Contribution of Dietary Fat to HDL-Triglycerides

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Background
Low levels of high-density lipoprotein cholesterol (HDLc) are thought to increase the risk for heart disease. Low HDLc in obesity is thought to result from greater flux of blood triglycerides (TG), particularly after meals, which stimulate lipid transfer mediated by the enzyme cholesterol ester transfer protein (CETP). CETP exchanges core lipids between TG-rich lipoproteins (TRL) and HDL, resulting in HDL that are enriched with TG. Evidence indicates that TG-rich HDL clear more quickly from plasma resulting in lower HDLc concentrations in both the fed and fasted states. This scenario of events, i.e., hypertriglyceridemia postprandially, elevated CETP activity, and the resulting low HDLc concentrations, is supported by strong in vitro data. However, the direct transfer of dietary-TG from TG-rich lipoproteins to HDL has not been measured in vivo in humans.

Objective
The goal of this study was to utilize an in vivo method to directly quantify the transfer of dietary TG from TRL to HDL during a period of nocturnal hypertriglyceridemia (midnight to 7:00 am). We hypothesized that the TG content of HDL will positively correlate with that in TRL and that the proportion of HDL-TG from the diet will positively correlate with the proportion of TRL-TG from dietary sources.

Methods
Previously, eight obese and hypertriglyceridemic subjects were fed a meal containing [3H]glyceryl-tripalmitate to track the contribution of dietary fat to lipoprotein TG. Blood sample were collected from midnight (3h post meal) to 7:00 AM and TRL-TG concentrations ([TG]) and the contribution of dietary fat to TRL-TG were determined. For the present study, HDL was isolated by ultracentrifugation, lipids extracted using the Folch extraction method, and TG isolated by thin layer chromatography. Total HDL-TG was quantified using an enzymatic assay, and the contribution of dietary fat to HDL-TG using gas chromatography/mass spectrometry. Pearson’s correlations and one-way ANOVAs were conducted in SPSS version 21.

Results

Total HDL-TG
Unexpectedly, a negative but non-significant correlation resulted between the average HDL-[TG] and TRL-[TG] (R = -0.54, P = 0.21). The proportion of HDL- and TRL-[TG] from diet decreased as time from the meal increased (P = 0.042 and P < 0.001). The proportion of HDL- and TRL-[TG] from diet decreased as time from the meal increased (P = 0.042 and P < 0.001).

Conclusions
• It was hypothesized that TG from a meal would increase in TRL and drive an increase of TG in HDL. However, a change in total HDL-[TG] over the 7 hour period was not observed, and there was no correlation between total TRL- and HDL-[TG].
• It is possible that the capacity for HDL to accept TG is saturated in these hypertriglyceridemic participants who were fed and infused with fats for 4 days prior to the collection of this data.
• However, the proportion of dietary fat in TRL and HDL did positively correlate, confirming that TG is being transferred from TRL to HDL over this period and being cleared at relatively the same rate.
• These results are the first to show that dietary fat is transferred to HDL from TRL in vivo, but suggest that in an overnight period in hypertriglyceridemic participants it does not lead to an increase in HDL-[TG] and may not contribute to decreased HDLc concentrations.

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References