Overexpression of Human Apolipoprotein A-II in Transgenic Mice Does Not Impair Macrophage-Specific Reverse Cholesterol Transport In Vivo

Noemí Rotllan, Vicent Ribas, Laura Calpe-Berdiel, Jesús M. Martín-Campos, Francisco Blanco-Vaca, Joan Carles Escolà-Gil

Background—Overexpression of human apolipoprotein (apo) A-II in transgenic mice induces high-density lipoprotein (HDL) deficiency, and increased atherosclerosis susceptibility only when fed an atherogenic diet. This may, in part, be caused by impairment in reverse cholesterol transport (RCT).

Methods and Results—[3H]cholesterol-labeled macrophages were injected intraperitoneally into mice maintained on a chow diet or an atherogenic diet. Plasma [3H]cholesterol did not differ from human apoA-II transgenic and control mice at 24 or 48 hours after the label injection. On the chow diet, human apoA-II transgenic mice presented increased [3H]cholesterol in liver (1.3-fold) and feces (6-fold) compared with control mice (P<0.05). The magnitude of macrophage-specific RCT did not differ between transgenic and control mice fed the atherogenic diet.

Conclusions—Human apoA-II maintains effective RCT from macrophages to feces in vivo despite an HDL deficiency. These findings suggest that the increased atherosclerotic lesions observed in apoA-II transgenic mice fed an atherogenic diet are not caused by impairment in macrophage-specific RCT. (Arterioscler Thromb Vasc Biol. 2005;25:e128-e132.)

Key Words: apolipoprotein A-II ■ atherosclerosis ■ high-density lipoprotein ■ macrophages ■ reverse cholesterol transport

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