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Summary

PANETH CELLS REGULATE INTESTINAL LYMPHANGIOGENESIS AND LIPID METABOLISM IN METABOLIC DYSFUNCTION-ASSOCIATED STEATOTIC LIVER DISEASE

Sheida Moghadamrad

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Paneth cell deficiency in mice attenuated high-fat diet-induced hepatic steatosis and fibrosis by impairing intestinal lymphatic-mediated lipid absorption, resulting in increased fecal fat excretion and reduced hepatic lipid delivery.

KEY POINTS

- Paneth cell-deficient mice fed high-fat diet for 16 weeks had significantly lower weight gain, less severe hepatic steatosis, and reduced liver fibrosis compared to controls.
- Imaging mass cytometry revealed lower collagen deposition and steatosis with fewer macrophages and neutrophil activation/infiltration in Paneth cell-deficient livers.
- Intestinal expression of lymphangiogenic genes VEGFC, VEGFR3, and Prox1 was reduced in Paneth cell-deficient mice on both high-fat and standard diets.
- Paneth cell-deficient mice on high-fat diet showed higher fecal fat excretion than controls, consistent with impaired intestinal lymphatic lipid absorption.
- In vivo microscopy demonstrated lower disruption of intestinal vascular barriers in Paneth cell-deficient mice on high-fat diet compared to controls.
- Fecal microbiota beta diversity differed significantly in Paneth cell-deficient mice fed standard diet after 16 weeks, with decreased Bacteroidota and increased Firmicutes in high-fat diet groups.

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