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Data Article

Data on importance of hematopoietic cell derived Lipocalin 2 against gut inflammation

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ABSTRACT

The data herein is related to the research article entitled "Microbiota-inducible innate immune siderophore binding protein Lipocalin 2 is critical for intestinal homeostasis" (Singh et al., 2016) [1]. In the present article, we monitored dextran sodium sulfate (DSS)-induced colitis development upon Lipocalin 2 (Lcn2) neutralization, and examined the survival of Lcn2 deficient (Lcn2KO) mice and their WT littermates upon DSS challenge. To dissect the relative contribution of immune and non-immune cells-derived Lcn2 in mediating protection against gut inflammation, we generated respective bone marrow chimera and evaluated their susceptibility to IL-10 receptor neutralization-induced chronic colitis.

Neutralization of Lcn2 in WT mice resulted in exacerbated DSS-induced colitis. Notably, mice lacking Lcn2 exhibited 100% mortality whereas only 20% mortality was observed in WT mice upon DSS challenge. Further, data from bone marrow chimera showed that immune cell-derived Lcn2 is the major contributor in conferring protection against colitis.

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Specification Table

Subject area	Biology
More specific subject area	Lipocalin 2, inflammatory bowel disease
Type of data	Graphs, figures
How data was acquired	Assessment of colitis parameters, percent survival, myeloperoxidase assay, enzyme linked immunosorbent assay (ELISA), Biotek Eon™ microplate spectrophotometer.
Data format	Analyzed
Experimental factors	The susceptibility to DSS-induced colitis was assessed in Lcn2KO mice and their WT littermates. Lcn2 bone marrow chimeras was generated and evaluated for susceptibility to IL-10 receptor neutralization-induced chronic colitis.
Experimental features	Percent survival and analysis of standard colitis parameters
Data source location	Pennsylvania, USA
Data accessibility	Data are provided with this article

Value of the data

- The data are valuable to researchers interested in investigating the role of Lcn2 in inflammatory bowel disease.
- The data provide information on protective roles of immune vs non-immune cell derived Lcn2 during gut inflammation.
- The data support future studies in delineating the role of Lcn2 in mucoprotection.

1. Data

In the data, we presented the higher mortality rate of Lcn2KO mice in DSS-induced acute colitis when compared to their WT littermates (Fig. 1). Further, neutralization of Lcn2 in DSS-treated WT mice resulted

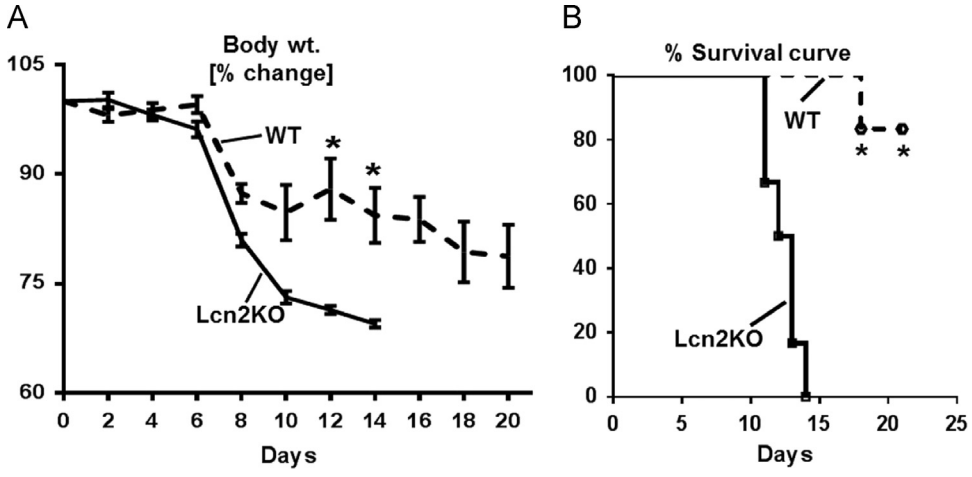

Fig. 1. Data on the susceptibility of Lcn2KO mice to DSS-induced acute colitis. (A) Percent body weight loss of DSS-treated Lcn2KO mice and their WT littermates ($n=5$). (B) Percent survival of WT and Lcn2KO mice upon challenge with 1.5% DSS for 7 days ($n=5$). Data are expressed as mean ± SEM. One-way analysis of variance with the Tukey multiple comparison test were used. $^{*}p<0.05$ was considered statistically significant.