

**Adipose tissue PC remodeling influences insulin sensitivity**

Lysophosphatidylcholine acyltransferase 3 (LPCAT3) is the major isoform of phosphatidylcholine remodeling enzyme in adipose tissues. To study the impact of LPCAT3 on adipose tissue lipid metabolism and its metabolic consequences, we prepared adipocyte-specific *Lpcat3* gene knockout (KO) mice using the Cre-Loxp approach. We found LPCAT3 deficiency caused a significant reduction of LPCAT3 mRNA and activity by about 60%, and a significant reduction of polyunsaturated phosphatidylcholines in the plasma membrane. Further, the deficiency increased insulin sensitivity, reflected by glucose and insulin tolerance studies. Importantly, LPCAT3 deficiency promoted insulin-dependent insulin receptor kinase tyrosine phosphorylation and Akt phosphorylation through influencing membrane lipid rafts. Furthermore, the deficiency protected mice from high-fat, high-caloric diet-induced insulin resistance. Taken together, our findings identify LPCAT3 as one of the major players in maintaining cell membrane structure and illustrate that manipulation of cell membrane phospholipid saturation can be a new strategy to treat insulin resistance.