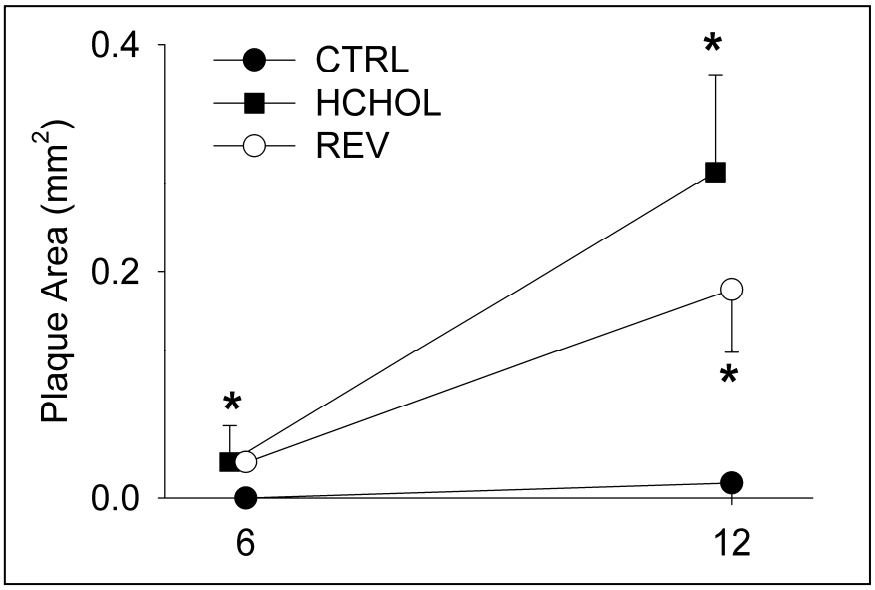

Figure I. Atherosclerotic plaque area in aorta. Plaque size is markedly elevated in hypercholesterolemic mice at 6 and 12 months, and tends to be attenuated by reducing cholesterol levels (p = n.s. versus 12 month HCHOL). CTRL = normocholesterolemic group, HCHOL = hypercholesterolemic group, REV = "reversed"/regression group. * = $p < 0.05$ versus iso-time CTRL group.

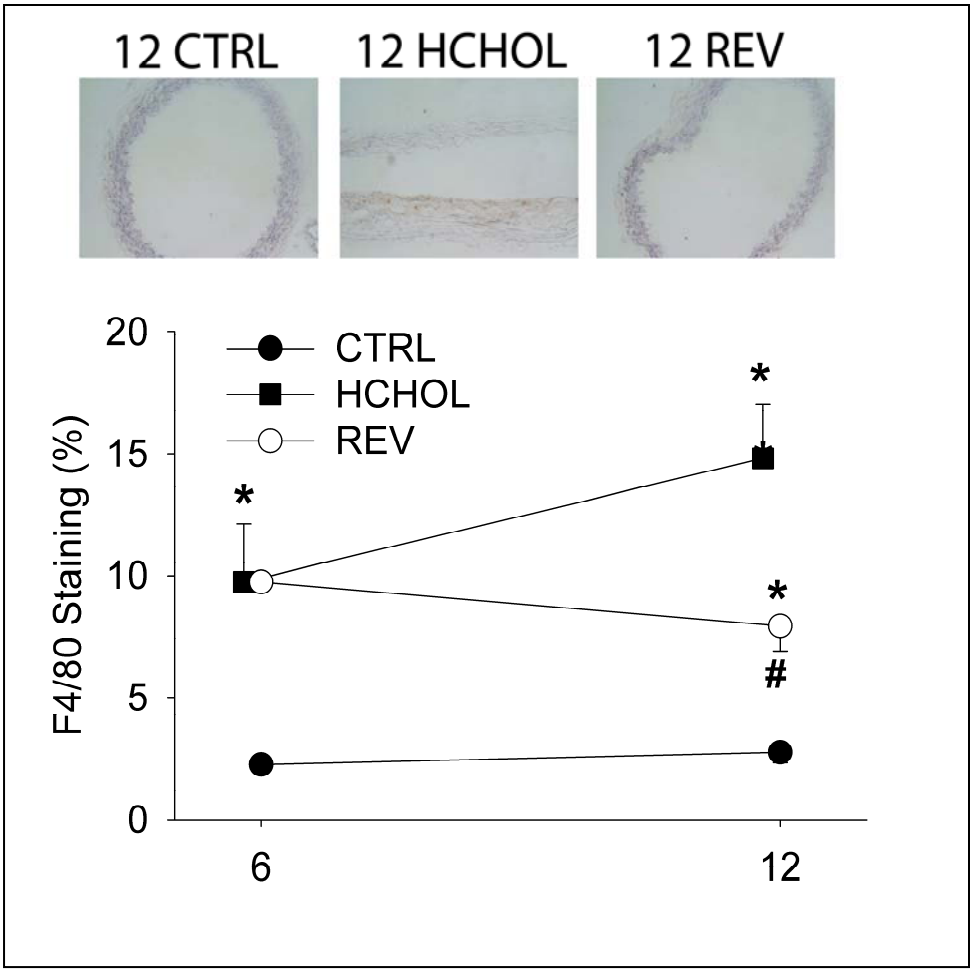

Figure II. Macrophage levels (F4/80 immunostaining) in aorta. Macrophage levels are markedly elevated in hypercholesterolemic mice at 6 and 12 months (brown staining), and are dramatically attenuated by reducing cholesterol levels. CTRL = normocholesterolemic group, HCHOL = hypercholesterolemic group, REV = "reversed"/regression group. * = $p < 0.05$ versus iso-time CTRL group; # = $p < 0.05$ versus 6 month HCHOL group.

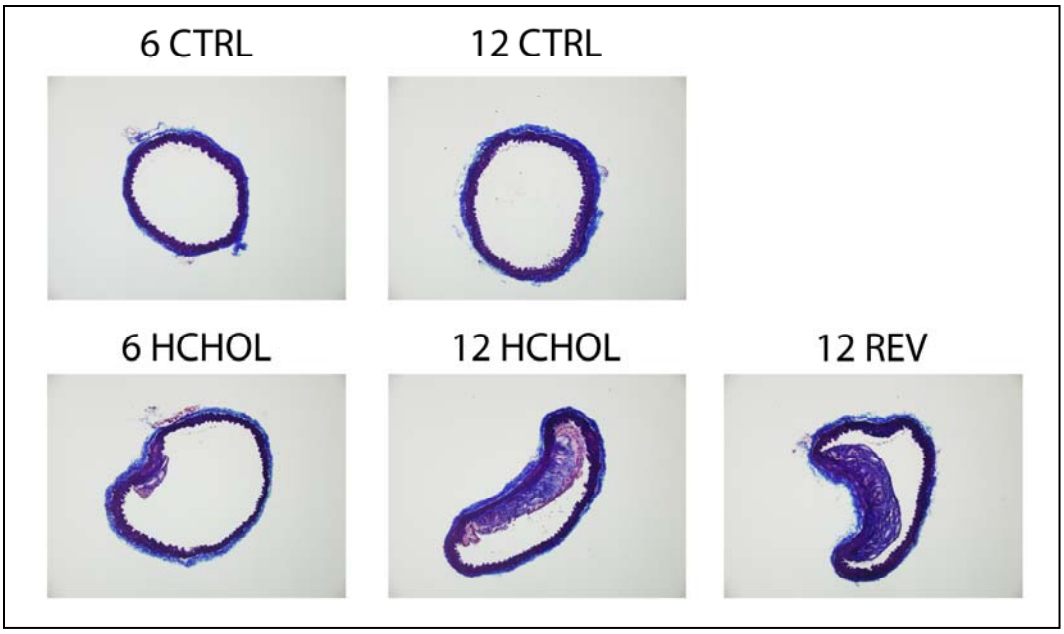

Figure III. Plaque fibrosis (Masson's trichrome staining) in aorta. Aortic plaques in hypercholesterolemic mice at 6 and 12 months have lighter blue staining, indicating lower collagen content/fibrosis. Note that intensity of blue staining is markedly increased by reducing cholesterol levels, indicating an increase in plaque fibrosis and stability. CTRL = normocholesterolemic group, HCHOL = hypercholesterolemic group, REV = "reversed"/regression group.

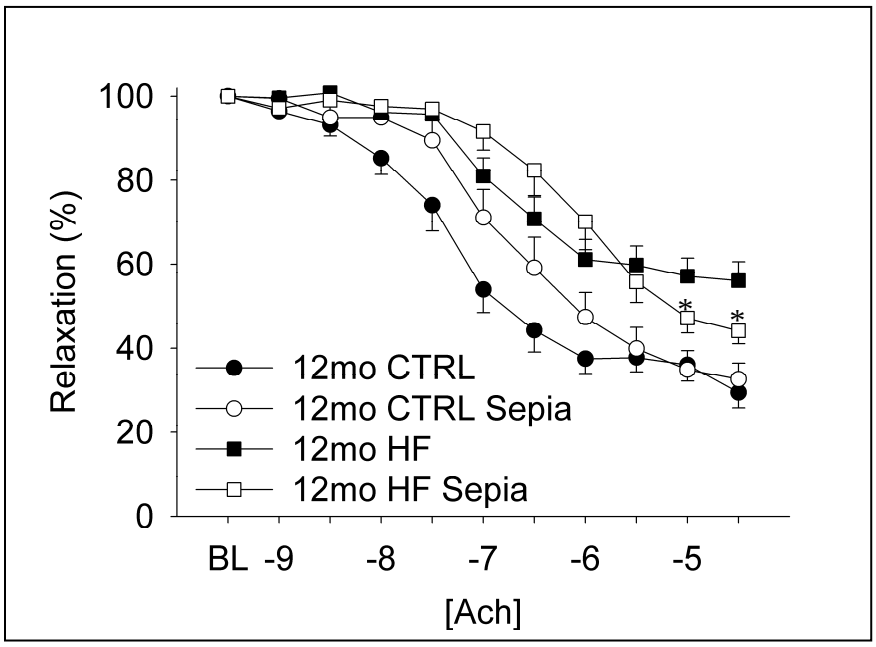

Figure IV. Changes in endothelial function after incubation of vessel segments with a tetrahydrobiopterin precursor (Sepiapterin) in control and hypercholesterolemic at 12 months. Acute treatment with Sepiapterin does not improve responses to acetylcholine in normocholesterolemic mice, but improves maximal relaxation to acetylcholine in mice with prolonged, severe hypercholesterolemia.