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Summary

CONSUMPTION OF ULTRA-PROCESSED FOOD PROMOTE INFLAMMATORY POTENTIAL OF FECAL MICROBIOME THROUGH ALTERING THE BALANCE OF TH17 AND RORYT TREG

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High ultra-processed food consumption alters gut microbiome inflammatory potential and shifts intestinal T-cell balance toward pro-inflammatory Th17 cells and away from protective regulatory T cells in germ-free mice colonized with human fecal microbiota.

KEY POINTS

- In 2,336 healthy first-degree relatives of Crohn's disease patients, higher ultra-processed food consumption was associated with significant differences in 36 serum proteins ($q<0.007$), including enrichment of immune-related proteins IL2RA, TNFRS9, TNFRSF11A, and CD4.
- Germ-free mice receiving feces from high ultra-processed food consumers exhibited reduced proportion of protective RORγt⁺ regulatory T cells in both ileum and colon ($p<0.001$).
- Mice fed a Western diet and colonized with feces from high ultra-processed food consumers showed significantly higher proportion of Th17 cells in the colon compared to mice receiving feces from low ultra-processed food consumers ($p=0.023$).
- Several taxa predominantly from Lachnospiraceae and Ruminococcaceae families were associated with ultra-processed food consumption, and presence of Marvinbryantia was associated with increased Crohn's disease risk among high consumers.
- The findings suggest dietary modulation of the microbiome could represent a potential strategy for Crohn's disease prevention by addressing immune dysregulation.

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