Low-grade chronic inflammation (hs-CRP) correlates with degree of postprandial hyperglycemia in lean, young subjects

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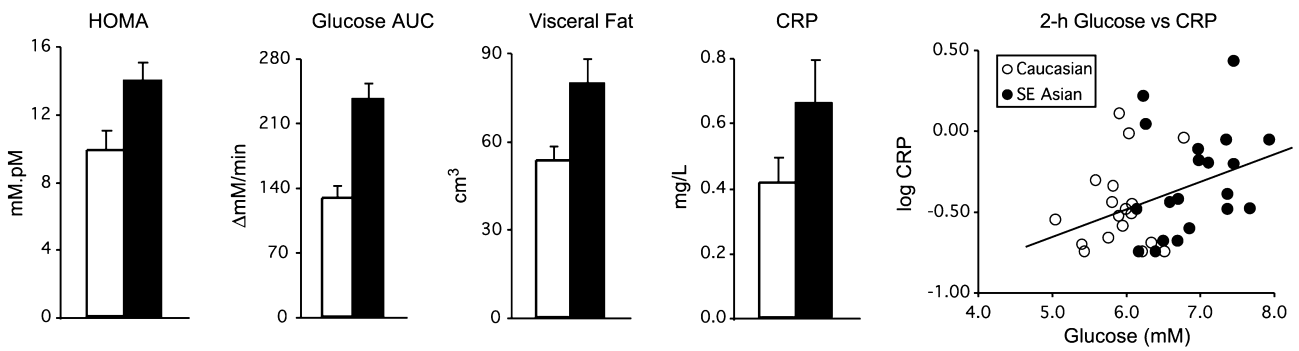
The prevalence of type 2 diabetes and coronary heart disease (CHD) is elevated among some ethnic groups, including people of Asian extraction. Insulin resistance resulting from both genetic and environmental causes is implicated in the pathogenesis of both diseases. Recent studies suggest that postprandial hyperglycemia is the earliest abnormality associated with type 2 diabetes and may also play a role in the development CHD. Postprandial glycemia increases oxidative stress and promotes low-grade chronic inflammation of the endothelium, a known predictor of CHD. A recognised marker of low-grade inflammation is the level of high sensitivity C-reactive protein (CRP) in the blood. Abdominal adipose tissue is a major determinant of hepatic CRP synthesis.

The present study was designed to detect differences in postprandial glycemia, insulin sensitivity, body fat, and blood CRP levels in 40 lean, young, healthy male subjects from two ethnic groups (20 European Caucasian and 20 South East Asian) who were matched for age, BMI, waist circumference, birth weight, current diet and physical activity. Our hypothesis was that the degree of postprandial hyperglycemia was more closely related to insulin sensitivity and CRP levels than body fat in both ethnic groups. Dual energy x-ray absorptiometry (DEXA) was used to determine total body fat, and ^{1}H-NMR imaging/spectroscopy (MRI/MRS) to measure levels of intra-abdominal visceral and intramyocellular fat (IMCL). Area under the plasma glucose curve (AUC) and 2-h glucose after a 75 g carbohydrate load (as bread) was used to assess the degree of postprandial hyperglycemia.



Despite comparable BMI and waist circumference, the SE Asian group had significantly higher body fat (P = 0.04), visceral fat (P = 0.02) and IMCL (P < 0.01) when compared to the Caucasians. Insulin resistance as determined by the homeostasis modelling assessment (HOMA) was also higher in the SE Asian compared with the Caucasian group (mean ± SEM: 13.9 ± 1.2 mM·pM vs 9.9 ± 1.1 mM·pM respectively). Pearson's correlation showed a positive association between IMCL and visceral fat, but not insulin sensitivity. CRP levels were significantly higher in the SE Asians (0.66 ± 0.1 mg/L vs 0.42 ± 0.08 mg/L, P = 0.05) and correlated most strongly with the degree of 2-h glycemia (P = 0.01), even after adjustment for body fat, visceral fat and IMCL.

The present study suggests that 1) postprandial hyperglycemia, insulinemia, insulin resistance and elevated CRP levels are common in young adults of SE Asian origin, 2) visceral and intramyocellular fat are higher at similar BMI and 3) chronic low-grade inflammation, as judged by CRP, may be a consequence of profound postprandial hyperglycemia rather than higher body fat. Minimising postprandial glycemia may be protective against vascular damage and the development of CHD in people of Asian origin.

Key words: postprandial hyperglycemia, inflammation, C-reactive protein