

# Multiomics Analyses Reveal that PRMT5 Regulates Membrane Transport and Cholesterol Synthesis in White Adipocytes.



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Xiyue Chen, Zhihao Jia, Xiashiyao Zhang, Feng Yue, James F Markworth, Christina R Ferreira, Jun Wan, et al. 2025.  
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*iMetaOmics* 2: e70054. <https://doi.org/10.1002/imo2.70055>



# Transcriptional profiling of white adipose tissue (WAT)

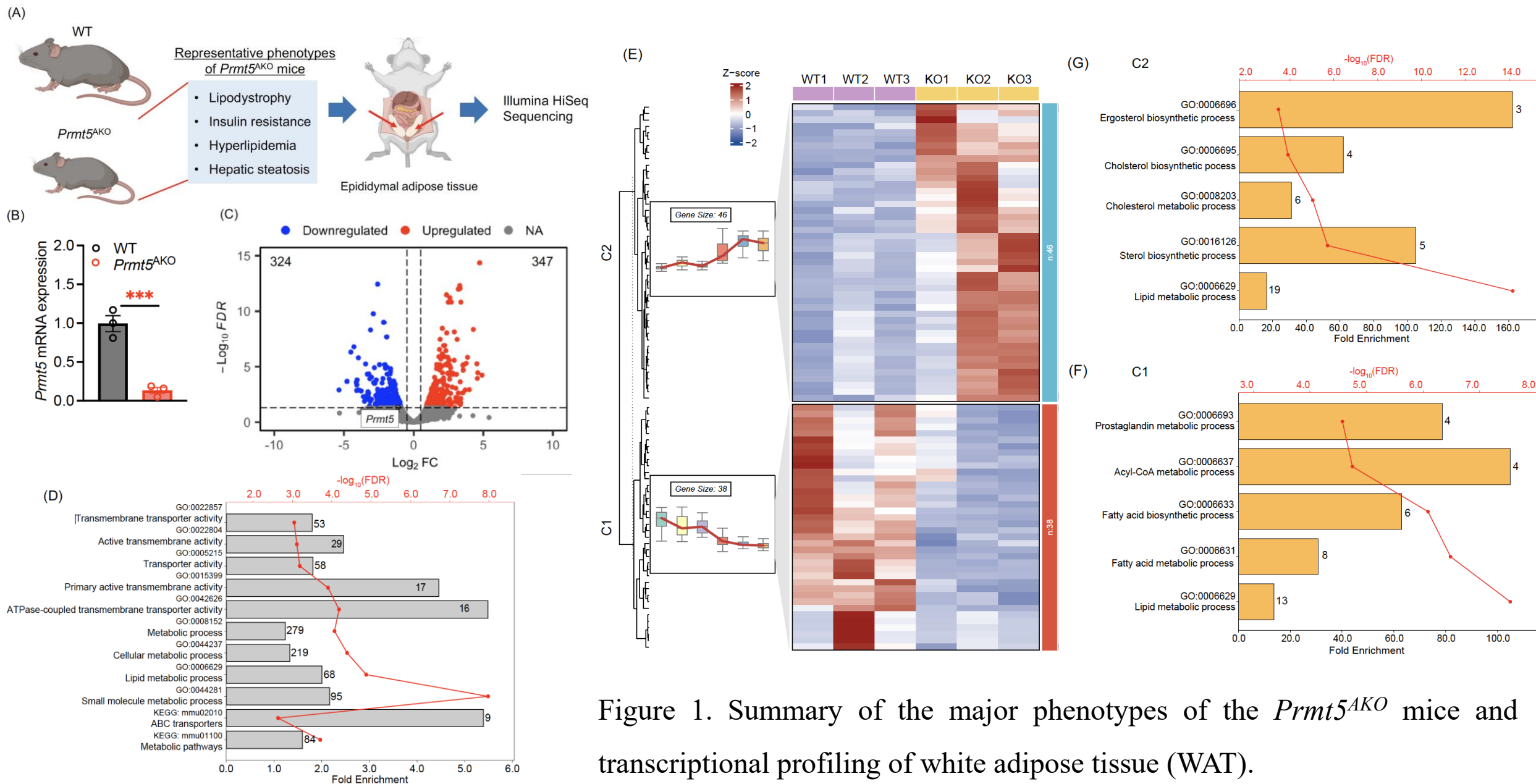


Figure 1. Summary of the major phenotypes of the *Prmt5*<sup>AKO</sup> mice and transcriptional profiling of white adipose tissue (WAT).



# Loss of *Prmt5* alters the metabolic performance of mice

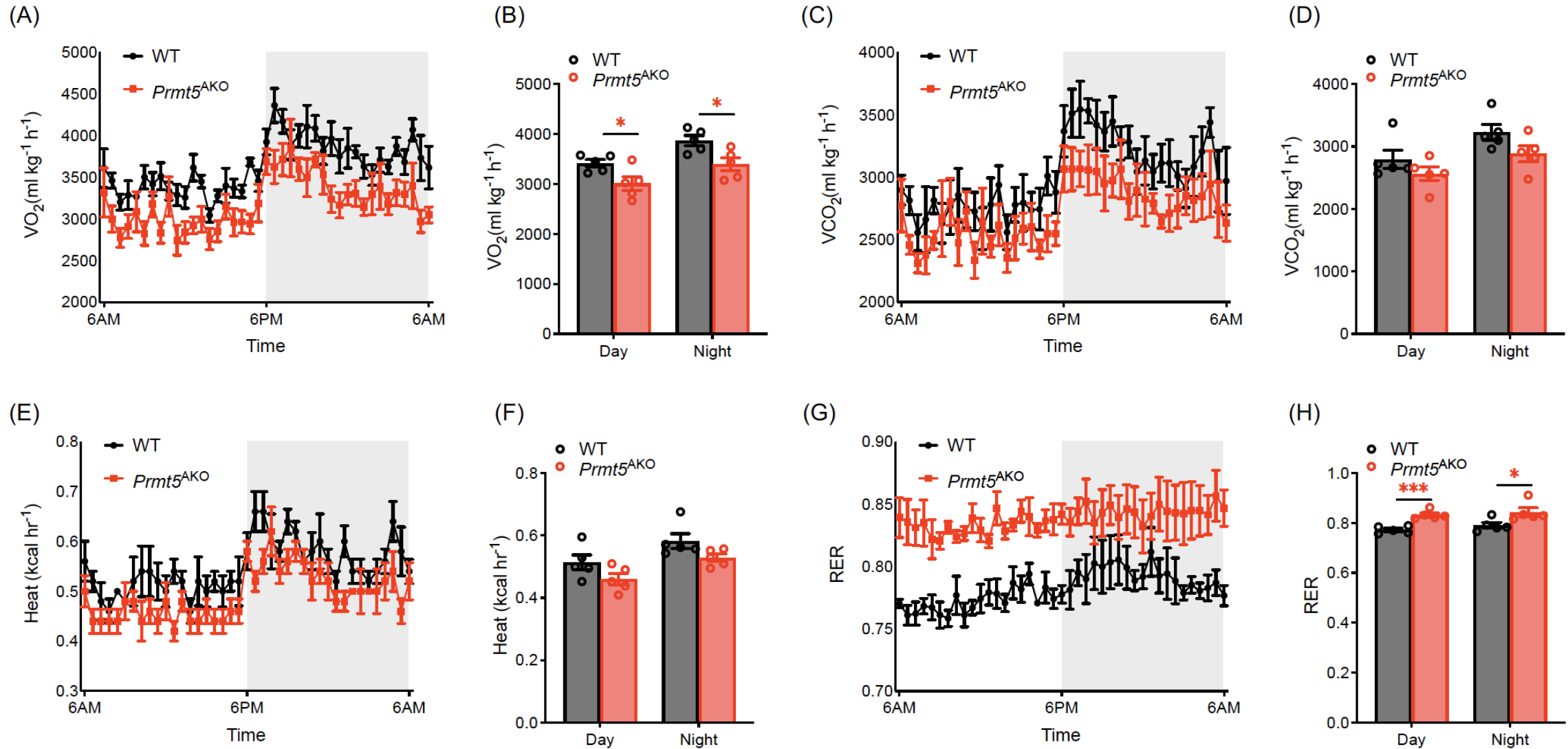
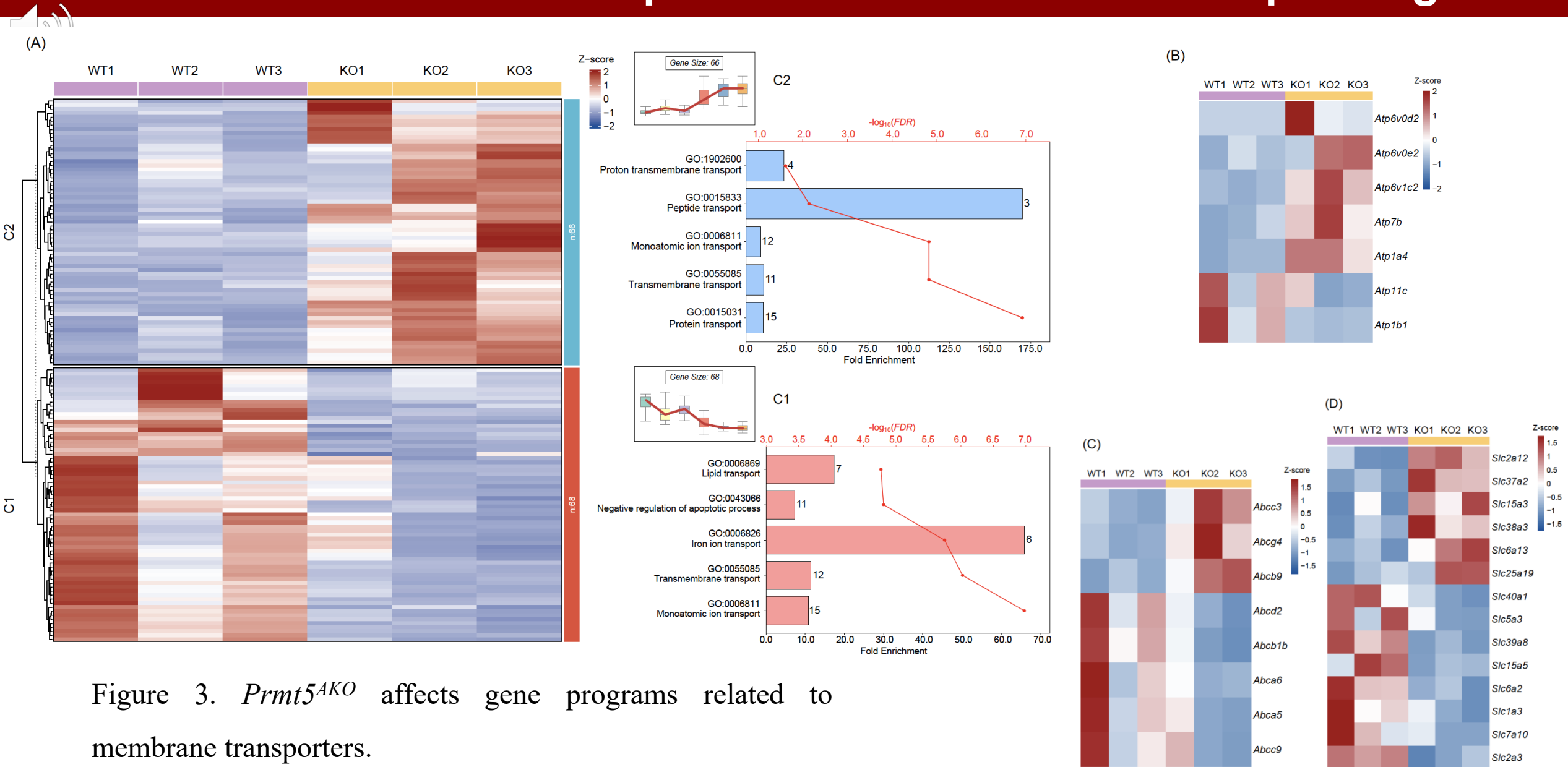


Figure 2. *Prmt5*<sup>AKO</sup> predominantly alters gene programs involved in metabolic pathways.

# *Prmt5* deletion alters the expression of membrane transporter genes



# Prmt5 deletion reduces lipid transport and increases cholesterol and glucose transport

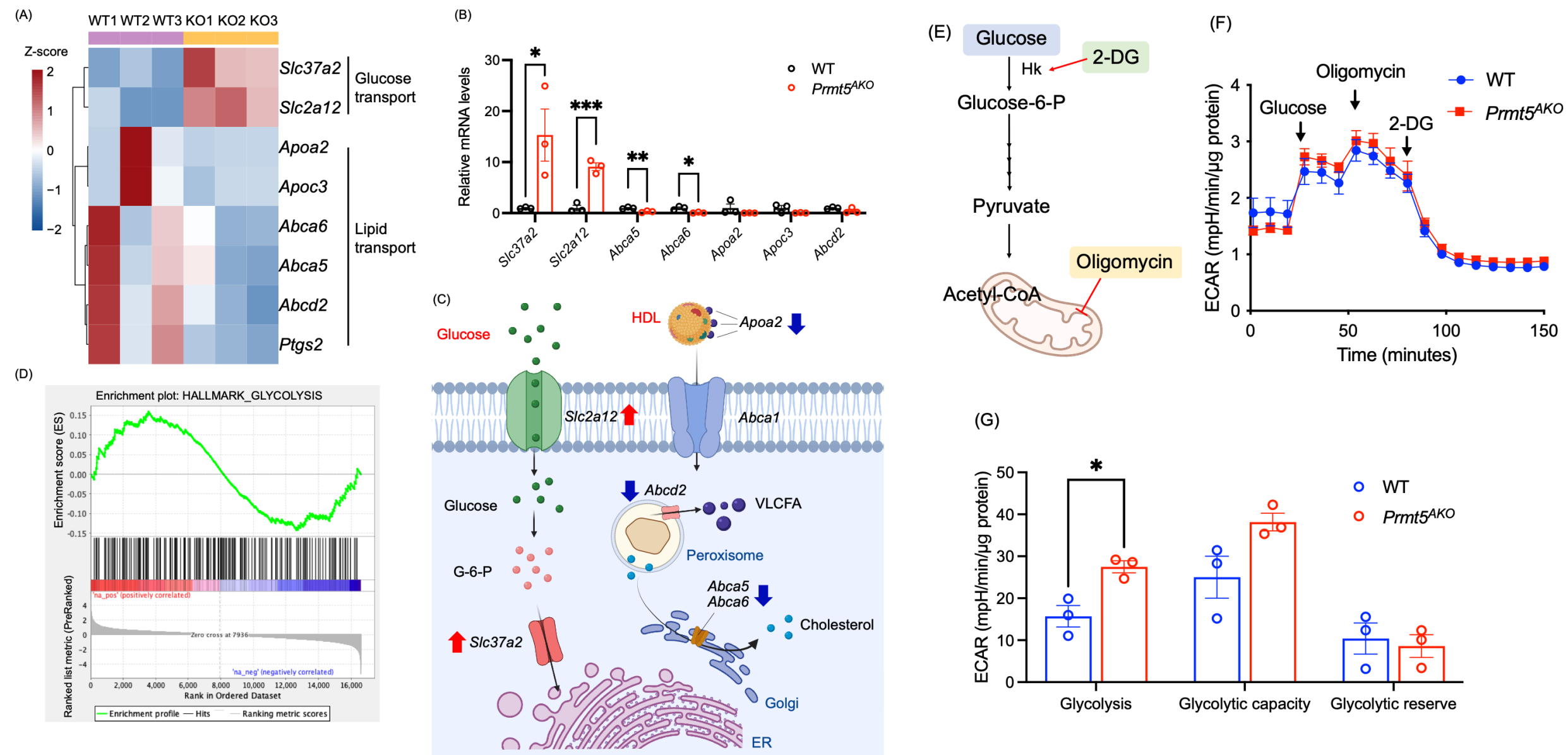


Figure 4. *Prmt5*<sup>AKO</sup> suppresses fatty acid transport, while promoting glucose transport and glycolysis.



# Prmt5 deletion causes fuel switch from lipid to glucose metabolism

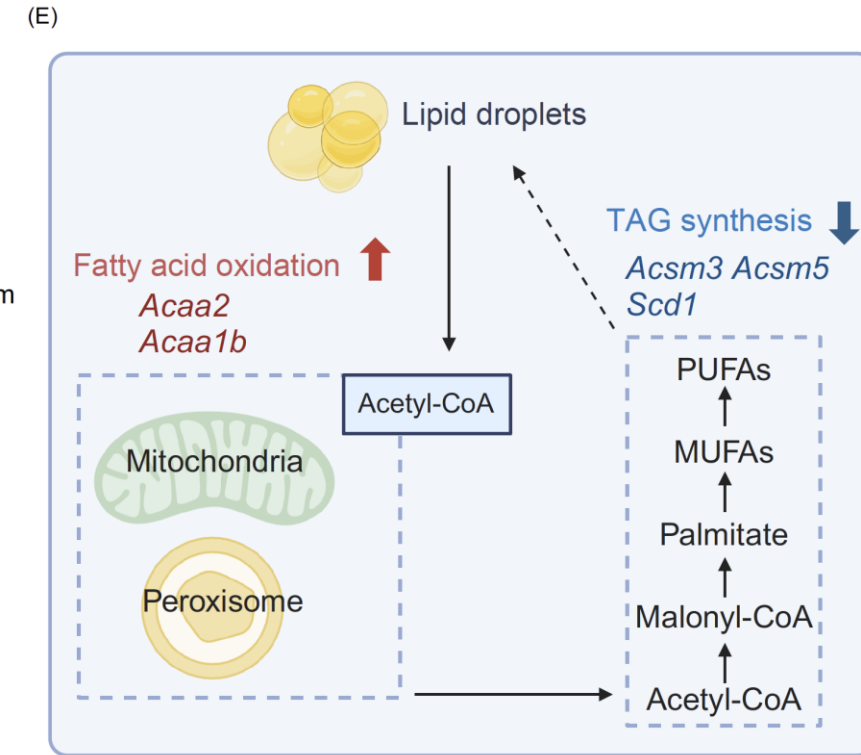
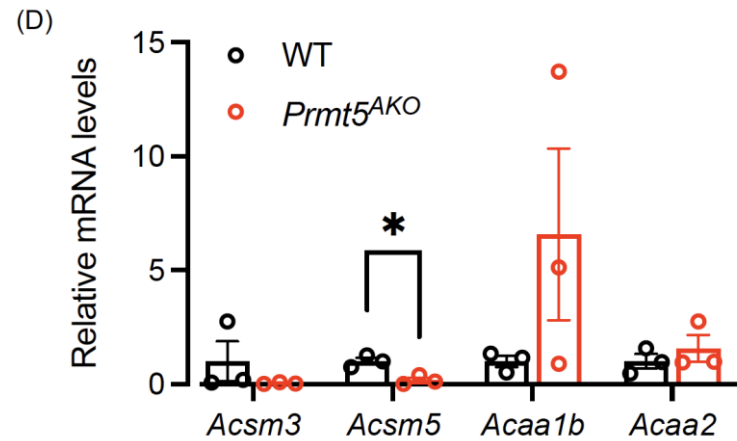
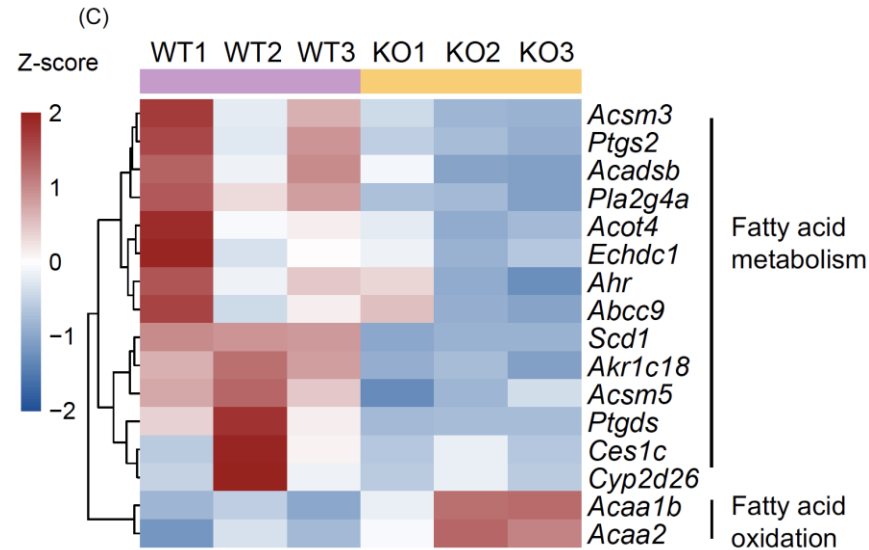
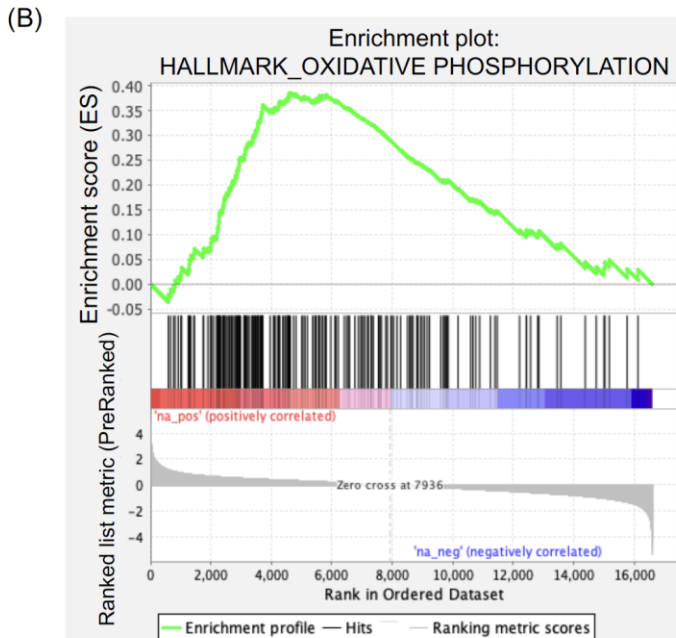
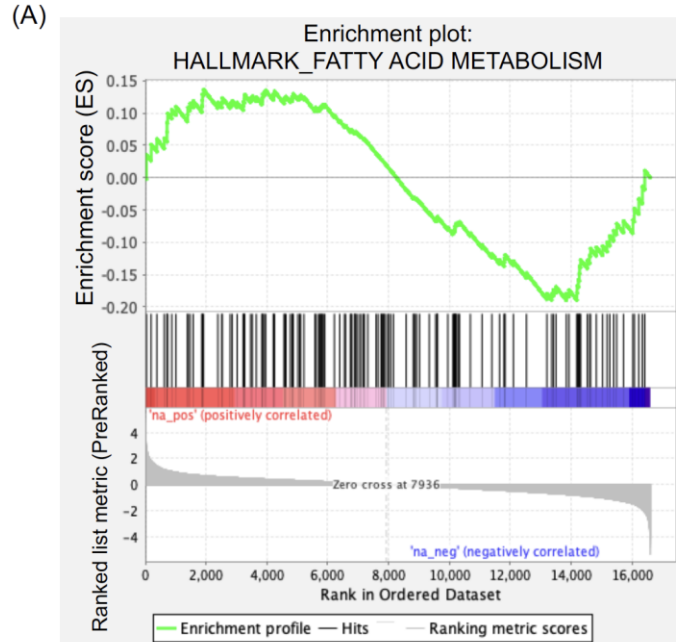


Figure 5. *Prmt5*<sup>AKO</sup> reduces fatty acid metabolism in WAT.



# Loss of *Prmt5* promotes cholesterol biosynthesis

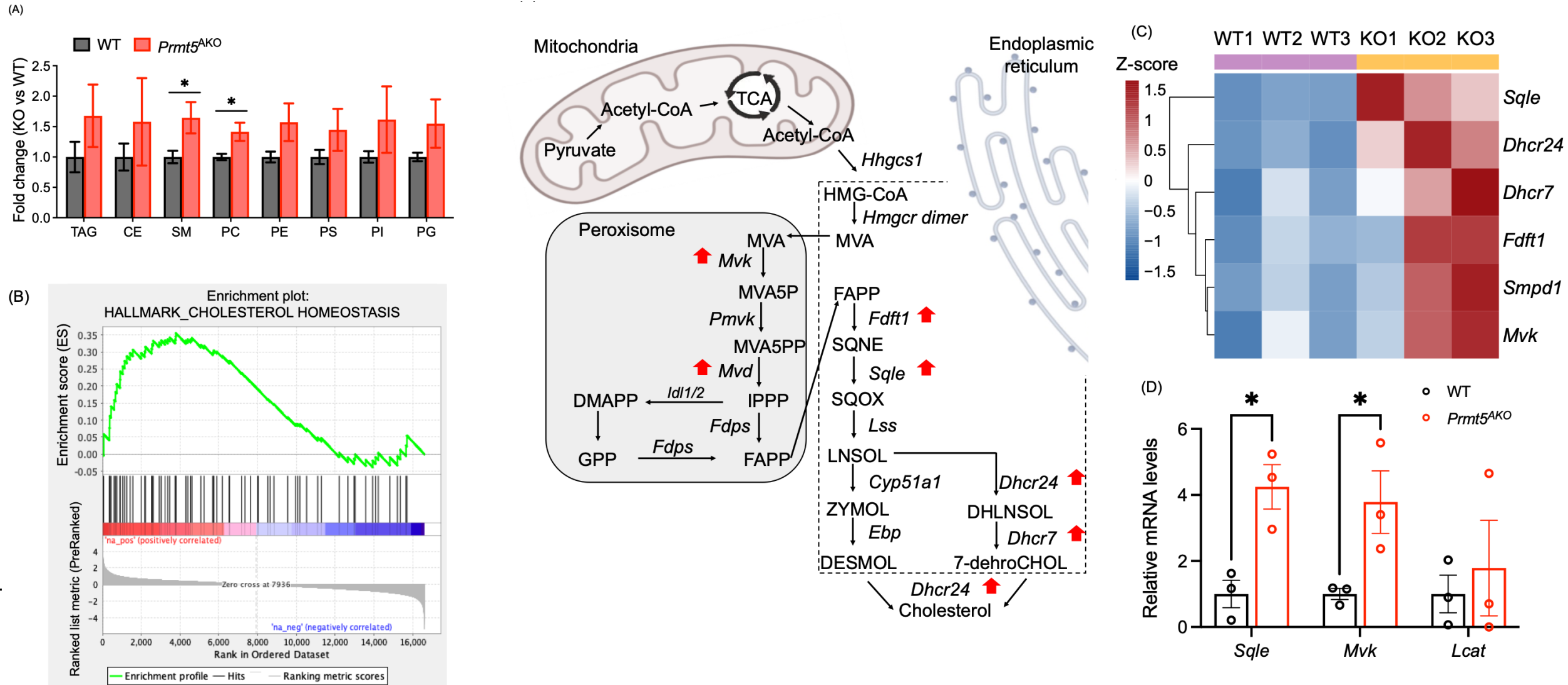


Figure 6. *Prmt5*<sup>AKO</sup> induces glycerophospholipid remodeling in WAT.



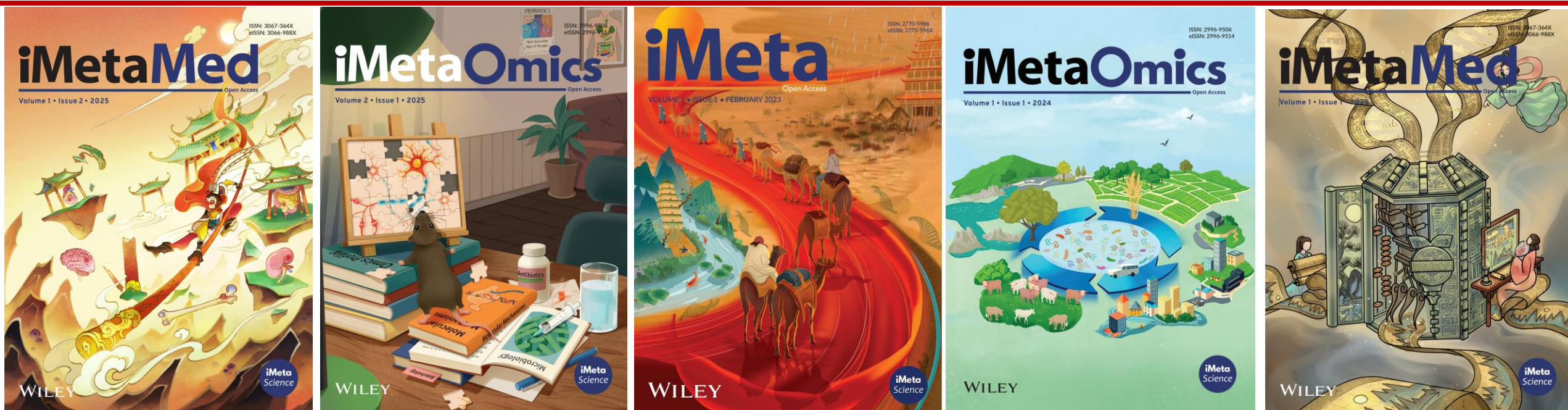
# Summary

- *Prmt5<sup>AKO</sup>* downregulates genes related to fatty acid (FA) uptake, triacylglycerol (TAG) biosynthesis and FA oxidation, while upregulating genes involved in glucose transport and glycolysis.
- In addition, *Prmt5<sup>AKO</sup>* upregulates genes involved in peroxisomes and cholesterol biogenesis.
- These changes led to alterations in lipid composition, notably reduced TAG content and selective increases in glycerophospholipid and sphingolipid species.

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