CALIFORNIA STATE SCIENCE FAIR
2005 PROJECT SUMMARY

Name(s)
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Project Number
S0411

Project Title
Underlying Mechanisms of the Lewis Negative Phenotype's Relationship with Cardiovascular Disease

Abstract

Objectives/Goals
In the Copenhagen Male Study and NHLBI Family Heart Study, the Lewis blood group Le (a-b-) phenotype was found to put patients at an increased risk for cardiovascular diseases, but the underlying mechanisms were unclear. For this study, the hypothesis was proposed that the Le (a-b-) phenotype was associated with high red blood cell aggregation, and whole blood and plasma viscosity, which are known to be risk factors for ischemic heart disease.

Methods/Materials
The Lewis blood type of 87 subjects (50 males, 37 females) from an ongoing B-Vitamin Study at the USC Institute of Genetic Medicine was determined. Hemorheological parameters such as RBC aggregation, plasma viscosity, and whole blood viscosity were measured for each subject. Data regarding conventional risk factors and drug therapy were also collected.

Results
The incidence of Le (a-b-) phenotype was 17.2% of all subjects. Statistical analysis revealed a significant difference in plasma viscosity ($p=0.004$) and RBC aggregation ($p<0.0001$ and $p=0.0002$) between patients with Lewis (a-b-) and Lewis positive phenotypes. Mean values for blood pressure, total cholesterol, low-density lipoprotein and high-density lipoprotein, fasting glucose, and homocysteine were not significantly different between Lewis (a-b-) and Lewis positive subjects.

Conclusions/Discussion
The positive association between Lewis (a-b-) phenotype and various hemorheological parameters has not been reported previously. The results showed a significant difference in RBC aggregation (plasma M: $p<0.0001$; plasma M1: $p=0.0002$) and plasma viscosity ($p=0.0004$) between patients with the Lewis negative phenotype and Lewis positive subjects. Other conventional risk factors for cardiovascular disease were also tested, but they were not found to be significantly different between the Lewis phenotypes. There was no significant difference in the RBC aggregation in 3% dextran (dextran M: $p=0.19$ and dextran M1: $p=0.90$) between the Le (a-b-) phenotype and Lewis positive phenotypes. Therefore, it seemed that plasma proteins combined with RBC surface properties in Le (a-b-) patients were more important factors in RBC aggregation than the RBC surface properties alone.
The finding that the Lewis negative phenotype had a higher plasma viscosity and RBC aggregation may provide an explanation for why the Le (a-b-) phenotype is an independent risk factor for heart diseases.

Summary Statement
Investigating the underlying mechanisms of the Lewish Negative Phenotype and its relationship with cardiovascular diseases.

Help Received
Student-Researcher in the Edmondson Summer Fellowship Program at the Univ. of Southern California.