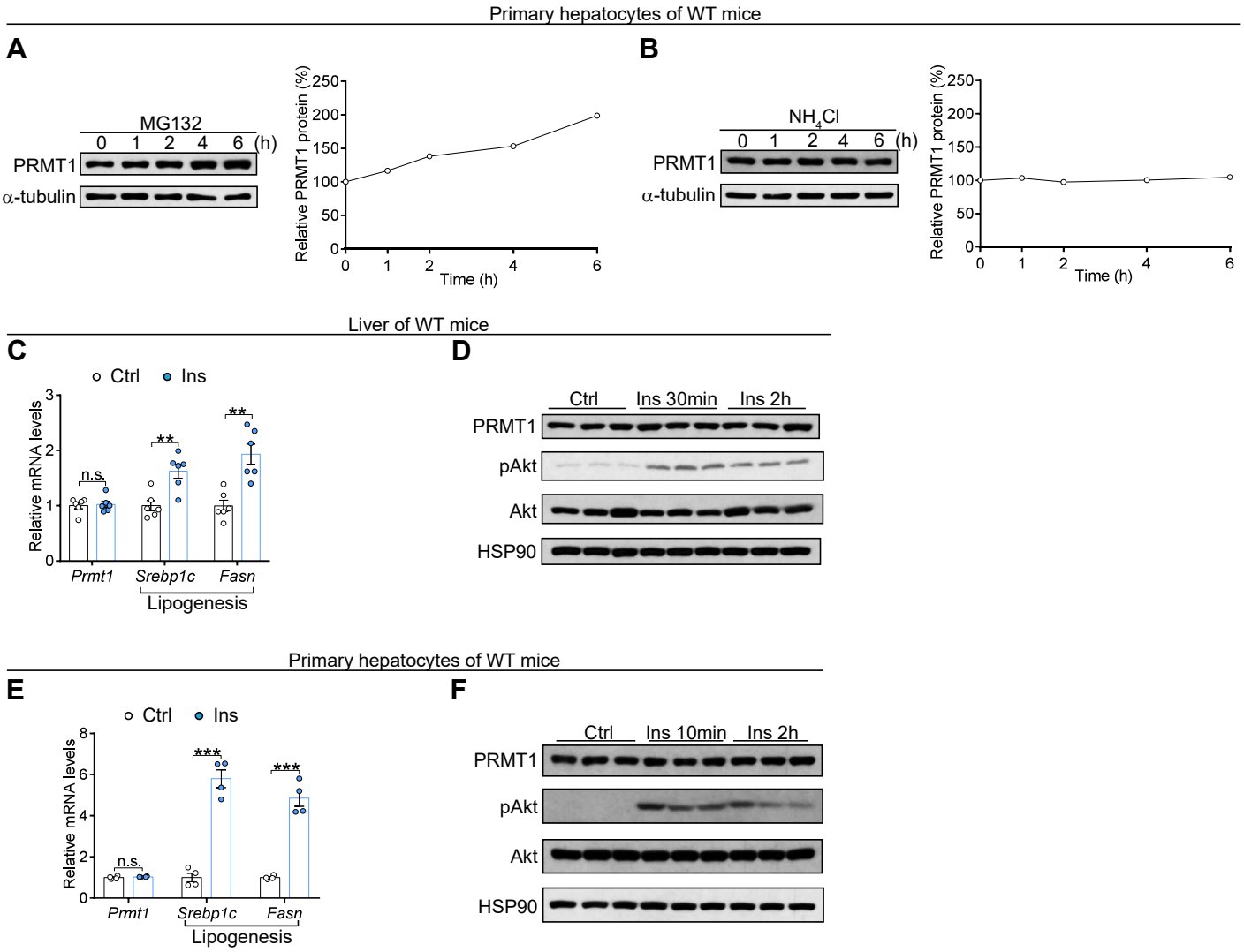
Supplementary Figure 1 (Related to Figure 1)



Supplementary Figure 1. Related to Figure 1. Loss of *Prmt1* reduces gluconeogenesis capacity in the liver. *A*) Immunoblot (left) and quantification (right) analyses of PRMT1 in the primary hepatocytes isolated from wild-type (WT) mice and treated with 20 μM MG132 for indicated time. α-tubulin was used as a loading control. *B*) Immunoblot (left) and quantification (right) analyses of PRMT1 in the primary hepatocytes isolated from WT mice and treated with 25 mM NH_4Cl for indicated time. α-tubulin was used as a loading control. *C*) qPCR analyses of *Prmt1* and lipogenesis marker mRNA levels in the liver of 16 hour-fasted WT mice intraperitoneally injected with vehicle (Ctrl) or 0.5 U/kg insulin (Ins) for 2 hours. *D*) Immunoblot analyses of PRMT1, pAkt, and Akt in the liver of 16 hour-fasted WT mice intraperitoneally injected with vehicle (Ctrl) or 0.5 U/kg insulin (Ins) for indicated time. HSP90 was used as a loading control. *E*) qPCR analyses of *Prmt1* and lipogenesis marker mRNA levels in the primary hepatocytes isolated from WT mice and stimulated with vehicle (Ctrl) or 100 nM insulin (Ins) for 2 hours. *F*) Immunoblot analyses of PRMT1, pAkt, and Akt in the primary hepatocytes isolated from WT mice and stimulated with vehicle (Ctrl) or 100 nM insulin (Ins) for indicated time. HSP90 was used as a loading control. Data are presented as mean ± SEM. $^{**}P < 0.01$; $^{***}P < 0.001$. n.s., not significant. 2-tailed Student's *t* test (C, E).