

## **Disruption of the mouse *Shmt2* gene confers embryonic anaemia via foetal liver-specific metabolomic disorders**

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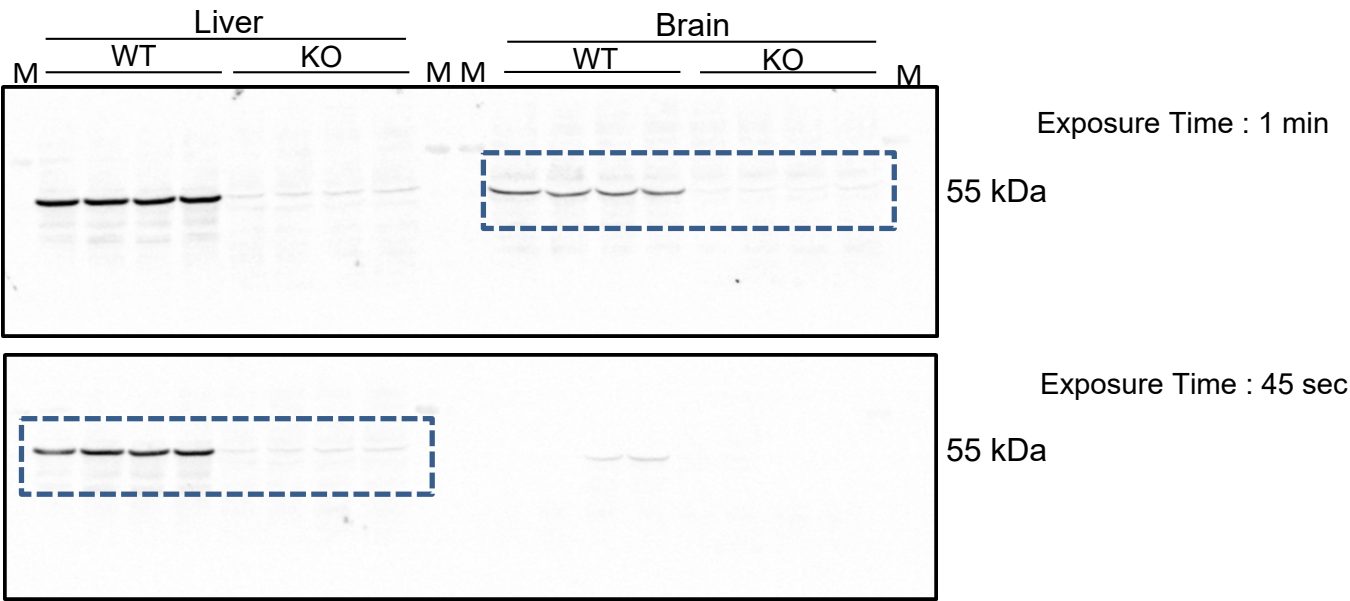
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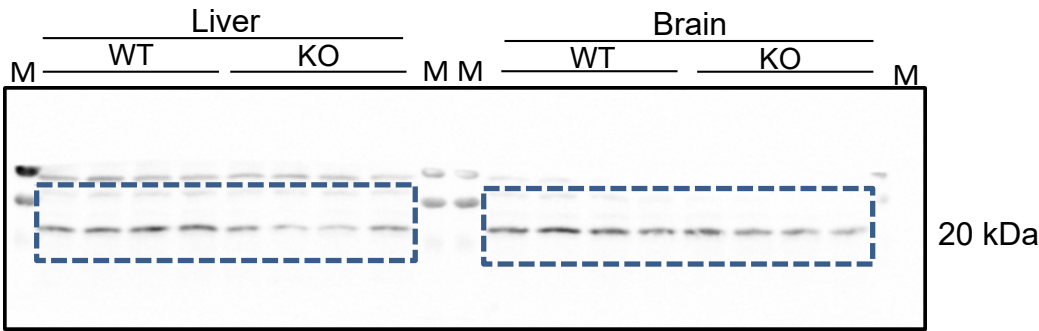
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Figure S1

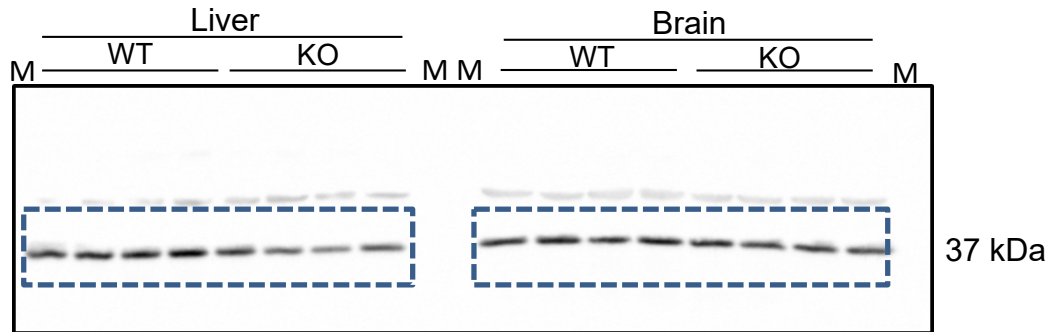
SHMT2 Antibody      M : Maker



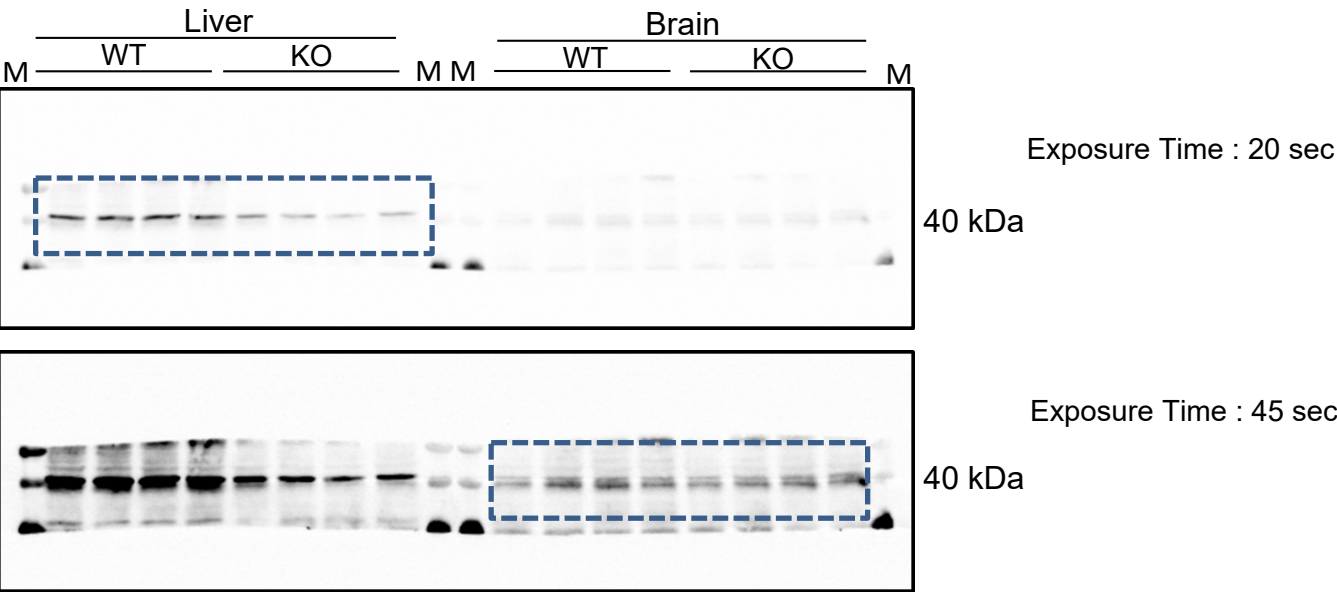
NDUFS4 Antibody



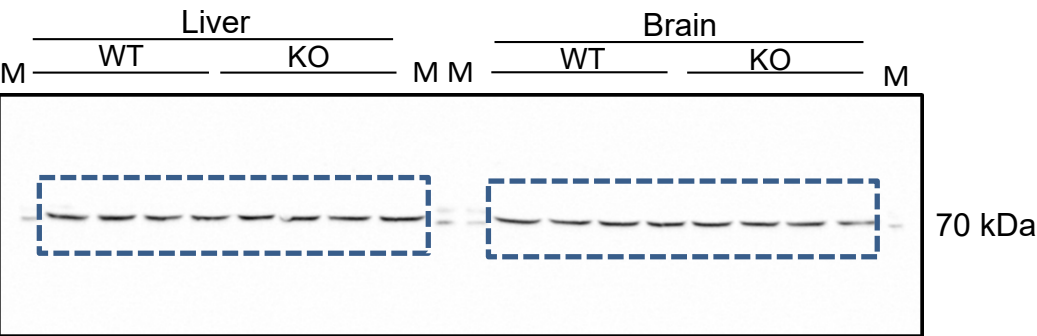
NDUFA9 Antibody



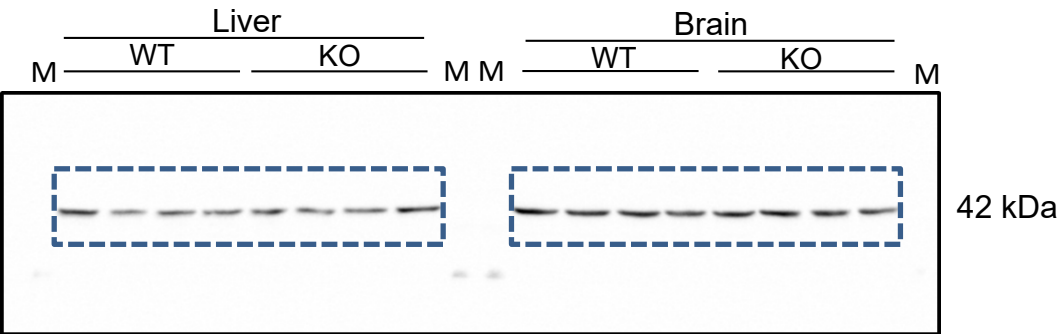
MT-CO1 Antibody



SDHA Antibody

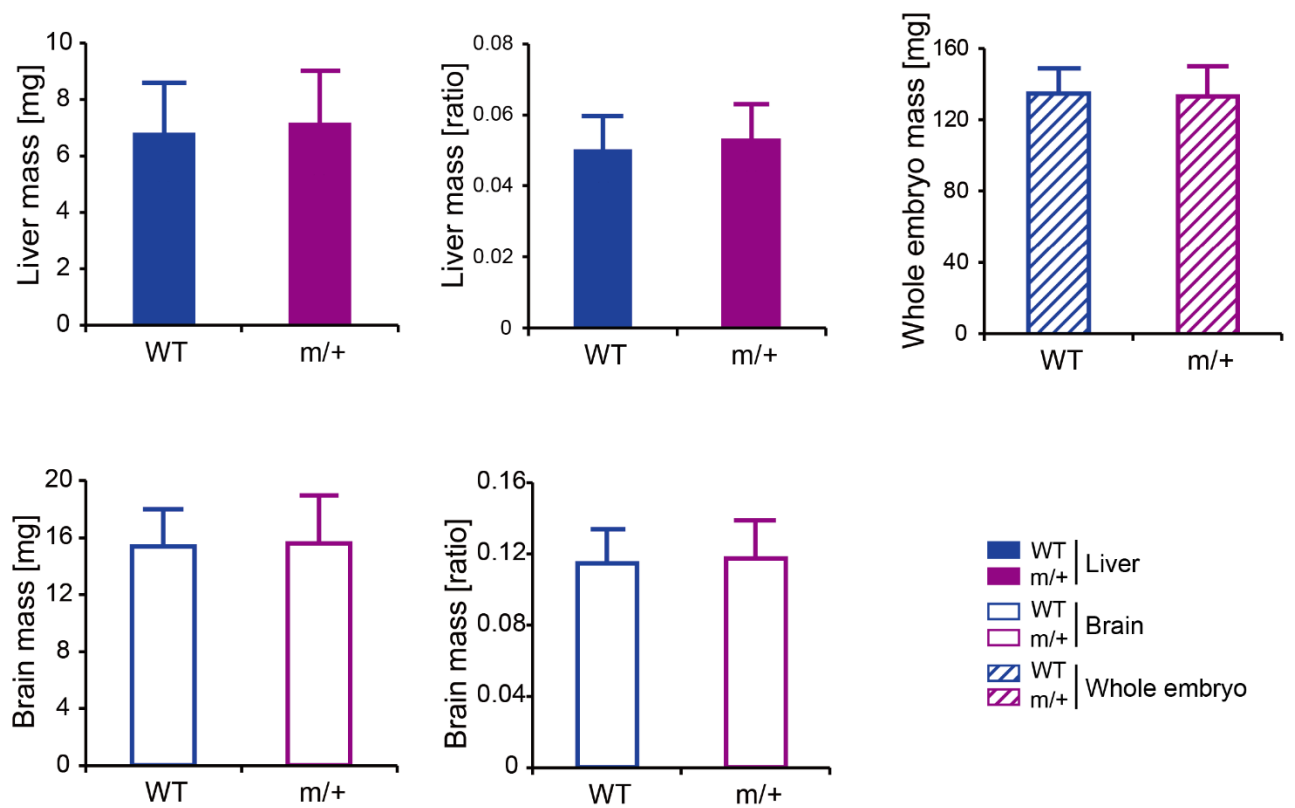


β-ACTIN Antibody



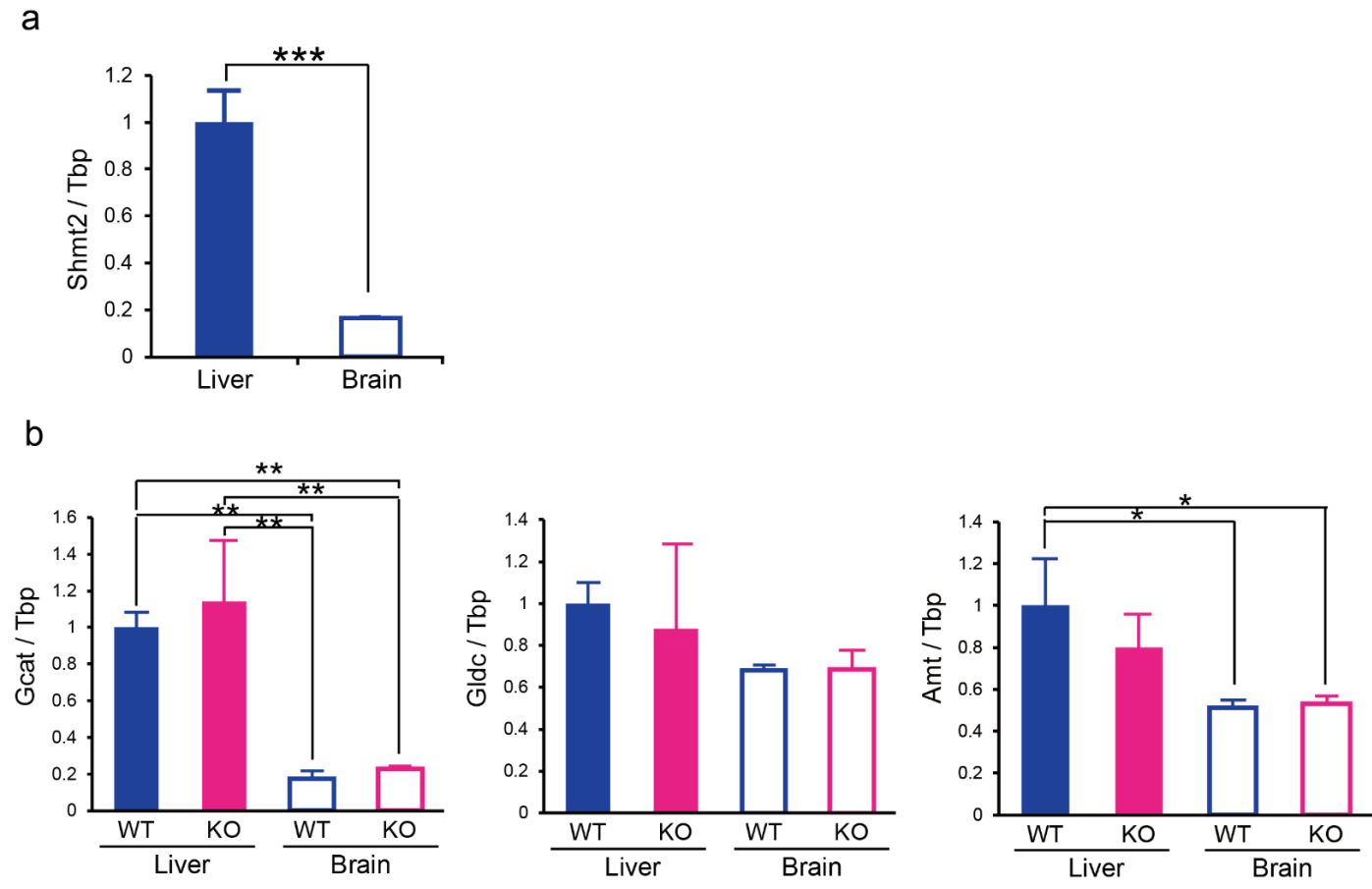
**Figure S1 Full-length blots of Figure 1**  
Membranes were cut to enable reacting of multiple antibodies. some membranes were detected at several exposure times. The dotted lines indicate the sites used in Figure 1.

Figure S2



**Figure S2 Effects of *Shmt2* disruption on the growth of livers and brains from E13.5 heterozygous *Shmt2* m/+ embryos.** Comparison of the weights of tissues or whole embryos between wild-type and heterozygous *Shmt2* m/+ mice. WT, wild-type foetal livers (filled blue bars), wild-type foetal brains (open blue bars), and wild-type whole embryos (a hatched blue bar). m/+, heterozygous foetal livers (filled purple bars), heterozygous foetal brains (open purple bars), and heterozygous whole embryos (a hatched purple bar). Data represent the means  $\pm$  S.D, Student's *t* test (WT, n=26; m/+, n=27).

Figure S3



**Figure S3 Real-time quantitative PCR to compare mRNA levels of genes involved in 1C metabolism.**  
(a) Comparison of mRNA levels of *Shmt2* involved in the serine pathway between wildtype foetal livers and brains. (b) Comparison of mRNA levels of *Gcat* involved in the threonine pathway and those of *Gldc* and *Amt* involved in the GCS in wild-type and *Shmt2*-knockout foetal livers and brains. Filled blue bars, wild-type foetal livers; filled red bars, *Shmt2*-knockout foetal livers; open blue bars, wild-type foetal brains; open red bars, *Shmt2*-knockout foetal brains. Data represent the means  $\pm$  S.D. \* $P < 0.05$ ; \*\* $P < 0.01$ , \*\*\* $P < 0.001$ , Tukey-Kramer method (WT, n = 3; KO, n = 3).