Simvastatin Reduces Neointimal Thickening After Experimental Angioplasty

To the Editor:

We read with interest the study by Chen and associates that showed that simvastatin reduces neointimal proliferation in mice after vascular injury in a cholesterol-independent manner. However, we already documented this finding in vitro and in vivo in a previous article.

In fact, it has been documented that simvastatin inhibits in vitro smooth muscle cell proliferation independent of cholesterol. In addition, in vivo experiments have shown that statins potently affect neointimal proliferation in a dose-dependent manner in a reliable model of balloon injury in the rat. This effect was abolished by local administration of mevalonate. We are delighted that the study of Chen et al confirms, in genetically modified mice, our findings.

With regard to the potential mechanism of statin on neointimal proliferation, we previously discussed a key role of ras pathway in neointimal proliferation after balloon injury. Recently, we also showed that statins powerfully inhibit ras farnesilation and activation. Therefore, statin-induced Ras-MAPKKs pathway inhibition may play a critical role in the effect of simvastatin on neointimal formation after vascular injury.

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