

P008 Postprandial leukocyte activation: a mechanism for atherosclerosis and inflammation by triglyceride rich lipoproteins
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Objective: Activation of leukocytes is obligatory for atherogenesis and is mediated by triglyceride rich lipoproteins (TRLs).

Methods: We investigated CD11B and CD66B expression (by FACS) after incubation with native and artificial TRLs (NTRL and ATRL) with and without DMTU. NTRL leukocyte uptake was quantitated in postprandial samples.

Results: 15 minutes incubation with 0.35 mM of NTRLs increased PMN CD11B by 1.6 times, but not CD66B. We found a dose-dependent CD11B on monocytes after 0.16, 0.35 and 0.6 mM NTRLs. ATRL 10 and 15 mM showed an increase of monocyte and PMN CD11B. DMTU blunted only the PMN CD66B compared to ATRL alone by 36%. Postprandial studies showed a concentration of ~2 μ M TG per liter in leukocytes, which became enriched postprandially with meal-derived fatty acids ([1-¹³C]16:0). Sudan black stained blood smears demonstrated intracellular fat in PMNs.

Conclusions: Oxidative stress by TRL's may induce PMN CD66B expression. Total transport capacity for TRLs is limited but this study suggests leukocyte activation possibly by interaction in the bloodstream supporting the concept that postprandial lipemia is a pro-atherogenic situation.