Atherosclerosis is an inflammatory disease. Macrophages actively participate to the atherogenesis, either by accumulating lipids and by the secretion of several cytokines. Nowadays, despite there is a strong evidence that post-prandial Triacylglycerol Rich Lipoproteins (ppTGRL) are atherogenic and induce macrophage lipid accumulation, little is known on the role of ppTGRL on inflammatory macrophage functions. With this study we aimed to investigate whether ppTGRL influenced chemokines macrophage secretion.

ppTGRL were isolated by sequential ultracentrifugation from plasma of normolipidemic subjects 150min after the consumption of a standard breakfast (energy intake 1,050 kcal, 63% fat). Human Monocyte-Derived Macrophages (HMDM) were incubated for 24hours either with 10 and 30 µg/ml cholesterol carried by ppTGRL or an equivalent amount of cholesterol transported by Fetal Bovine Serum (FBS). Control incubations were performed without lipids (w/o FBS), and for positive control, HMDM were stimulated with 1µg/ml LPS. The supernatants were used for the determination of MIP-1alpha, MIP-1beta, IL8, TARC, RANTES, EOTAXIN and MDC by SearchLight™ Proteome Arrays.

LPS stimulated the secretion of analysed chemokines. We found that both concentrations of ppTGRL induced a significant and similar increase (n=3, p<0.05) in the secretion of the main macrophage effectors chemokines. MIP-1alpha (195±100 and 156±89 pg/ml) and MIP-1beta (1282±662 and 1112±571 pg/ml) were increased by about 3 and 5 fold with respect to HMDM incubated w/o FBS (53±16 and 240±61 pg/ml for MIP-1alpha and MIP-1beta, respectively). The IL8 secretion (1100±187 and 1139±37 pg/ml with 10 and 30 µg ppTGRL /ml, respectively), a strong chemoattractant for monocytes as well as for leukocytes, was also increased (n=3, p<0.05) in comparison to HMDM incubated w/o FBS (667±113 pg/ml). RANTES, EOTAXIN and MDC secretion was not affected, while ppTGRL significantly reduced in a dose-dependent manner the release of TARC, a platelet activator which lacks the capability to recruit monocytes. These effects were specifically induced by ppTGRL but not from FBS-lipids. Present results suggest that postprandial phase is associated with an increased production of monocyte/macrophages chemoattractant factors, which could be related to the higher cardiovascular risk found in diseases with a delayed clearance of ppTGRL, such as diabetes, obesity and chronic renal failure.