

This week in techniques

Approach	Summary	Licensing status	Publication and contact information
Disease models			
Apolipoprotein E (APOE)-mediated regulation of factors contributing to atherosclerotic lesion formation	<p>Mouse studies showing how APOE regulates downstream factors that contribute to atherosclerosis could help guide the development of treatments of the disease. ApoE-deficient mice had greater hematopoietic stem and progenitor cell proliferation and higher monocyte accumulation in atherosclerotic plaques than nondeficient controls. In ApoE-deficient mice, infusion of recombinant high-density lipoprotein (HDL) suppressed stem and progenitor cell proliferation compared with saline infusion. Next steps could include identifying APOE-associated targets that decrease proliferation of those cells and lower atherosclerotic plaque formation.</p> <p><i>SciBX</i> 4(40); doi:10.1038/scibx.2011.1126 Published online Oct. 13, 2011</p>	Patent and licensing status undisclosed	<p>Murphy, A.J. <i>et al.</i> <i>J. Clin. Invest.</i>; published online Sept. 26, 2011; doi:10.1172/JCI57559 Contact: Andrew J. Murphy, Columbia University, New York, N.Y. e-mail: am3440@columbia.edu</p>