Study reveals the role of ERRγ in the regulation of FGF23 gene expression following acute liver injury

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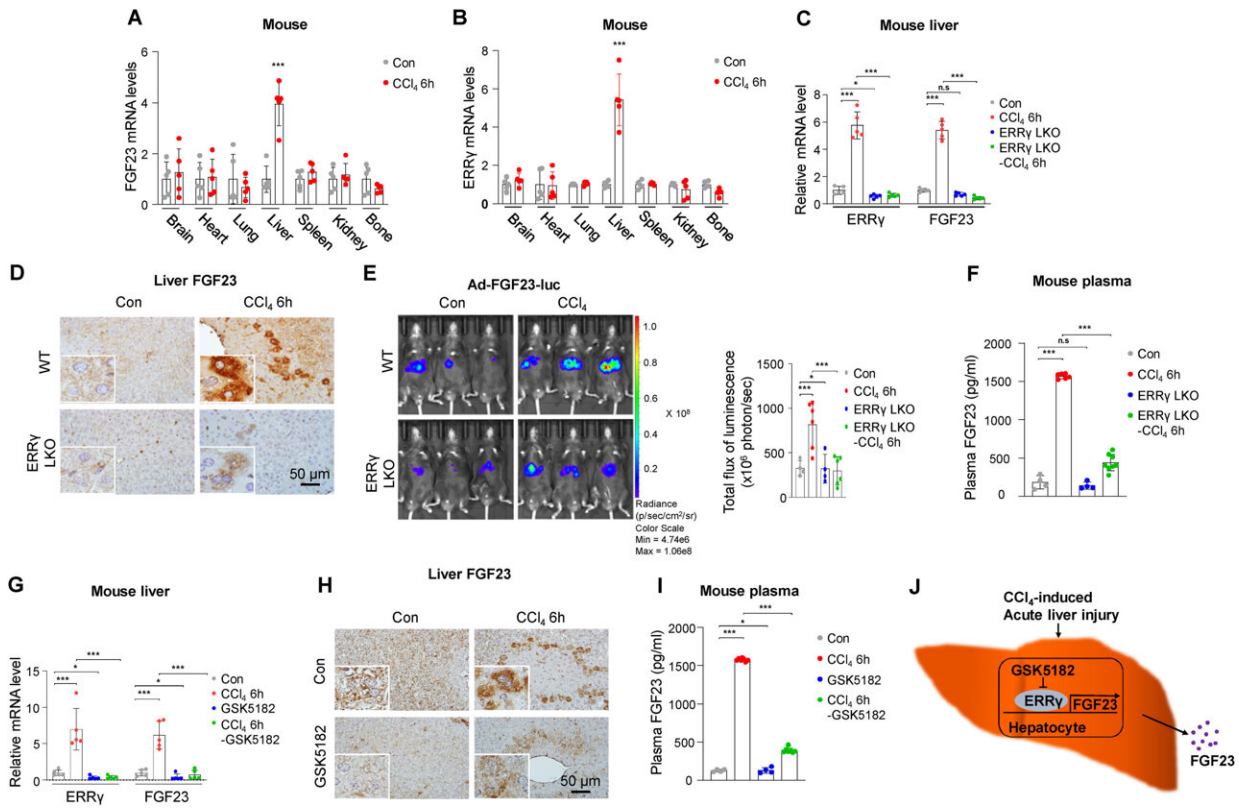

CCl_4-induced acute liver injury increases FGF23 gene expression and secretion in mouse liver through ERRγ. (A, B) Quantitative PCR analysis of total RNA obtained from the livers of mice injected with CCl_4 (1 mL/kg body weight of 10% CCl_4 dissolved in corn oil) for 6 h (n = 5 per group). (C–F) WT and ERRγ-LKO mice were injected with CCl_4 for 6 h (n = 5 per groups). (C) Quantitative PCR analysis of total RNA isolated from livers. (D) Representative images of FGF23 immunohistochemical analysis in liver sections. (E) Representative in

vivo images of hepatic FGF23 promoter WT-luciferase (Ad-FGF23-luc) activity in WT and ERRγ-LKO mice injected with or without CCl_4 (n = 4 for WT-Con and ERRγ-LKO Con; n = 6 for WT-CCl_4 and ERRγ-LKO CCl_4 group). (F) Plasma FGF23 levels measured by ELISA. (G–I) WT mice were injected with CCl_4 in the presence or the absence of GSK5182 and sacrificed after 6 h (n = 5 per group). (G) Quantitative PCR analysis of total RNA isolated from liver. (H) Representative images of FGF23 immunohistochemical analysis in liver sections. (I) Plasma FGF23 levels measured by ELISA. (J) Schematic diagram of ERRγ-mediated hepatic FGF23 gene expression and secretion in CCl_4-induced acute liver injury. Data indicate mean ± SEM values. Data in (A) and (B) were analyzed by two-tailed Student's t test. Data in C, E, F, G and I were analyzed by ordinary one-way ANOVA with Tukey's multiple comparisons test. Significance levels denoted as ∗P