increasing vulnerability to lipoprotein thrombi in kidney. Further lowering of monocyte CD36 with statins would be of little benefit if CD36 is already low in CKD. Defining changes in lipid handling in CKD could lead to novel, targeted CVD treatment approaches in the CKD population.

10 CHOLESTEROL FLUX PATHWAY ABNORMALITIES INDUCED BY PLASMA FROM PATIENTS WITH CHRONIC KIDNEY DISEASE

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Purpose of Study Chronic kidney disease (CKD) is a known risk factor for cardiovascular disease (CVD). Patients with CKD have a high prevalence of atherosclerosis. However, CVD risk associated with CKD is not entirely explained by standard lipid profile or liver handling of cholesterol, as evidenced by the resistance to statin benefits seen in later stages of CKD. This study aims to detect changes in expression of cholesterol transport proteins in the setting of CKD and to determine if such changes adversely affect lipid handling by macrophages leading to cholesterol overload and atheromatous foam cell formation.

Methods Used THP-1 human macrophages (10^6 /ml) were incubated for 18 h–24 h with plasma obtained from 10 CKD patients (7 male, 3 female) or 10 healthy control subjects (4 male, 6 female). CKD patients were not on dialysis and had not received renal transplant. Following incubation, mRNA was isolated and reverse transcribed. The resulting cDNA was subjected to quantitative real-time PCR using specific primers for ATP binding cassette transporter (ABC)A1 (cholesterol efflux protein) and CD36, (a scavenger receptor with the capacity to endocytose oxidized LDL).

Summary of Results PCR analysis showed that ABCA1 mRNA was reduced by $23 \pm 5\%$ ($p < 0.0001$) while CD36 mRNA was decreased by $36 \pm 7\%$ ($p < 0.0001$) in macrophages exposed to CKD plasma as compared to healthy control.

Conclusions These findings suggest a different mechanism of lipid dysregulation associated with CKD that may explain the pathogenesis of elevated CVD risk in CKD and lack of response to statins. This mechanism, through pro-atherogenic suppression of ABCA1, differs from our finding in autoimmune rheumatic diseases where, in addition to lowering of ABCA1, augmentation of CD36 was also observed. In CKD, a paradoxical decrease in CD36 could compromise macrophage clearance of lipids,