

DISRUPTION OF TUFT CELL–ILC2 IMMUNE HOMEOSTASIS BY THE NANOPLASTICS–GUT MICROBIOTA–SUCCINATE AXIS EXACERBATES ILEAL INFLAMMATION

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Oral exposure to polystyrene nanoplastics in mice causes ileal immune dysfunction through microbiota-dependent depletion of succinate and Tuft cells, which is reversed by succinate supplementation.

KEY POINTS

- Nanoplastics undergo enterohepatic–gallbladder recirculation, resulting in sustained high concentrations in the intestinal mucosa.
- Prolonged nanoplastic exposure (3–6 weeks) induced chronic inflammatory cell infiltration, with significant reductions in crypt density, Tuft cells, goblet cells, Paneth cells, and group 2 innate lymphoid cells, most pronounced in the ileum.
- Nanoplastic exposure significantly reduced cecal succinate levels and markedly decreased the relative abundance of succinate-producing bacterial taxa, without altering tissue succinate.
- Succinate supplementation effectively reversed all nanoplastic-induced alterations, including inflammatory infiltration, crypt loss, and depletion of Tuft cells, goblet cells, Paneth cells, and ILC2s.
- Under germ-free conditions, nanoplastic-induced toxicity was abolished, and no direct toxic effect on Tuft cells was observed in intestinal organoids, demonstrating that toxicity depends on the gut microbiota.
- The proposed mechanism is that nanoplastic-induced dysbiosis impairs luminal succinate production, leading to depletion of ileal Tuft cells, which in turn diminishes proliferation and activation of goblet cells, Paneth cells, and ILC2s.

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