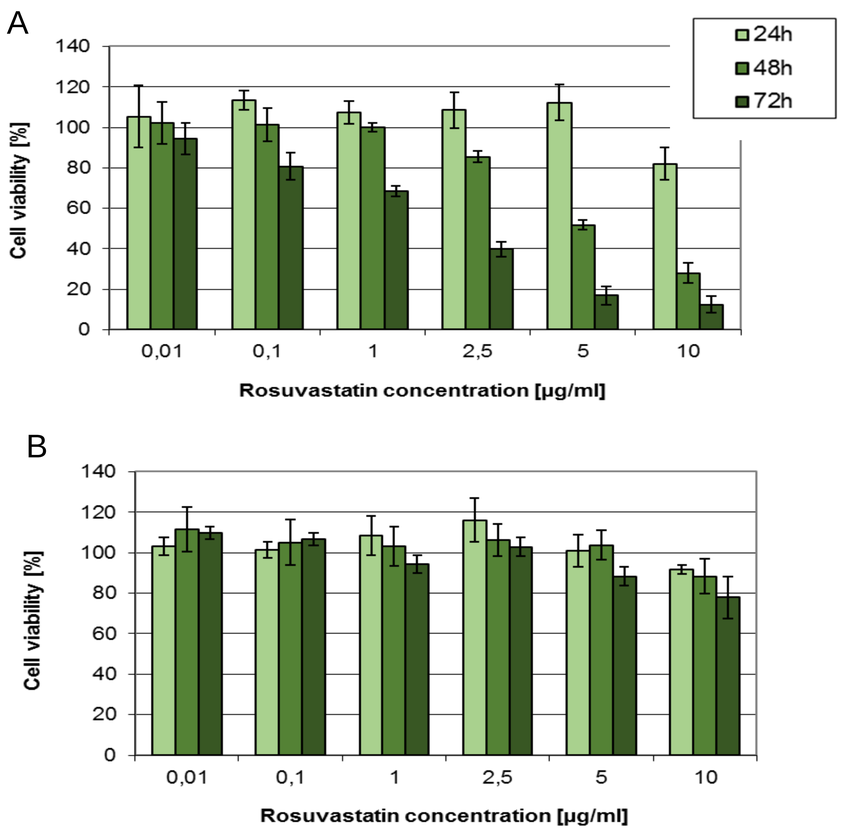

Fig. 1 Viability of (A) A375 and (B) WM1552C cells after 24-72h treatment with rosuvastatin assessed on the basis of MTT assay.

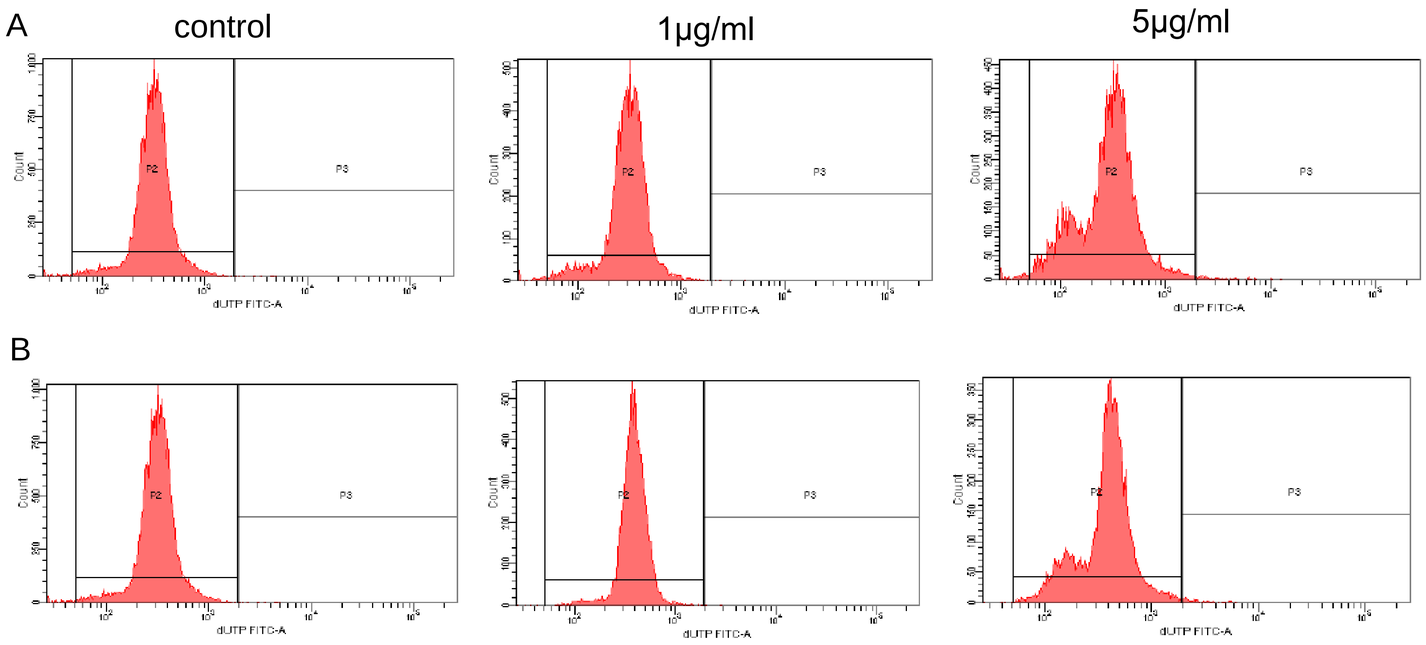

Fig. 3 Apoptosis detection by labeling DNA breaks with FITC-dUTP (end labeling/TUNEL) in (A) A375 and (B) WM1552C cells after 48h treatment with rosuvastatin.

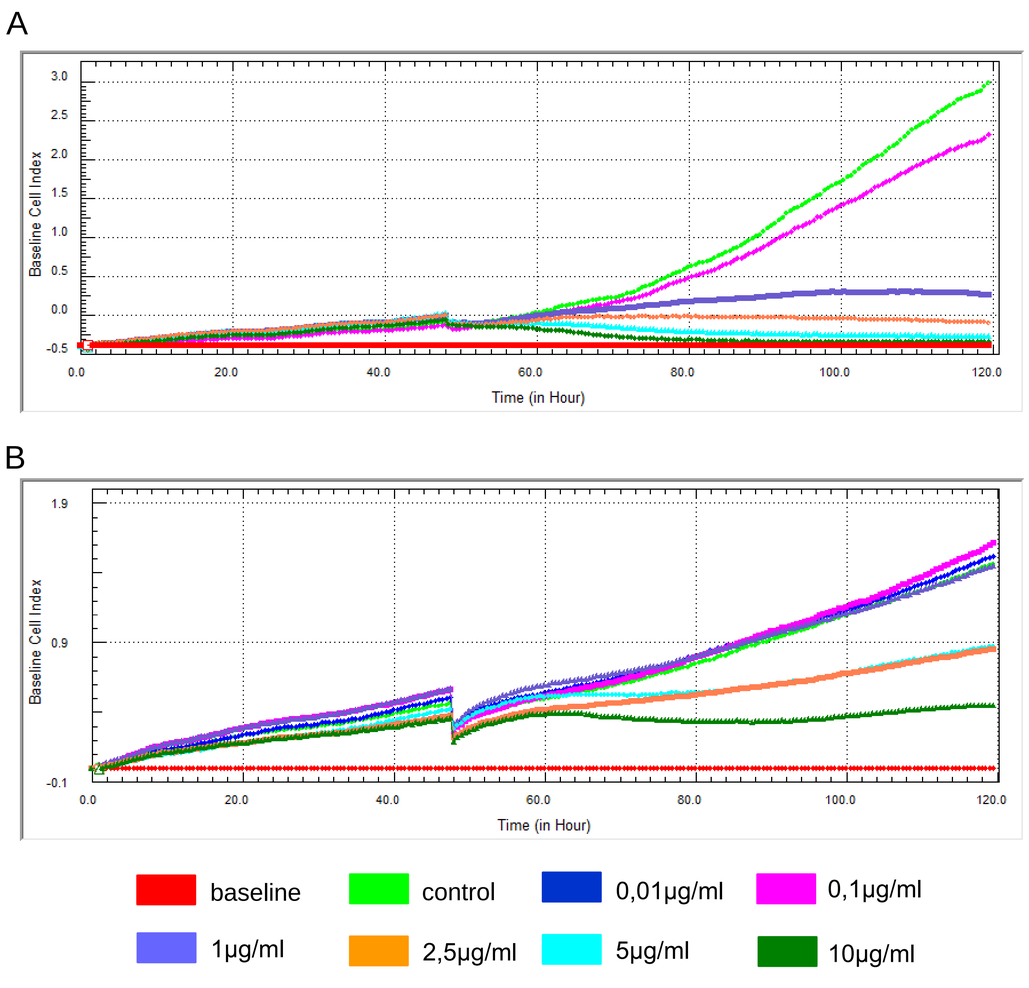

Fig. 2 Viability of (A) A375 and (B) WM1552C cells after 72h treatment with rosuvastatin assessed on the basis of real time cel growth analysis (xCELLigence system).

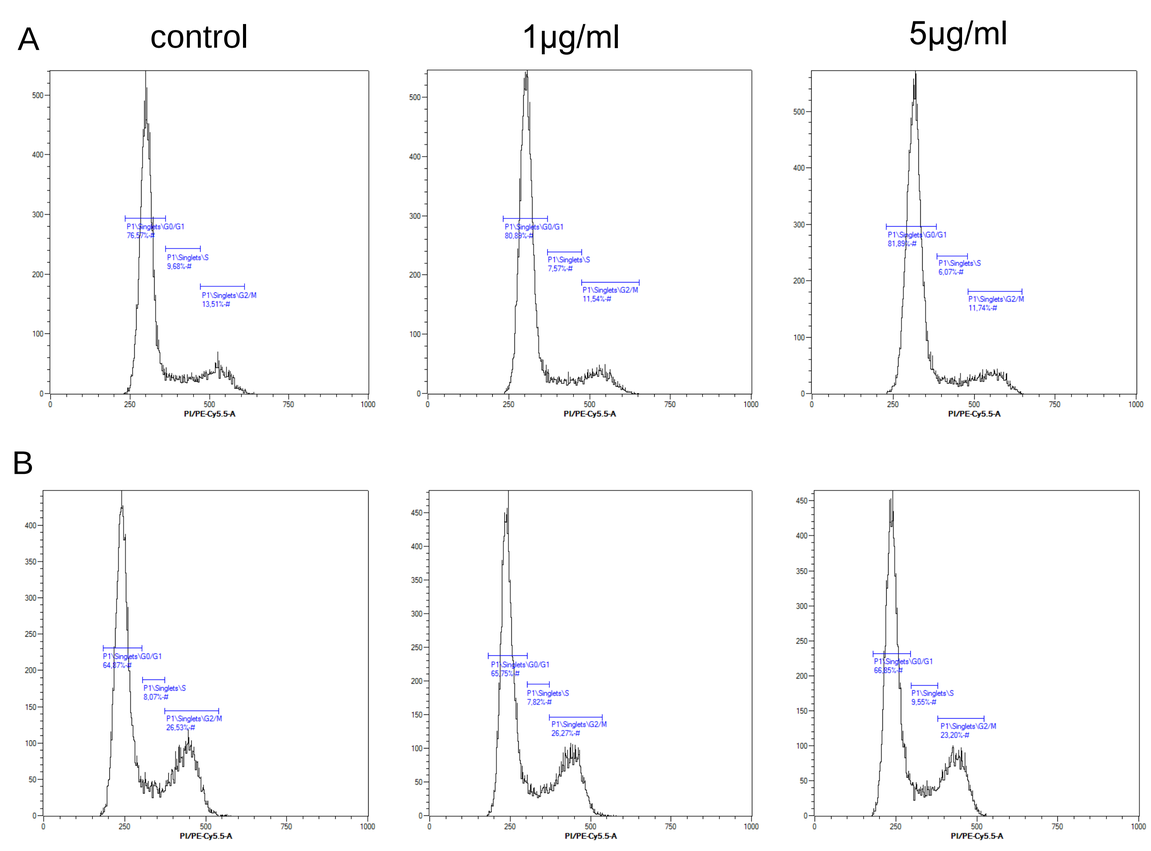

Fig. 4 Cell cycle analysis by quantitation of DNA content (propidium iodide staining) in (A) A375 and (B) WM1552C cells after 48h treatment with rosuvastatin.

Conclusion

The results of our study indicates that sensitivity to rosuvastatin is highly dependent on cancer cell line assessed. We also showed that rosuvastatin does not induce apoptosis and cell cycle perturbations. Thus, considering high concentrations required to decrease cancer cells viability *in vitro*, that exceed 10 to 50-fold plasma concentrations reached in patients treated with rosuvastatin, its potential use in melanoma chemoprevention seems to be limited.