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Data Article

Data on Lipocalin 2 and phosphatidylinositol 3-kinase signaling in a methionine- and choline-deficient model of non-alcoholic steatohepatitis

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ABSTRACT

The data presented in this brief report support the research article "*Altered mitochondrial and peroxisomal integrity in lipocalin-2-deficient mice with hepatic steatosis*" [1, doi: http://dx.doi.org/10.1016/j.bbadis.2017.04.006]. We tested whether the absence of Lipocalin-2 (LCN2) could dysregulate the phosphatidylinositol 3-kinase/protein kinase B (PI3K-PKB) pathway and hepatic homeostasis in Non-Alcoholic-Steatohepatitis (NASH). The article highlights the role of LCN2 in hepatic homeostasis.

Specifications Table

Subject area	*Biology*
More specific subject area	*Liver biology*

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Type of data	*Figures (Immunofluorescence staining, Western blot analysis)*
How data was acquired	*Microscope (Nikon ECLIPSE 80i)* *Chemiluminescent detection (Boehringer Mannheim GmbH Lumi-Imager)*
Data format	*Raw*
Experimental factors	*Liver sections were derived from WT and Lcn2$^{-/-}$ mice [2] after feeding a MCD or standard chow diet for 6 weeks.* *Protein extracts were prepared from the same livers for Western blot analysis.* *Hepatocytes were isolated from WT and Lcn2$^{-/-}$ mice for in vitro experimentation.*
Experimental features	*Liver sections from WT and Lcn2$^{-/-}$ mice were immunostained for PIP3. Negative and positive controls were also used.* *100 μg of protein extracts from livers were used to detect PKB, phospho PKB, LCN2 and* Glyceraldehyde 3-phosphate dehydrogenase (*GAPDH) by Western blot.* *Hepatocytes were cultured from WT and Lcn2$^{-/-}$ mice. These were left untreated or treated with bovine insulin to induce PI3K and PIP3. Respective cells were immunostained for PIP3.*
Data source location	*Aachen, Germany*
Data accessibility	*Data not deposited elsewhere outside this article. Primary data are published in [1].*

Value of the data

- LCN2 is a versatile molecule participating in several pathways of hepatic homeostasis. The data describe the regulation of the PI3K/PIP3/PKB pathway with regard to the presence of LCN2 in a NASH model.
- The data study the value of LCN2 in PI3K signaling in NASH.
- The data are useful to understand how the absence of LCN2 affects PIP3 production.
- Signaling analyses could lead to novel treatment strategies to modulate medical conditions where the PI3K-PKB signaling pathway is dysregulated such as fatty liver and diabetes type 2 [3].
- The data presented in this article, could be compared with data from other animal NASH models to verify the strength of the effect. Moreover, comparison of this data to human NASH data on PI3K signaling and PIP3 functions could drive the development of novel LCN2 targeted therapies.

1. Data

The data include protein detection of members of the PI3K signaling and PIP3 quantification (Figs. 1-3). The detection of respective biomolecules was done with fluorescence immunohistochemistry/immunocytochemistry and Western blot. The analyzed liver sections and primary hepatocytes originated from WT and *Lcn2$^{-/-}$* mice fed on a standard chow diet or an MCD diet. The hepatocytes were treated with insulin to trigger PIP3 and PI3K before immunodetection. Untreated control cells were used to compare.

2. Experimental design, materials and methods

2.1. Liver cryosections

Mice, WT and LCN2 deficient, were sacrificed under isoflurane (Forene®) anaesthesia (Abbott, Wiesbaden, Germany), and whole livers were resected. The livers were snap frozen in liquid nitrogen