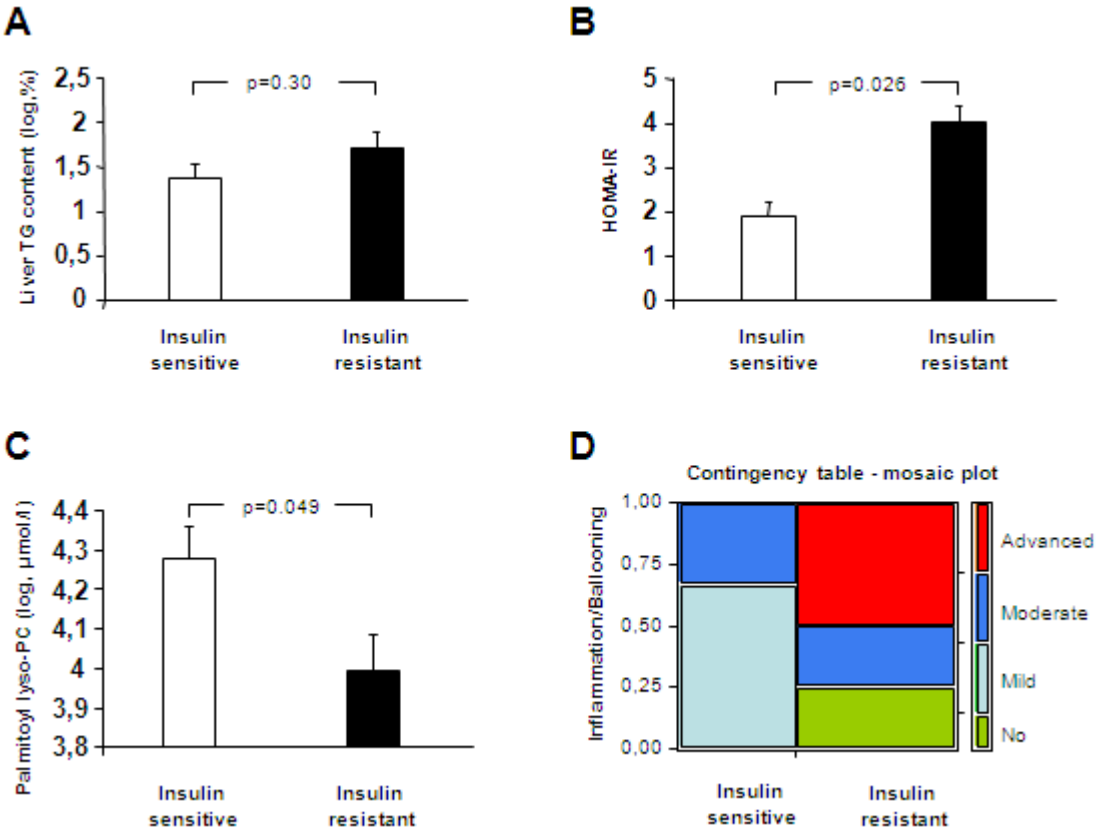
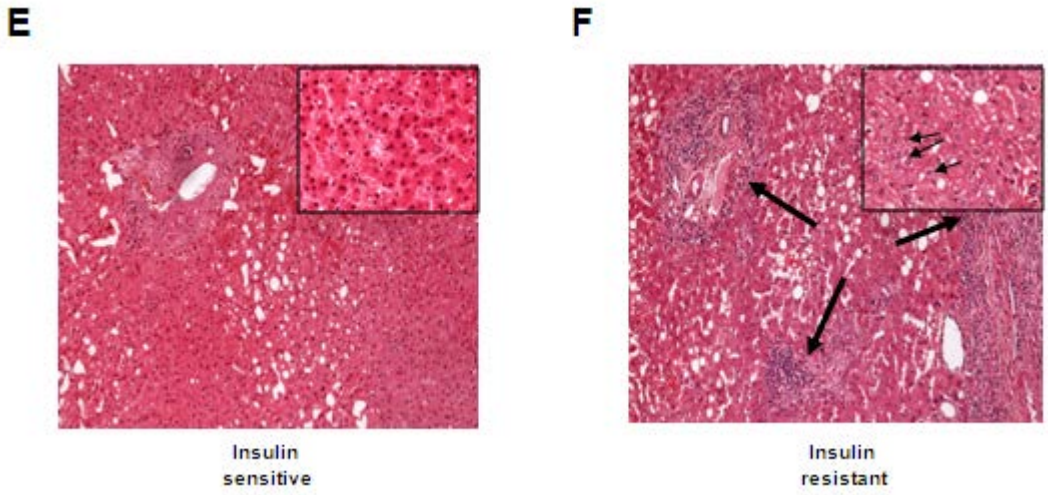
SUPPLEMENTARY DATA

Supplementary Figure 1. Relationships of liver triglyceride (TG) content (A), insulin sensitivity (HOMA-IR) (B), palmitoyl lyso-PC (C) and liver histological parameters (D-F) in subjects in the upper quartile (N=7) of liver TG content (all N=29), who were divided by the median HOMA-IR.Panel D: p=0.08 ($\chi 2$ likelihood ratio) for having a cumulative value of mild, moderate, and advanced hepatic inflammation and ballooning. Mallory-Hyaline was not found in these subjects. Panel E: Liver histology of a patient representative for the insulin sensitive group (N=3). No inflammatory infiltrates are seen (original magnification 100x H&E). The hepatocytes show no ballooning and no Mallory-Hyaline (insert; original magnification 400x H&E). Panel F: Liver histology of a patient representative for the insulin resistant group (N=4). A mild lymphoid infiltrate, accentuated in the periportal tracts, but also in the lobules is shown (arrows; original magnification 100x H&E). Focally a cytoplasmic ballooning of hepatocytes is detected (insert; arrows; original magnification 400x H&E).



SUPPLEMENTARY DATA



SUPPLEMENTARY DATA

Supplementary Figure 2. Schematic working models of the potential role of lyso-PCs in NAFL-induced insulin resistance. Lyso-PCs may inhibit hepatic inflammation by regulating the function of naturally occurring CD4(+)CD25(+) regulatory T cells (Tregs) and, thereby, protect from insulin resistance (model 1). Alternatively, elevated circulating lyso-PCs may represent activated pathways in the synthesis or the metabolism of phospholipids and phosphatidylcholine and thus, in the generation of hepatic endoplasmatic reticulum stress (model2).