



Article

Fructose Causes Liver Damage, Polyploidy and Dysplasia in the Setting of Short Telomeres and p53 Loss

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Supplementary Materials

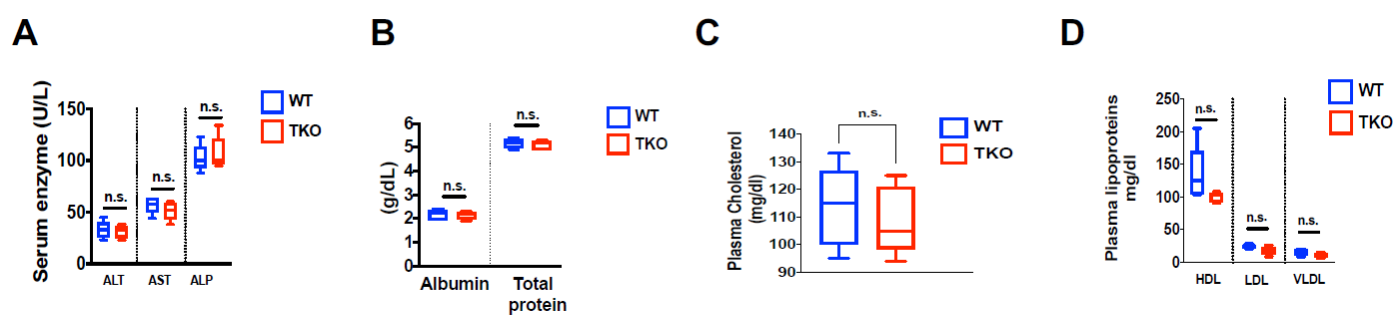


Figure S1. TKO livers are indistinguishable from WT mice on regular chow diet (A–D) Transaminases, albumin, globulin, cholesterol, HDL, LDL and VLDL are in normal range in TKO mice indicating hepatocytes are not damaged and have normal synthesis capacity. 5 mice were analyzed per group for each experiment and statistical differences were calculated by t-test. n.s. = not significant.

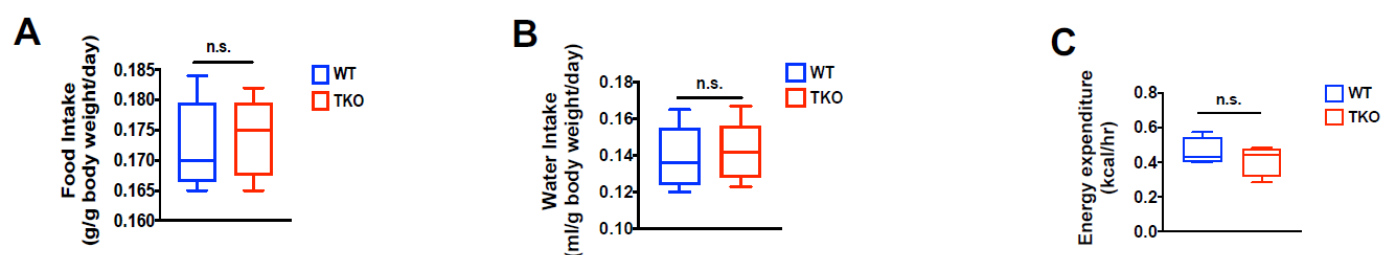
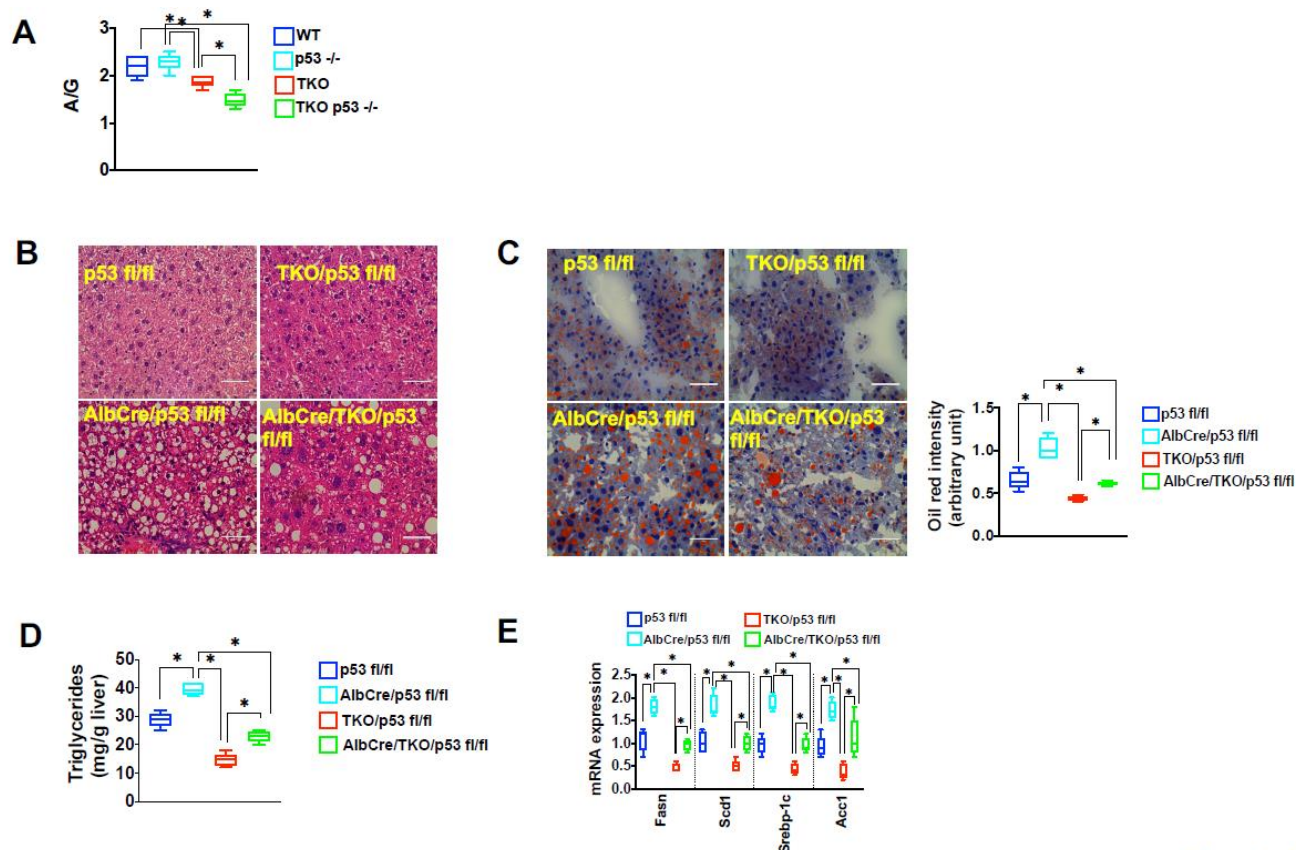


Figure S2. Mice with short telomeres accumulate less fat in liver tissue upon fructose treatment (A–C) No differences in food, water intake and energy expenditure between WT and TKO mice on a fructose diet. 5 mice per group were analyzed analyzed for each experiment and statistical differences were calculated by t-test. * denotes $p < 0.05$.



Supp. Fig. 5

Figure S3. p53 deletion increases fat accumulation in TKO mice (A) Albumin/Globulin ratio is further reduced in TKO p53^{-/-} mice indicating further compromise in TKO liver synthesis capacity compared to TKO mice with intact p53 (B) H&E staining shows increase in vacuole size in TKO p53^{-/-} mice suggesting increased fat (C) p53 deficiency in TKO livers alone is sufficient to increase fat content as determined by ORO staining (D) TG content in TKO liver-specific p53 null mice (AlbCre/TKO/p53^{fl/fl}) is significantly increased compared to TKO mice with intact p53 (E) RT-qPCR analysis demonstrates increased expression of TG synthesis regulating genes in AlbCre/TKO/p53^{fl/fl} mice. 5-8 mice per group were analyzed for each experiment and statistical differences were calculated by t-test. * denotes $p < 0.05$.