

# Liver Fibrosis

## CCl<sub>4</sub>-Induced Mouse Model

Liver fibrosis occurs in most types of chronic liver diseases. To test the efficacy of new drugs against liver disease, mice can be systemically injected with carbon tetrachloride (CCl<sub>4</sub>). CCl<sub>4</sub> is an organochloride and known as one of the most potent hepatotoxins and as such inducing liver fibrosis.

C57Bl/6 mice at an age of 7 weeks are intraperitoneally treated three times per week with CCl<sub>4</sub> or vehicle for a total of 9 weeks to induce liver fibrosis.

- Liver to body weight ratio
- Increased Col1a1 mRNA and protein levels
- Increased Acta2 mRNA levels
- Increased hydroxyproline protein levels
- Increased smooth muscle actin (SMA) protein levels

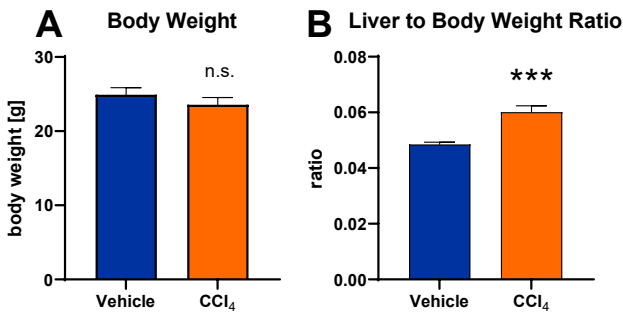


Figure 1: Body weight and liver to body weight ratio of CCl<sub>4</sub>- and vehicle-treated C57Bl/6J RccHsd mice. A: body weight. B: Liver to body weight ratio. Unpaired t-test. n = 6 per group; mean + SEM. \*\*\*p<0.001; n.s.: not significant.

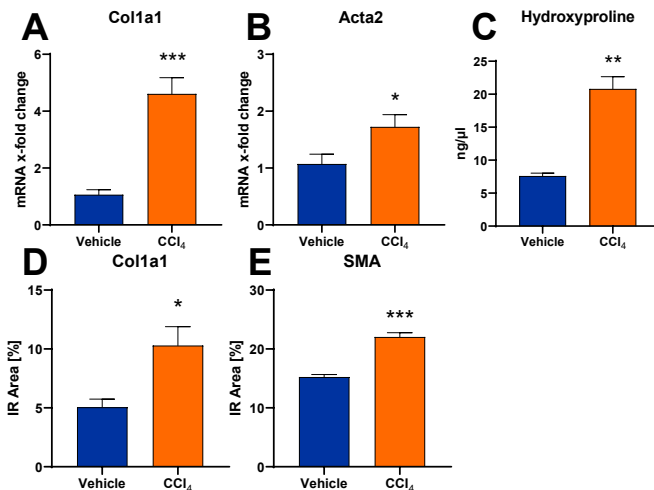


Figure 2: Expression levels of typical liver disease markers after CCl<sub>4</sub> treatment. mRNA expression levels of Col1a1 (A) and Acta2 (B). Levels are presented as x-fold change using the 2<sup>Δ(-ΔΔCT)</sup> method in relation to the HKG HPRT and vehicle. C: Hydroxyproline protein levels in ng/μl. Quantification of Col1a1 (D) and SMA (E, smooth muscle actin) immunoreactive (IR) area in percent. A-E: All markers were measured in the liver after 9 weeks of CCl<sub>4</sub> or vehicle treatment. Unpaired t-test; n = 5/6 per group; mean + SEM; \*p<0.05, \*\*p<0.01, \*\*\*p<0.001.