

**Interaction of Blood Pressure and LDL Cholesterol in Early Atherosclerosis. The Los Angeles Atherosclerosis Study**

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**Background.** The response to injury model of atherosclerosis has been investigated in animal models but not in epidemiologic studies. Relations between LDL cholesterol and carotid intima-media thickness (IMT) within levels of SBP provide a test of this model.

**Methods.** Data are from a longitudinal study of 573 randomly sampled asymptomatic employees of a large company aged 40-60 years. IMT and change in IMT over 18 months ( $\Delta$ IMT) were determined sonographically in the common carotid artery. 497 subjects were available for cross-sectional analysis. To investigate interactive effects of SBP and serum LDL on IMT, linear slopes ( $\beta \pm$ SE in mm/mmol/L) of IMT regressed on LDL were computed within SBP tertiles: Low 93-122, Middle 123-131, and High 132-175 mmHg. Covariates were age, body height, sex, body mass index, ethnicity, diabetes, smoking status, and treatment for hypertension or hypercholesterolemia. Analysis of  $\Delta$ IMT in 414 subjects was similar.

**Results.** In cross-sectional models, IMT was positively related to LDL in the high SBP group ( $\beta=0.028 \pm .008$ ,  $p=0.0006$ ), but not in the middle ( $\beta=-0.005 \pm .008$ ,  $p=0.51$ ) or low ( $\beta=-0.003 \pm .009$ ,  $p=0.78$ ) SBP groups. These differences in slope between SBP groups were statistically significant ( $p=0.004$  for high vs middle,  $p=0.011$  for high vs low). Results were comparable for the longitudinal analysis:  $\Delta$ IMT was significantly related to LDL in the high SBP group ( $\beta=0.013 \pm .005$ ,  $p=0.009$ ), but not in the middle ( $\beta=-0.006 \pm .005$ ,  $p=0.18$ ) or low ( $\beta=-0.005 \pm .005$ ,  $p=0.31$ ) groups. The differences in slope between SBP groups were again significant ( $p=0.005$  and  $p=0.010$ , respectively).

**Conclusion:** These cross-sectional and longitudinal findings are consistent with the hypothesis that wall injury due to elevated SBP increases the susceptibility of the artery wall to LDL induced atherogenesis.