C-Reactive Protein and Cardiovascular Diseases in Andean Population

To the Editor:

We read with interest the study by Chambers et al. In 1025 healthy male subjects (518 Indian Asians and 507 European whites), they found a mean C-reactive protein (CRP) concentration higher in Indian Asians than in European whites, thereby accounting for a greater central obesity and insulin resistance in Indian Asians. These results suggest that inflammation or other mechanisms underlying elevated CRP may contribute to the increased coronary heart disease risk among Indian Asians.

In a cross-sectional population of Andean subjects that included a random sample of 300 participants aged 30 years or more, we found that the overall hypertension rate was 46%. The unadjusted prevalence of hypertension was 58.7% in the highest quartile of CRP but only 34.7% in the lowest quartile. After adjustment for age, sex, body mass index, family history of hypertension, fasting glycemia, sedentary behavior, and alcohol consumption, the prevalence of hypertension was 1.14 (95% confidence interval [CI], 0.82, 1.58; \( P = 0.4422 \)), 1.36 (95% CI, 0.99, 1.87; \( P = 0.057 \)), and 1.56 (95% CI, 1.14, 2.13; \( P = 0.005 \)) times higher in subjects in the second, third, and fourth quartiles of CRP, as compared with subjects in the first quartile, suggesting that CRP level may be an independent risk factor for the development of hypertension.2 In another cross-sectional study that included 72 high-risk Andean women, we found higher values of CRP in preeclampsia (mean 4.11 SEM 0.37 mg/dL) compared with normal pregnant women (mean 2.49 SEM 0.26 mg/dL) and non-pregnant controls (mean 1.33 SEM 0.15 mg/dL). Moreover, the levels of tumor necrosis factor-\( \alpha \) (TNF\( \alpha \)) and interleukin-6 (IL-6) were significantly higher in preeclamptic women. Furthermore, the levels of CRP, IL-6, and TNF\( \alpha \) detected were significantly higher than those reported in European and North American women and increased substantially during normal pregnancy and preeclampsia.3

Our results support the findings of Chambers et al1 regarding the association between ethnic origin, CRP concentrations, and risk of cardiovascular disease. However, whether the differences are related to genetic or environmental factors (eg, infection) remains speculative. A study of the effects of a recently described CRP polymorphism4 on CRP and cardiovascular diseases may be advisable. Whatever the underlying mechanism, the concentration of CRP and proinflammatory cytokines may have implications for vascular function and contribute to the epidemic of cardiovascular diseases observed in developing countries.5

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