

The Ludwigshafen Risk and Cardiovascular Health Study: LDL-TG

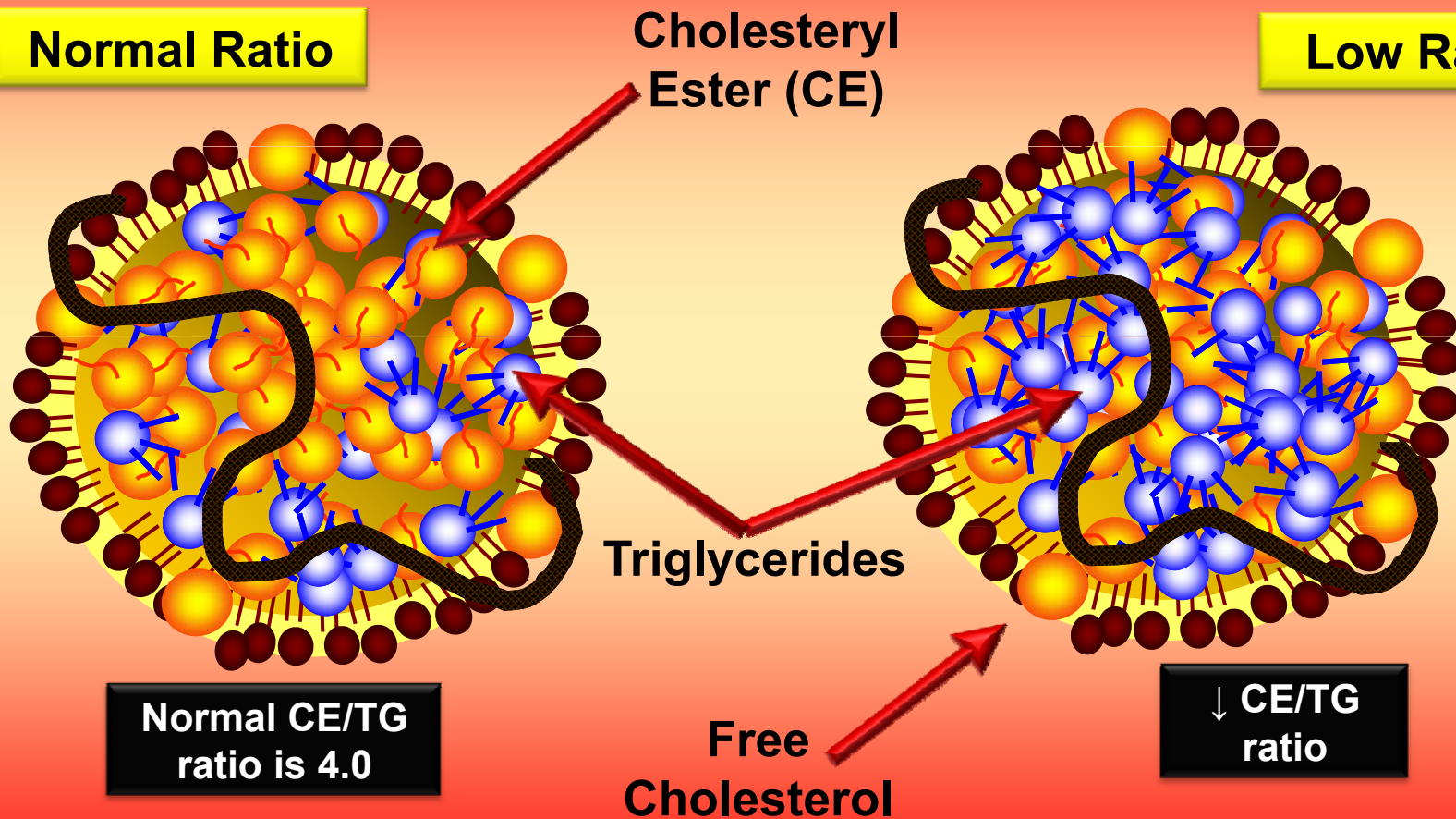
- ▶ 739 subjects with stable angiographic CAD and 570 matched control subjects in which CAD had been ruled out by angiography and not taking lipid medication
- ▶ The association of **LDL triglycerides** (LDL-TGs) (odds ratio [OR], 1.30; 95% CI, 1.19 to 1.43; $P < 0.001$) with CAD was stronger than that of LDL-C (OR, 1.10; 95% CI, 1.00 to 1.21; $P = 0.047$).
- ▶ In subjects with high LDL-TG, LDLs were depleted of cholesteryl esters (CEs), and VLDLs, IDLs, and dense LDLs were significantly elevated.
- ▶ The predictive value of LDL-TG for CAD was independent of LDL-C.

LDL Particle Composition

LDL Particle CE/TG composition differences

Normal Ratio

Low Ratio



Normal CE/TG
ratio is 4.0

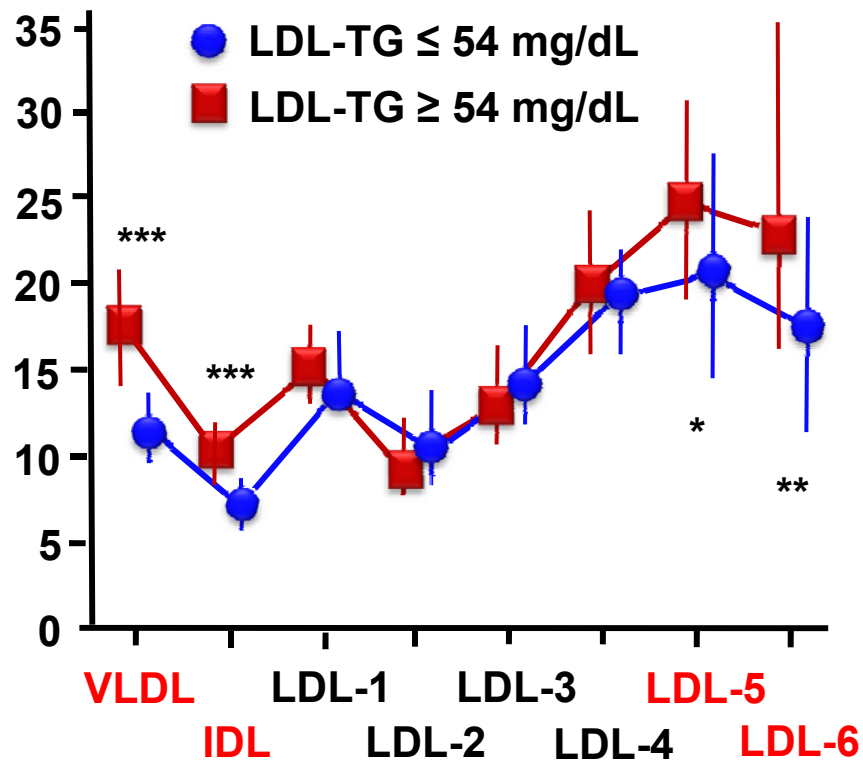
↓ CE/TG
ratio

Free
Cholesterol

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LDL-C & LDL-TG and ApoB

Apolipoprotein B (mg/dL)



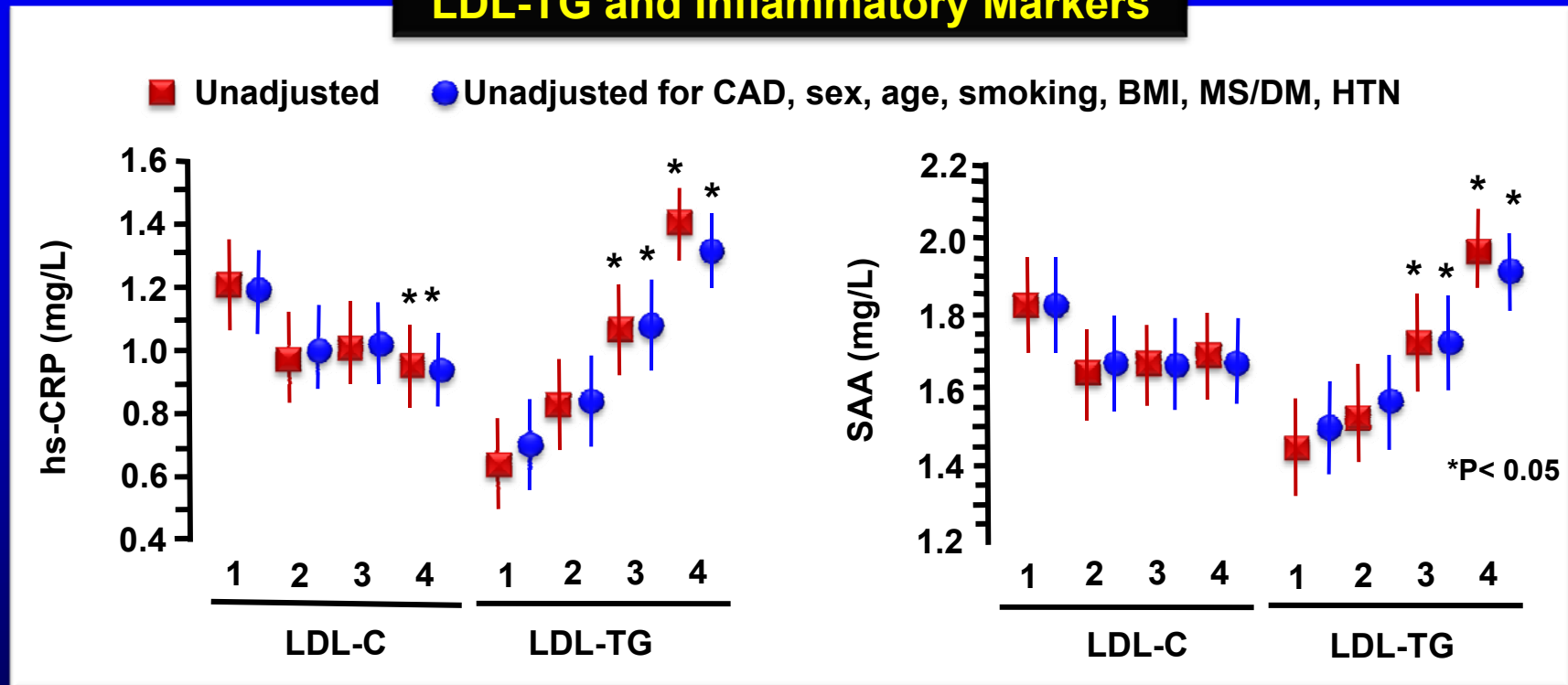
Median (25th and 75th percentile) concentrations of apoB in lipoprotein subfractions in 114 patients with elevated fasting glucose, impaired glucose tolerance, or T2DM having LDL-TG ≤ 54.4 mg/dL (n57, circles) or LDL-TG ≥ 54.4 mg/dL (n57, squares). *P0.05 (Mann-Whitney U test).

Subjects with LDL-TGs above the median had significantly higher concentrations of VLDL, IDL, and dense LDL (LDL-5 and LDL-6).

In contrast, buoyant LDL (LDL-1 and LDL-2) and intermediate LDL (LDL-3 and LDL-4) were not different

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LDL-TG and Inflammatory Markers

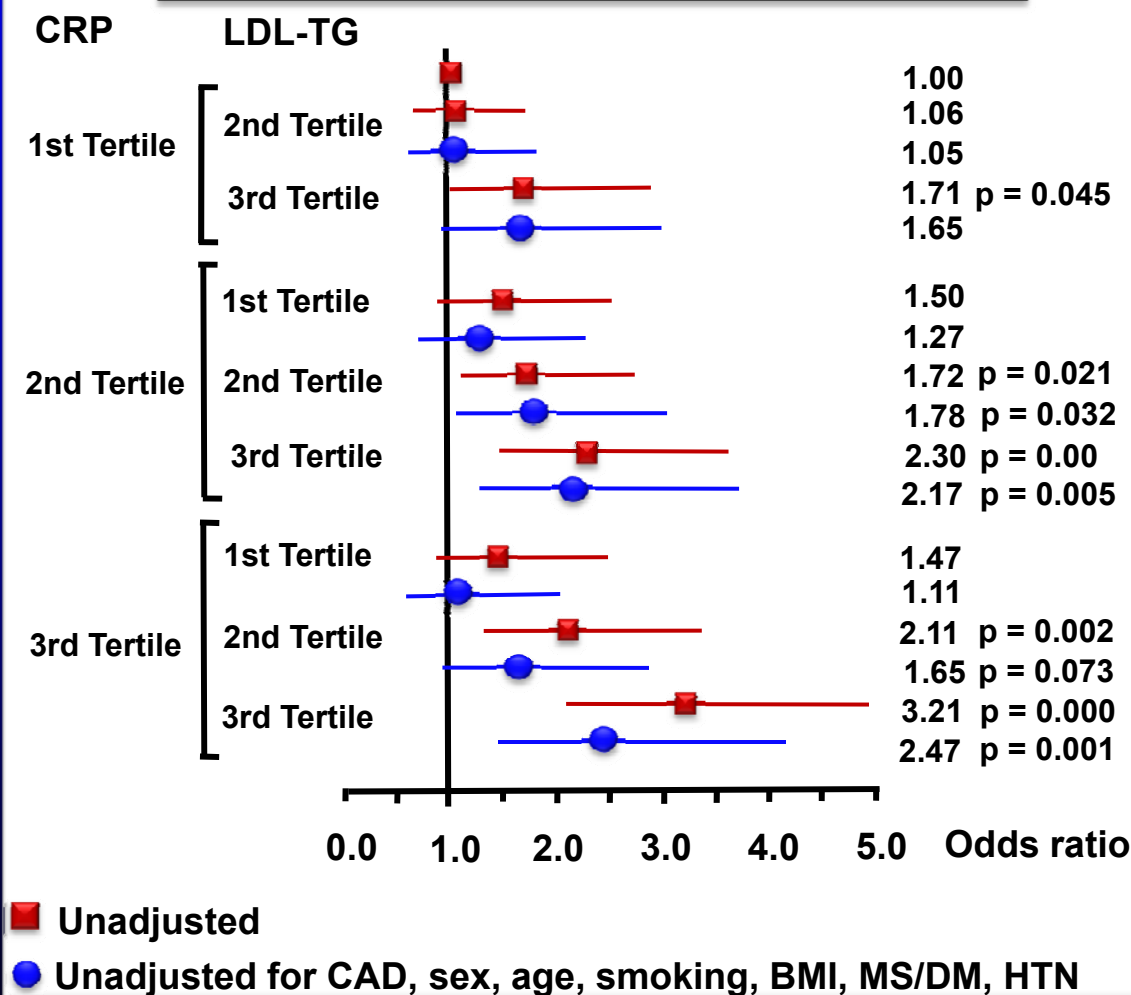


Estimated marginal means (95% CI) of concentrations of CRP & SAA by quartiles of LDL-C and LDL-TG.

CRP, SAA, fibrinogen, and IL-6 increased in parallel to LDL-TG. This relationship was independent of conventional coronary risk factors. In contrast, SAA, fibrinogen, and IL-6 were not different across the quartiles of LDL-C, and CRP was significantly lower even in the highest quartile of LDL-C compared with the lowest quartile

The Ludwigshafen Risk and Cardiovascular Health Study: LDL-TG

LDL-TG and Inflammatory Markers



ORs (95% CI) for angiographic CAD by sensitive CRP and LDL-TG. CRP and LDL-TG were broken down into tertiles, and ORs were calculated for each resulting layer in reference to group with both CRP and LDL-TG in their lowest tertiles.

These findings suggest that **LDL-TG**, although positively related to systemic markers of inflammation, is an **independent risk factor of CAD**.

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- ▶ Alterations of LDL metabolism characterized by high LDL-TG are related to CAD, systemic low-grade inflammation, and vascular damage.
- ▶ High LDL-TGs are indicative of CE-depleted LDL, elevated IDL, and dense LDL.
- ▶ **LDL-TG may better reflect the atherogenic potential of LDL than LDL-C.**

The Ludwigshafen Risk and Cardiovascular Health Study: LDL-TG

In conclusion, we show that disorders of LDL metabolism characterized by elevated LDL-TG rather than by elevated LDL-C may incite or promote vascular inflammation.

The determination of LDL-TG would thus add to LDL-C in predicting CAD.