A Gilbert syndrome-associated haplotype protects against fatty liver disease

in humanized transgenic mice

Steffen Landerer, Sandra Kalthoff, Stefan Paulusch, Christian P. Strassburg*

Department of Internal Medicine I, University Hospital Bonn, 53127 Bonn, Germany

* Corresponding authors

Correspondence: Christian P. Strassburg, MD

Department of Internal Medicine I,

University Hospital Bonn,

Venusberg-Campus 1,

53127 Bonn, Germany

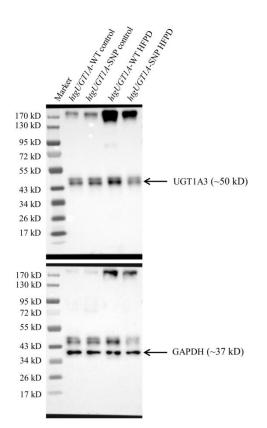
Telephone: +49 22828715216, Fax: +49 22828719657

Email: christian.strassburg@ukbonn.de

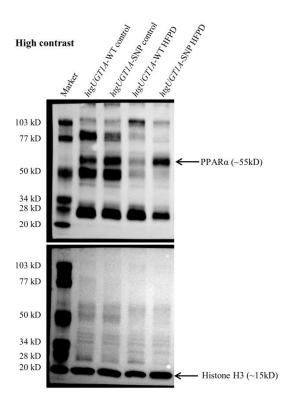
Contributing authors:

| Steffen Landerer: | steffen.landerer@ukbonn.de |
|-------------------|----------------------------|
| Sandra Kalthoff: | sandra.kalthoff@ukbonn.de |
| Stefan Paulusch: | stefan.paulusch@ukbonn.de |

Supplementary figure S1



Supplementary figure S2



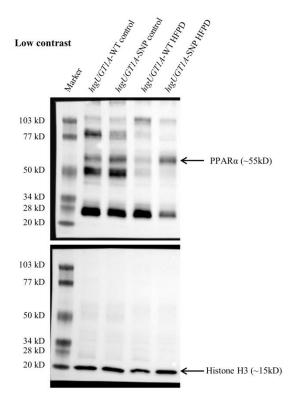


Figure Legends Supplementary figures

Fig. S1: Western blot analysis of hepatic UGT1A3 protein quantity. Higher protein amount was detected in *htgUGT1A*-WT mice.

Fig. S2: Western blot analysis of nuclear peroxisome proliferator-activated receptor alpha (PPARα) protein levels (high contrast left panel, low contrast right panel).