MICE WITH A CONDITIONAL INTESTINAL EPITHELIAL ABLATION OF TISSUE-NONSPECIFIC ALKALINE PHOSPHATASE EXHIBIT DISTINCT INTESTINAL AND EXTRAINTESTINAL CHANGES

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Deletion of tissue non-specific alkaline phosphatase (TNAP) in intestinal epithelial cells affects cellular physiology and has systemic effects on liver metabolism in mice.

KEY POINTS

- Epithelial-specific Alpl knockout mice showed decreased Alpl expression in the colon with compensatory upregulation of other alkaline phosphatase isozymes (Akp6 in colonic cells, Akp3 and Akp6 in jejunal organoids).
- Loss of Alpl in intestinal epithelial cells caused downregulation of the ectonucleotidase Nt5e and altered chemokine expression (increased Cxcl10, reduced Cxcl1) in jejunal organoids.
- Intestinal epithelial Alpl deletion led to substantial changes in liver gene expression, including increased Tff3, Tnf and Tgfb1, modulation of metabolic genes (Fads2 and Sult3a1), and decreased liver choline levels.
- No differences were detected in colonic inflammatory markers (Il6, Tnf, S100a8, S100a9) in Alpl knockout mice under basal conditions.
- This experimental study in mice provides evidence that intestinal epithelial TNAP plays a role in gut-liver axis communication and may be relevant for researchers investigating alkaline phosphatase function in gastrointestinal and hepatic physiology.

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