Supplemental Figure 4: Pairwise scatterplots (below the diagonal), histograms (across the diagonal) and pairwise correlation coefficients (above the diagonal) using RMA normalized gene expression data for *ALOX5*, *ALOX5AP*, *LTA4H, GGTLA1, LTC4S* and *CYP4F2*

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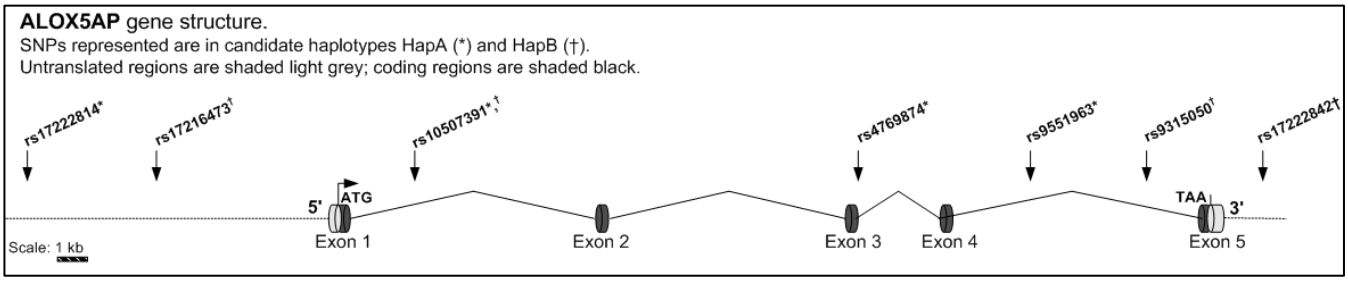
Supplemental Tables

Supplemental Table 1: Table of power for the CATHGEN sample given the HapA, HapB and HapK case-control frequencies and effect sizes found in the Helgadottir et al. Icelandic cohort with MI and EOCAD as the clinical endpoints. The last row contains power estimates for the GENECARD sample at a recurrence risk of ≥ 1.4.

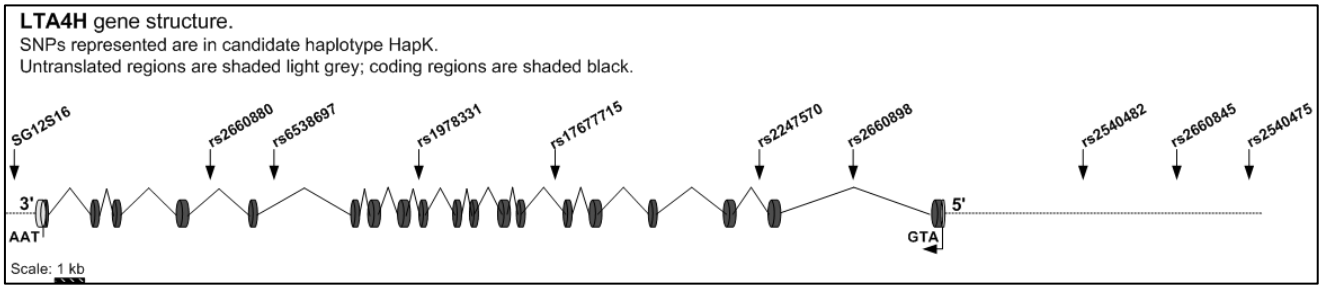
Haplotype	Effect size Relative Risks	Case/Control frequencies reported by Helgadottir et al.	Clinical endpoint in CATHGEN sample	Power
HapA	1.79	0.158 / 0.095	MI	73%
HapA	1.79	0.158 / 0.095	EOCAD	82%
HapB	1.95	0.075 / 0.040	MI	50%
HapB	1.95	0.075 / 0.040	EOCAD	58%
HapK	1.37	0.186 / 0.143	MI in Caucasians	27%
HapK	1.37	0.186 / 0.143	EOCAD in Caucasians	29%
HapK	6.50	0.103 / 0.017	MI in African Americans	49%
HapK	6.50	0.103 / 0.017	EOCAD in African Americans	55%
	Recurrence Risk ≥ 1.4		GENECARD 400 ASPs	>80%

Supplemental Figures

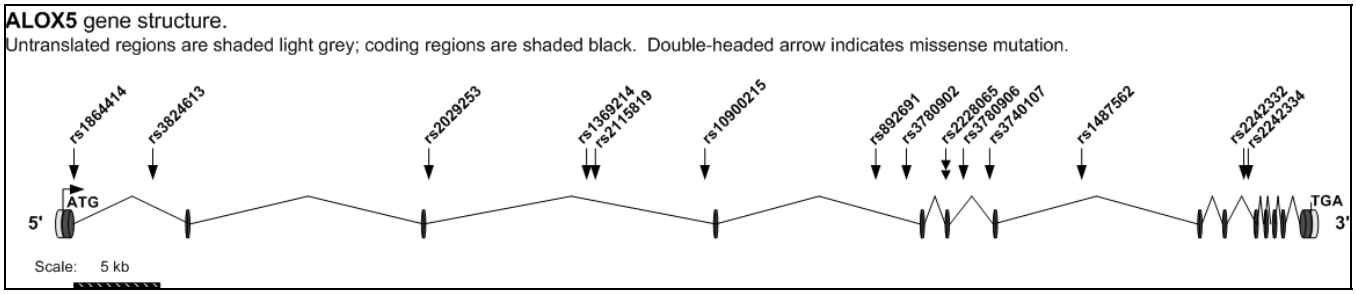
A.



B.

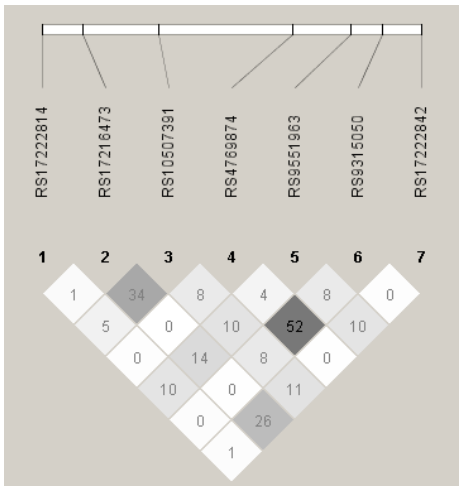


C.

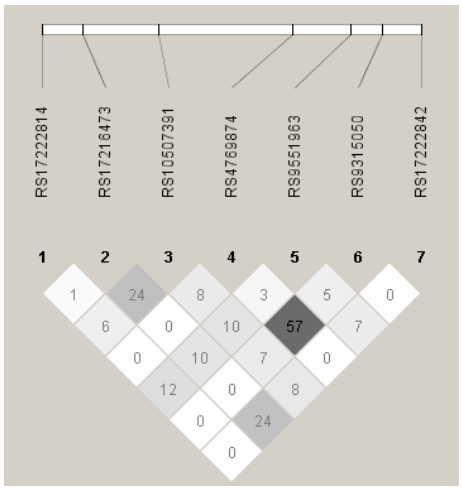


Supplemental Figure 1: Illustration of the *ALOX5AP* (A) *LTA4H* (B.) and *ALOX5* (C.) gene structures with the SNPs composing haplotypes A, B and K. Untranslated regions are shaded light grey; coded regions are shaded black; double-headed arrow indicates missense mutation

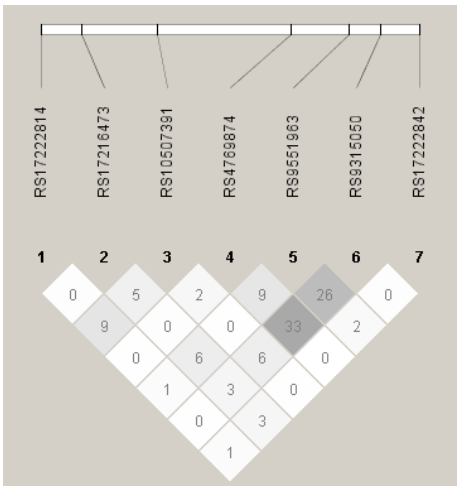
A. *ALOX5AP*



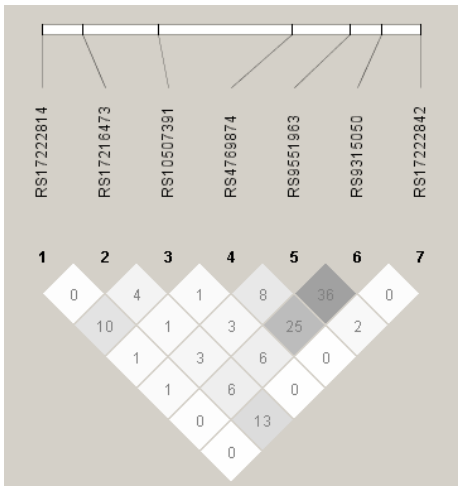
Caucasians CAD



Caucasians Normal



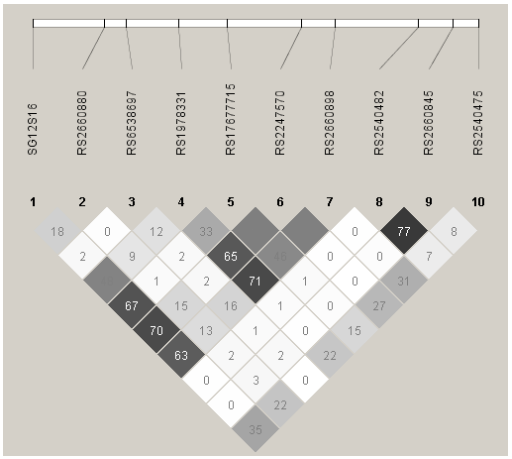
African-American CAD



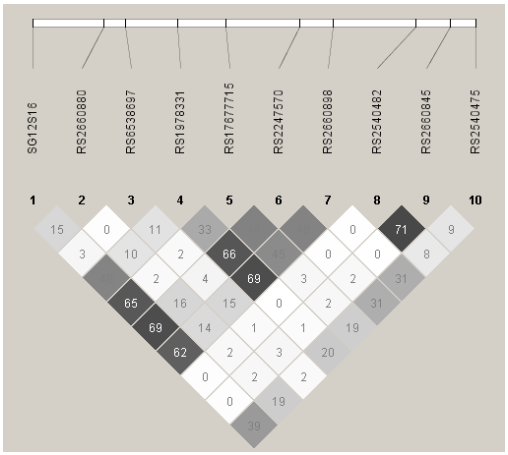
African-American Normal

Supplemental Figure 2A: *ALOX5AP* (including SNPs for HapA and HapB) linkage disequilibrium figures for CAD and controls by ethnicity in the CATHGEN sample

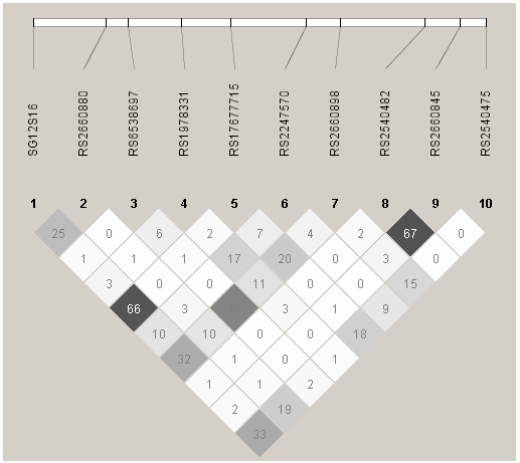
B. *LTA4H*



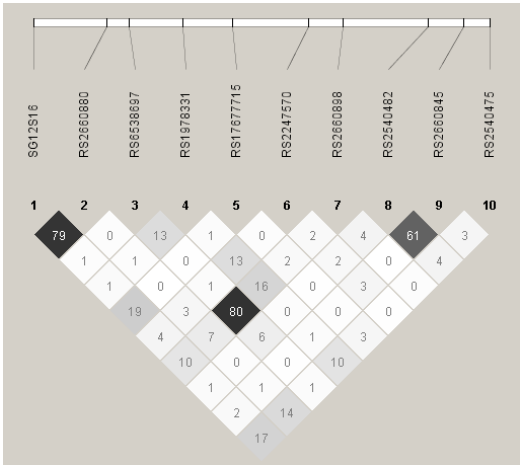
Caucasians CAD



Caucasians Normal

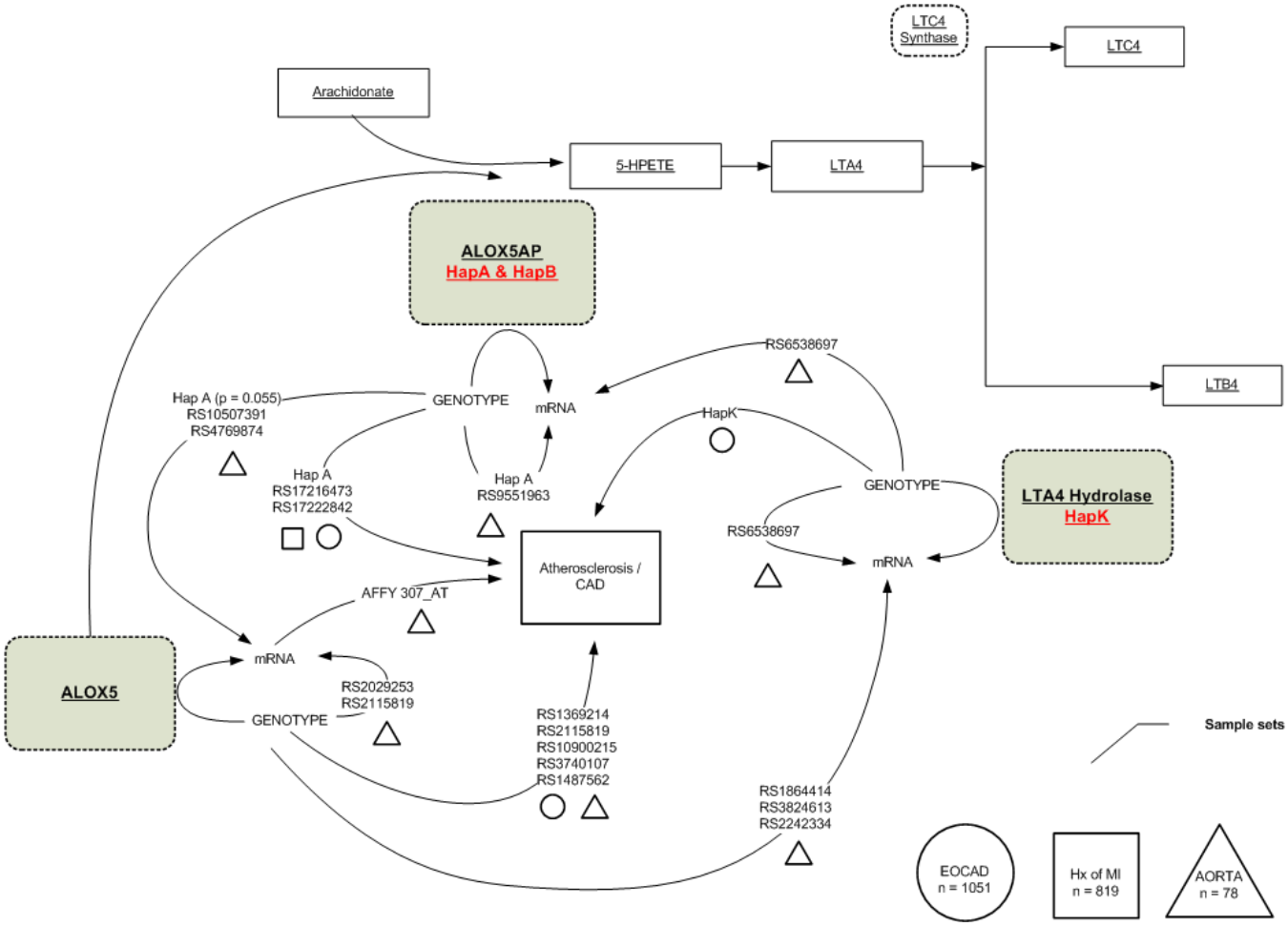


African-American CAD

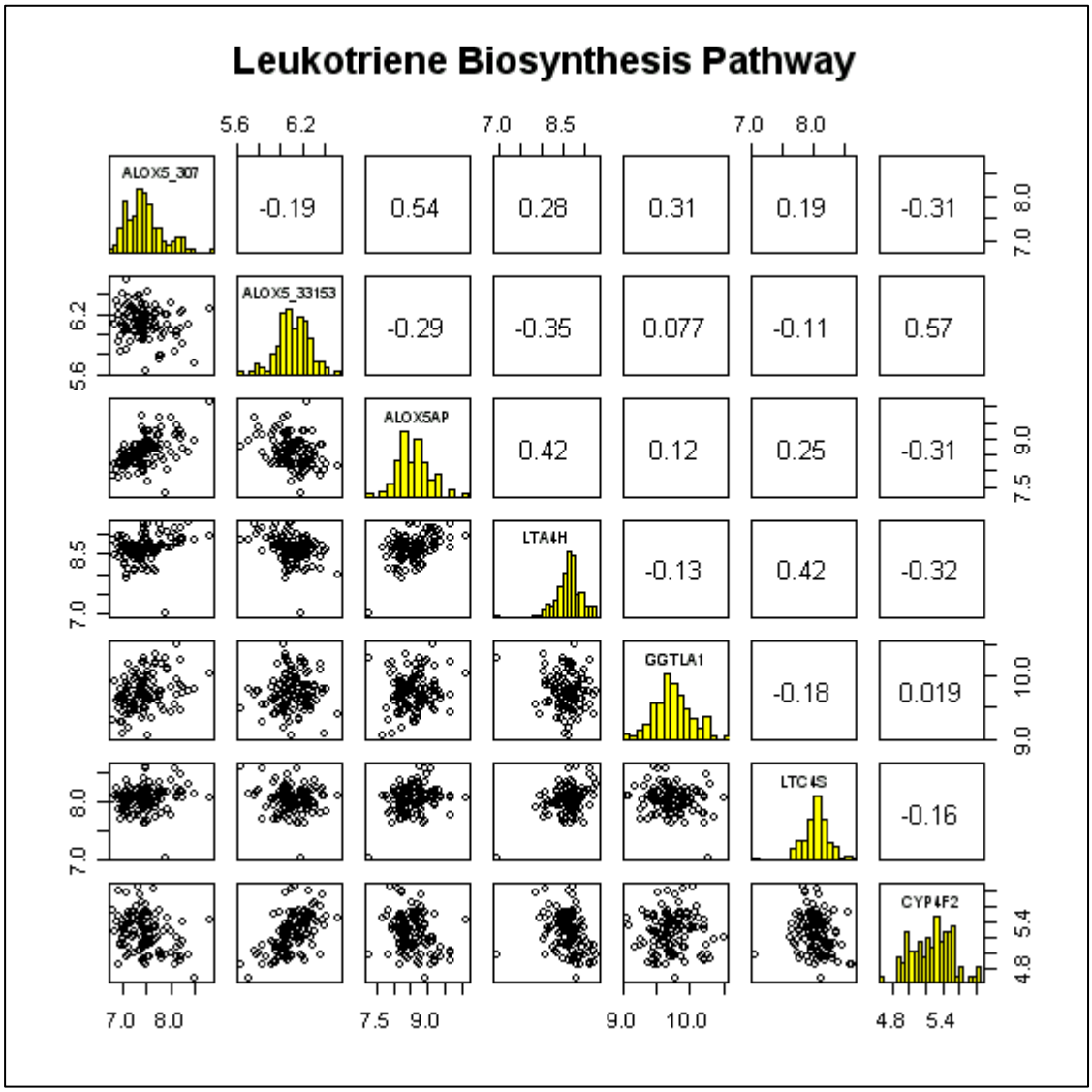


African-American Normal

Supplemental Figure 2B: *LTA4H* (including SNPs for HapK) linkage disequilibrium figures for CAD and controls by ethnicity in the CATHGEN sample



Supplemental Figure 3: Summary of *cis* and *trans* association findings in the leukotriene biosynthesis pathway for the CATHGEN subjects with EOCAD versus unaffected controls (○), subjects with myocardial infarction (MI) versus unaffected controls (□), and the AORTA case-control samples (raised lesion mapping and Sudan IV staining) (△); SNPs and haplotypes identified are significant ($p \leq 0.05$) in statistical models for CAD or transcript expression outcomes. See Table 2 for detailed results.



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