PIII-40: The -844 A>G - 4G/5G - HindIII haplotype at the PAI-1 locus is associated with variable levels of LDL among Caucasians and African American Women

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Abstract Categories:
Thematic Areas: Basic Science
Thematic Areas: Health Disparities

Abstract:
**Introduction:** We hypothesized 1) the PAI-1 genotype would affect LDL/total cholesterol levels, 2) PAI-1 levels would explain a significant proportion of the variance of LDL/total cholesterol, and 3) ethnic variation would exist.

**Method(s):** SNPs near 14.3 kb region in the PAI-1 gene were analyzed: -844A>G, 4G/5G and HindIII. LDL/total cholesterol were quantified. Sample: 129 women, 63 Caucasians & 66 African American (AA), ages 19-66. LDL/total cholesterol were analyzed using haplo.glm with three-SNP haplotype, and serum PAI-1 levels.

**Results:** For Caucasians, the mean LDL was 105.86 (s.d. 31.70), 25% were above the threshold of 130. In the AA, mean LDL was 91.875 (s.d. 26.64), about 5% were above 130. The means were significantly different (p = 0.008) using a one-sided t-test. LDL was analyzed separately by race. Age was not significantly different(p=0.4). Among the AA, haplotype A-4G-G is significant (p-value= 0.002) and associated with increased LDL. Serum PAI-1 level was borderline significant (p-value = 0.04). Among Caucasians, the haplotype is borderline significant with PAI-1 in the model, p=0.06 (without PAI-1 in the model, p=0.04), and associated with less LDL. Serum PAI-1 levels explains significantly more of the LDL variance among Caucasians, p-value = 0.00102. Haplotype A-4G-G also significantly affects the total cholesterol level among AA (p=0.00625). However, the same haplotype does not significantly affect the total cholesterol level in Caucasians. No haplotype significantly affected HDL levels.

**Discussion & Conclusions:** A significant association between the A-4G-G haplotype in PAI-1 and increased LDL among AA was found in this study, and a borderline significance with decreased LDL among Caucasians was found. Consistent with this possible reversal in effect of the haplotype on LDL across the two population samples, -844 A>G has a minor allele of G with frequency 0.4 in the HapMap CEU sample with (N=60), but a minor allele of A with a frequency of 0.25 in the HapMap Yoruban sample (N=60). It may be that a causal polymorphism for determining LDL/total cholesterol levels lies nearby this SNP. Increasing the size of the study and replication in other populations will help to elucidate the role of polymorphisms in this locus.

**Abstract History:**
This abstract has not been presented or accepted for presentation in whole or in part at the SNRS or other scientific meeting.

**Financial Disclosure:**
No, I (or a member of my immediate family) have not received something of value* from or own stock (or stock options) in a commercial company or institution related directly or indirectly to the subject of my presentation.
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I will not be describing any pharmaceutical and/or medical device.

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