GW26-e5047
Postprandial Triglyceride-Rich Induced Atpogenesis Differentiation Is Dependent on Apolipoprotein E Carried on Lipoprotein
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OBJECTIVES
Postprandial hypertriglyceridemia is closely related with obesity. Postprandially increased triglyceride-rich lipoproteins (TRLs) took an important role in adipocyte hyperplasia. The aim of this study was to investigate the effects of apolipoprotein E (ApoE) carried on postprandial TRLs on adipogenesis and the potential mechanisms underlying this.

METHODS
Postprandial TRLs were isolated by density gradient ultracentrifugation from plasma in patients or mice at 4h after a high-fat meal. T37-L1 cells were cultured in various concentrations of human TRLs (h-TRLs) (25, 50, 100, 150 g/mL) in the presence or absence of 10 g/µL insulin for two weeks, or with both 100 µg/mL h-TRLs and 10 g/µL insulin for various days (0, 4, 7, 10, 14 days). To explore the effect of TRL-bound apoE in TRL-induced adipogenesis, T37-L1 cells were incubated with 100 µg/mL TRLs from wild type mice (WT-TRLs) or apoE knock-out mice (EKO-TRLs) and 10µg/mL insulin for 14 days. Oil-red O staining and expressions of adipogenesis markers were detected. Differentiating adipocytes were incubated with different kinds of TRLs labeled by Atto-555-NHS that emits red fluorescence. Then laser confocal microscopy was performed to determine the locations of TRLs to further investigate the effect of TRL-bound apoE on endocytosis of TRLs by differentiating adipocytes. Immunofluorescence and confocal microscopy were adopted to prevent the interaction between TRLs and LDLR family receptors, heparan sulfate proteoglycan (HSPG) or both, respectively, to investigate the receptor-mediated pathway in the endocytosis of TRLs. Real-time PCR and western blot were used to detect the expressions of endocytic receptors associated with apoE during TRLs-induced adipogenesis.

RESULTS
h-TRLs with insulin (10 g/µL) successfully induced T37-L1 to form mature adipocytes. Both protein and mRNA expressions of adipocyte fatty acid binding protein 2 (aP2) and peroxisome proliferator activated receptor y (PPAR-y) increased not only along time increase of TRLs concentration (P<0.05), but also with the treatment time of 100µg/mL TRLs (P<0.05). With the assistance of insulin, WT-TRLs induced 3T3-L1 to produce lipid droplets, whereas EKO-TRLs did not. Confocal microscopy analysis clearly revealed that red fluorescence could be seen within the differentiating adipocytes treated with h-TRLs or WT-TRLs, but not in those with EKO-TRLs. Compared with control group, RAP markedly reduced red fluorescence within the differentiating adipocytes, while heparin had little impact. The protein level of LRPs showed upward trend with the increase of TRLs concentrations. Compared with undifferentiated preadipocytes, the strong expression of LRPI protein was detected throughout 14 days.

CONCLUSIONS
Postprandial TRLs with insulin induced adipogenesis differentiation in dose- and time-dependent manner. Lipoprotein-bound apoE was required in TRLs-induced adipogenesis and the eventual yields of TRLs by the differentiating adipocytes that internalized TRLs via LDLR family members, probably LRPI.

GW26-e0383
Effects of PKC Activity on the Adhesion Reaction of Atherosclerosis
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OBJECTIVES
To investigate the effects of protein kinase C (PKC) activity on the adhesion reaction of atherosclerosis

METHODS
The present study consisted of an in vivo investigation and four in vitro investigations. In the in vivo investigation, 24 New Zealand rabbits were induced into atherosclerosis by the administration of high cholesterol diet for 12 weeks. The changes of PKC activity in atherogenesis were measured by the non-radioactive Detection and the distribution of PKCs in plaques was detected by immunohistochemistry staining. In in vitro experiments, PKC activity in modified low density lipoprotein (LDL)- loaded cells (including HASMC smooth muscle cells, THP-1 monocytes/macrophages and HAC endothelial cells which play a key role in atherogenesis) were detected. Furthermore, the mechanisms of PKC, ICAM-1, IκBα and ezrin in the adhesion reaction of endothelial cells with monocytes were explored.

RESULTS
PKC activity rose significantly in the atherosclerotic aorta of New Zealand Rabbits which were fed by high cholesterol diet for