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Early features of systemic and adipose-tissue low-grade inflammation in a model of postprandial endothelial dysfunction

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During the postprandial state a low-grade inflammatory response may contribute to the adverse effects of high-saturated-fat diets and high-sucrose diets on vascular endothelial function, a hallmark in atherogenesis[1–3]. A rat model of endothelial dysfunction induced by a high-fat load has been characterized, in which the earliest feature was a marked increase in the plasma pro-inflammatory cytokine IL-6, as measured 2 h after the meal. Thus, the present study has investigated whether a high-fat load can induce at a very early stage (1) the expression of endothelial adhesion markers on blood leucocytes and (2) the nuclear translocation of NF-κB, a transcription factor known to activate target genes implicated in the inflammatory cascade including cytokine production and the expression of leucocyte-adhesion molecules.

Expt 1: using flow cytometry, the expression of CD11B, CD62L, CD45RA and CD3 on blood leucocytes was studied before and 2 h after an oral high-fat load (% total energy: saturated fat 60; sucrose 20; proteins 20) or a water load in ten healthy Wistar-kyoto rats. Expt 2: twelve rats were killed and visceral adipose tissue sampled 2 h after a high-fat or a water load and the activation of NF-κB was assessed from nuclear extracts.

The neutrophil count markedly increased 2 h after the high-fat load (by 33 (SE 14) % compared with 0 h; $P<0.05$), while remaining steady after the water load. The high-fat load decreased the B-cell count and the expression of the activation marker CD62L (*v.* water load; $P<0.01$). No significant changes were observed in monocyte and T-cell populations. In nuclear extracts of adipose tissue an increase in activated NF-κB was observed after the fat load (*v.* water load; $P<0.05$).

As expected, an increment in neutrophils characterized the postprandial state after a fat or sucrose load[4]. As far as is known, the present study is the first to show a meal-specific decrease in blood B-cells and an increase in activated NF-κB in adipose tissue after a fat load. These results suggest that B-cell recruitment and adipose tissue play early roles in the activation of the postprandial complex pro-atherogenic phenotype. Thus, this rat model provides relevant markers of early inflammatory responses associated with postprandial endothelial dysfunction that are useful in understanding the mechanisms of atherogenesis and studying their modulation by nutritional conditions.

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