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Summary

DEFICIENCY OF TOLL-LIKE RECEPTOR 4 SIGNALING INFLUENCES SMALL INTESTINE NEUROMUSCULAR FUNCTION DURING AGING

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TLR4 deficiency protects against age-related small intestine dysmotility and myenteric neuroglial alterations in mice.

KEY POINTS

- In wild-type mice, aging caused dysmotility, delayed gastrointestinal transit, reduced cholinergic receptor response (Emax -43%), defective neurotransmission (-45% at 10 Hz), 26% loss of HuC/D+ neurons, and increased glial markers S100β (+25%), GFAP (+28%), and alpha-synuclein (+96%).
- TLR4-deficient mice showed no age-related dysmotility, gastrointestinal transit delay, or alterations in the myenteric neuroglial network, and exhibited increased EFS-mediated response (+40% at 10 Hz) with aging.
- Both genotypes showed age-related increases in NANC relaxation sensitive to iNOS and pan-NOS inhibitors.
- The authors conclude that TLR4 signaling influences aging-related small intestine dysfunction and myenteric neuro-glial plasticity, suggesting TLR4 as a potential target for promoting healthy aging.
- This experimental mouse study may interest researchers investigating mechanisms of age-related gastrointestinal dysfunction and enteric nervous system changes.

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