

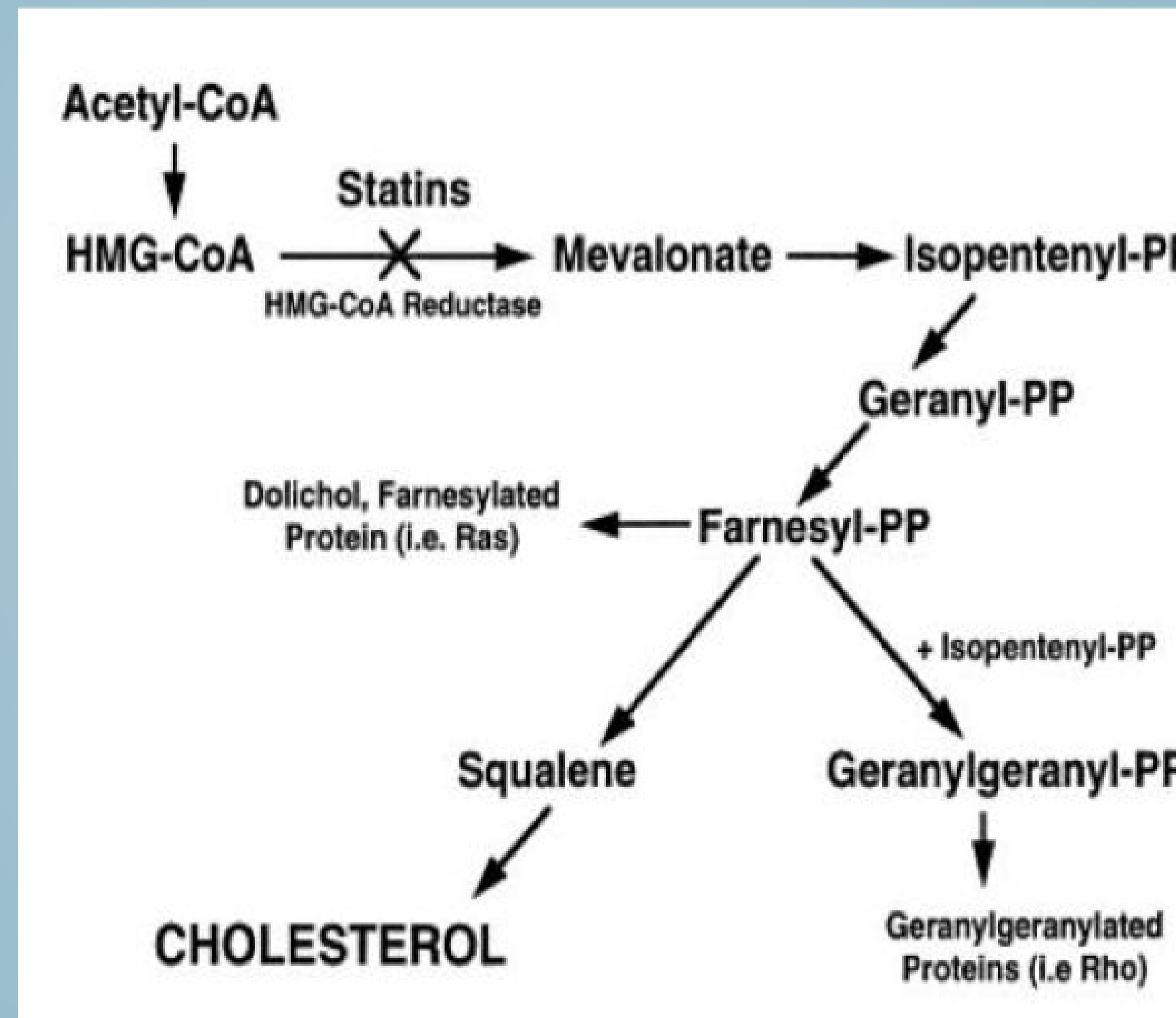
# Use of Lovastatin in Fibroblast Healing and Inhibition of HMGCoA

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## Introduction

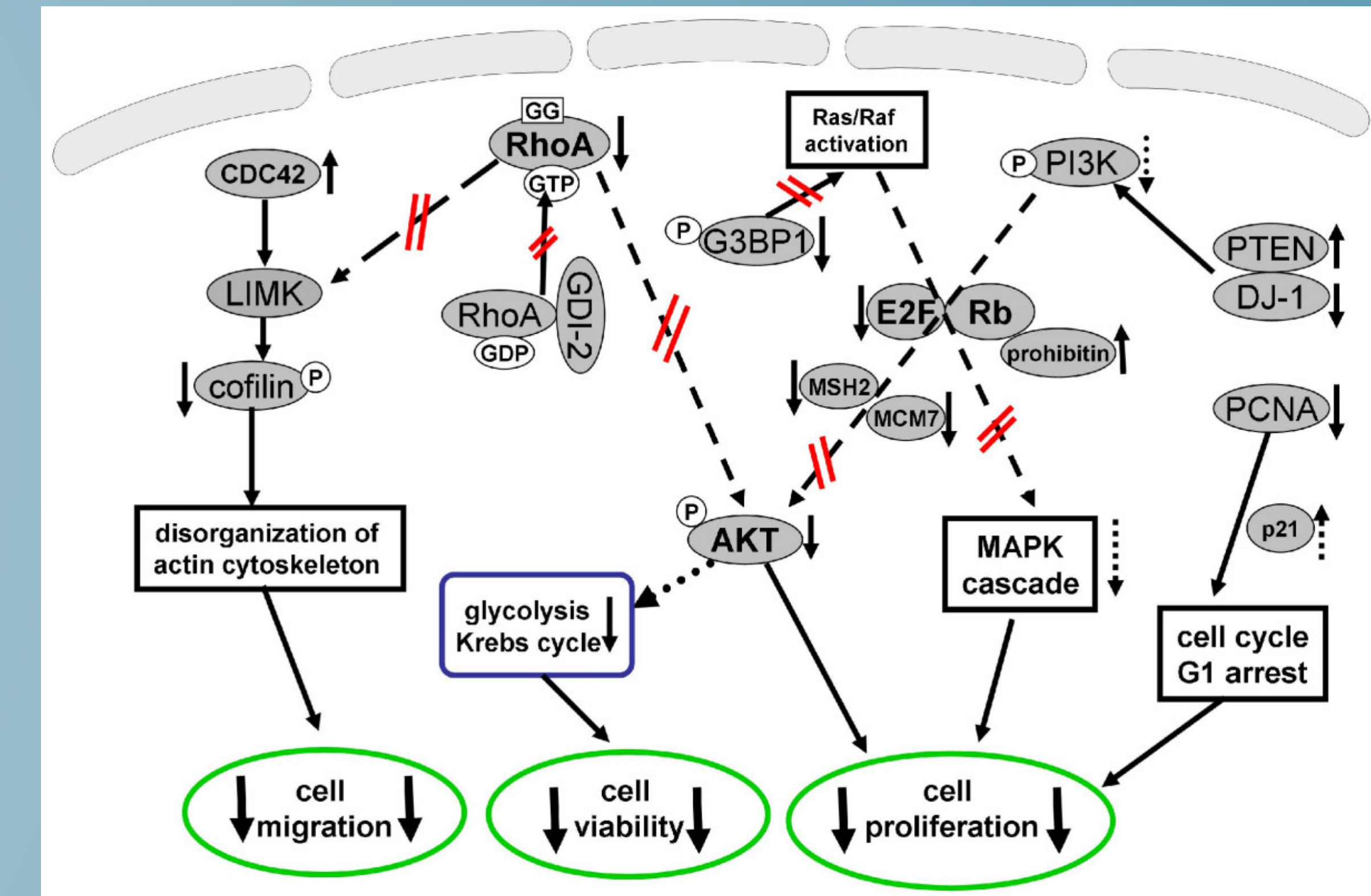
Lovastatin is a competitive inhibitor to HMG-CoA, which is a necessary material to the synthesis of Cholesterol. Resultantly, introducing Lovastatin to a system causes a reduction in cholesterol levels. However, multiple independent teams have shown statins to increase the rate of wound healing at low concentrations (~6nM). The purpose of this investigation is to determine the mechanism by which lovastatin functions in wound-healing, as influenced by its role in cholesterol inhibition.

Lovastatin was applied to the media surrounding 3T3 fibroblasts in various concentrations, listed at the right of the table below, and wounds were applied to the cell layers. The linear regression of wound closures for different groups are listed below.



**Above:** Role of Statins in the inhibition of cholesterol production.

**Below:** Effect of high-concentration lovastatin on the closure of wound gaps in fibroblasts.



## Conclusion

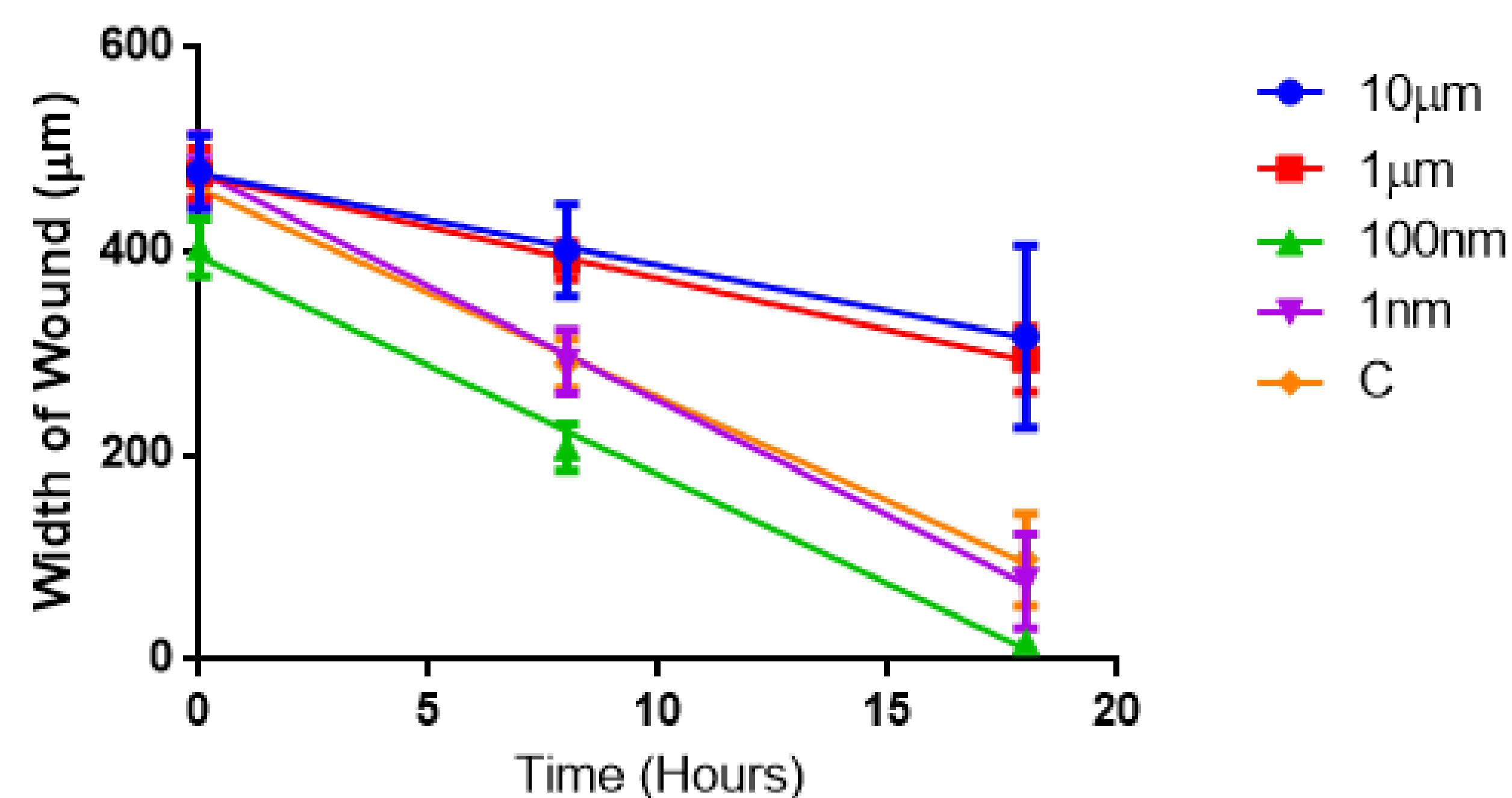
Lovastatin, acting as an inhibitor to HMG-CoA, limits a cell's capacity to produce cholesterol. In larger concentrations, this results in stunting cell growth, while in small concentrations, this appears to have no significant effect on cell growth.

This implies that, in the small concentrations at which Lovastatin is applied for accelerated wound-healing, the cholesterol inhibition is either at a beneficial level or effectively counteracted by the secondary function of the drug.

## Acknowledgements

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## Wound Closure



For the larger two concentrations applied, wound closure was significantly impaired, while, at concentrations of 100nM and 1nM, there was no significant difference from the control displayed.

