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Koenig1999 (cited)

Rothe1996 (cited) & 1997 (not cited)

Schieffer2003 (not cited) Peter1997 (not cited) Zaldivar2006 (not cited) Tall2004 (not cited) Plenge2003 (not cited)

Atherosclerosis is clearly multifactorial and itis now universally recognised that inflammation within the lessions contributes importantly to their initiation and progression.

The immunologic response in atherosclerosis involves not only cells of the arterial wall, but also circulating lymphocytes and macrophages.

The amount of soluble ICAM-1 and E-selectin released has been demonstrated to be directly correlated with the surface expression of ICAM-1 and E-selectin in endothelial cells in culture.

CaliBRITE beads and FACSComp software were used for setting the photomultiplier tube voltages, the fluorescence compensation and for checking instrument sensitivity prior to use.

The cellular light-scatter signals and three fluorescence signals were analysed in list mode at a channel resolution of 1024, with forward scatter (FSC) as trigger parameter. The photomultiplier gains were calibrated with polychromatic fluorescent reference beads (Polysciences). Compensation was adjusted with FITC. R-PE and PerCP-coated microbeads (BD) and triple- stained (CD4, CD8 and CD3) peripheral blood lymphocytes as a biological control.
Cellular antigen densities were calculated with the assumption that there was only one cellular binding site for each mAb on its target antigen, on the basis of calibration with reference beads that carried a defined number of anti-mouse binding sites.

fasting venous blood was obtained after nontraumatic venipuncture and was allowed to clot at room temperature (RT) for 30 minutes. Serum was separated after centrifugation for 15 minutes at 3000 rpm and was stored at -70°C. In a blinded manner, ELISAs were used to determine serum concentration of sICAM. Standard curves based on six reference concentrations were created according to the manufacturer's recommendations.
For patient characteristics the Mann-Whitney test was performed for continuous data and Fisher's exact test for categorical data.

CRP could also reflect a state of arterial inflammation. A new idea in cardiology is that patients with acute coronary syndromes may have underlying, diffuse atherothrombosis of the coronary arteries, precipitated by the infiltration of atheroma by macrophages, the secretion of proteases, and the erosion or rupture of plaque. This inflammatory reaction, initiated in response to the retention of atherogenic lipoproteins in the artery wall, could also lead to the release of cytokines and production of CRP by hepatocytes.

Statins could reduce CRP by a number of mechanisms. One possibility is by reduction of inflammation within the artery, presumably by reducing the amount of LDL available for oxidative metabolism.
The administration of a statin in the setting of an acute coronary syndrome may have a role as powerful as that of early treatment with aspirin, a β-blocker or an ACE inhibitor. CRP-lowering effect of statins, independent of effects on other acute-phase reactants and lipids, could change the way physicians manage all patients at risk for CHD, both in the early setting and in the prevention of long-term events.