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Summary

INTESTINAL INFLAMMATION AND ASSOCIATED CHANGES OF CYTOKINE AND TRANSMITTER TRANSCRIPTS IN THE BRAIN DURING ACTIVE CELIAC DISEASE IN THE HUMANIZED NOD DQ8 MOUSE MODEL

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In a NOD-DQ8 mouse model of celiac disease, gluten exposure for 2 weeks induced significant proinflammatory cytokine upregulation in the brain (IL17a, IL23a, IL22b, IL6, IL33, Nlrp3, COX2) and altered neurotransmitter metabolism, including upregulation of dopamine beta hydroxylase, kynurenine-3-monooxigenase, and quinolinate phosphoribosyl transferase.

KEY POINTS

- Mice were sensitized to gliadin with cholera toxin and fed a gluten-containing or gluten-free diet for 2 weeks, with brain and small intestine analyzed by qPCR and FACS.
- Brain tissue showed highly significant upregulation of IL17a, IL23a, IL22b, IL6, IL33, Nlrp3, and COX2, confirming gut-brain immune communication in celiac disease.
- Neuroactive tryptophan metabolism was upregulated via increased kynurenine-3-monooxigenase and quinolinate phosphoribosyl transferase, and dopamine/noradrenaline metabolism was enhanced by increased dopamine beta hydroxylase.
- The upregulation of enzymes encoding neurotransmitter synthesis may explain fatigue and mental dysfunction observed in patients with active celiac disease.
- The presenter observed that gluten-treated mice behaved more apathetically during handling compared to controls, prompting the brain analysis.

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