Supplementary Information

Lithium and exercise ameliorate insulin-deficient hyperglycemia by independently attenuating pancreatic α-cell mass and hepatic gluconeogenesis

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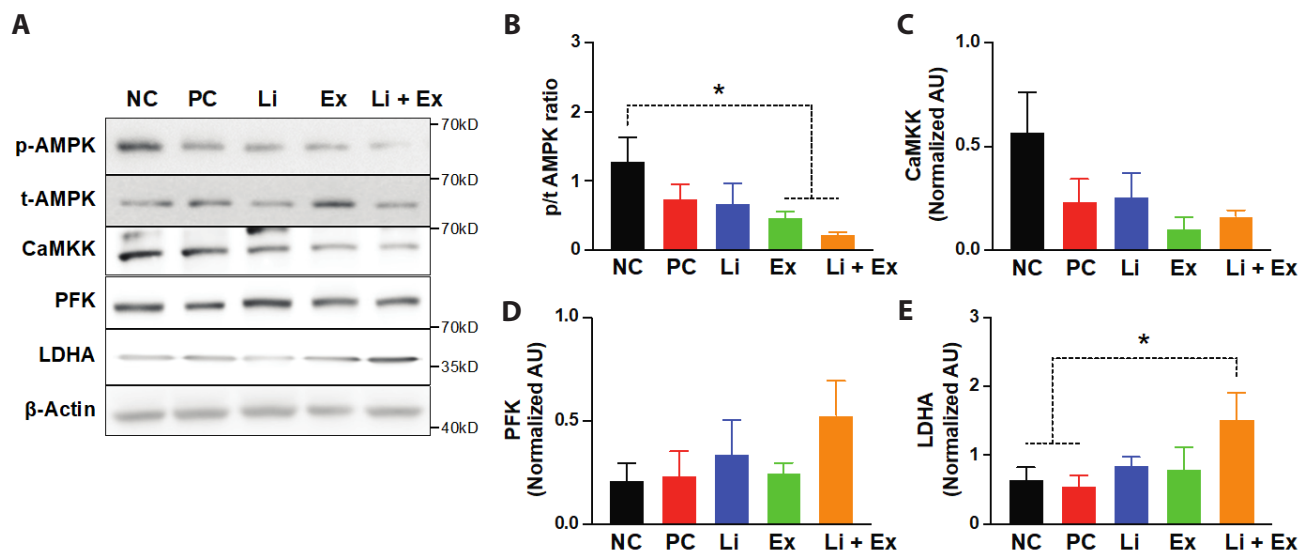
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Supplementary Figures

Supplementary Fig. 1. Lithium and exercise showed limited metabolic effects on skeletal muscle in type 1 diabetic conditions. (A–E) Immunoblot for the indicated proteins (A) and their quantified results (B–E) of type 1 diabetic skeletal muscles after 12 weeks of lithium and/or exercise administration. The ratio of phosphorylated AMPK to total AMPK (B) and protein levels of CaMKK (C), PFK (D), and LDHA (E). Data are presented as means ± S.E.M. NC, saline-received normal glycemic group; PC, streptozotocin (STZ)-induced hyperglycemic group; Li, lithium(Li)-administration to STZ-received mice; Ex, moderate exercise training to STZ-received mice; Li + Ex, co-administration of Li and exercise to STZ-received mice. *$p < 0.05$.

A AST

B ALT
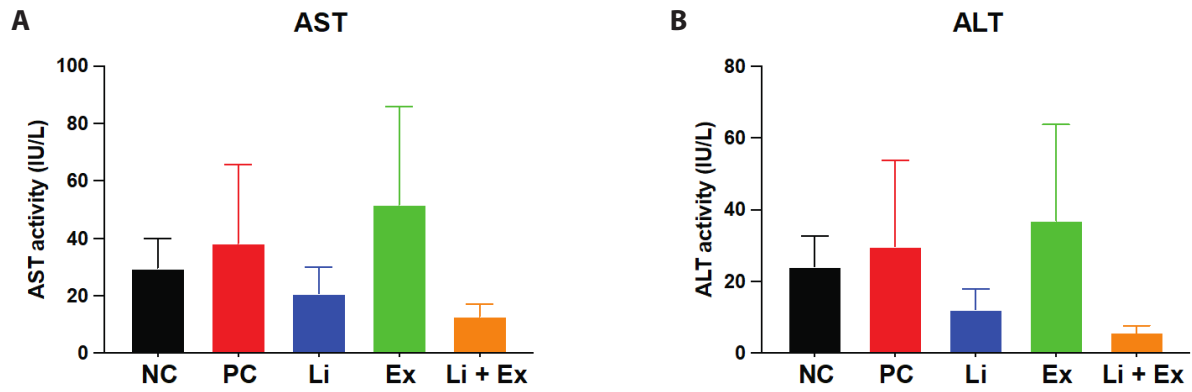
Supplementary Fig. 2. Streptozotocin treatment and additional lithium and exercise did not elicit any significant liver damage. (A, B) Blood aspartate aminotransferase (AST) (A) and alanine aminotransferase (ALT) (B) activity after streptozotocin and additional 12 weeks of lithium and/or exercise administration. Data are presented as means ± S.E.M. NC, saline-received normal glycemic group; PC, streptozotocin (STZ)-induced hyperglycemic group; Li, lithium(Li)-administration to STZ-received mice; Ex, moderate exercise training to STZ-received mice; Li + Ex, co-administration of Li and exercise to STZ-received mice.