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Summary

EXPLORING POTENTIAL THERAPEUTIC IMPLICATION OF ANTI INFLAMMATORY DRUGS AND PROBIOTIC IN EXPERIMENTAL MODELS OF IBD-DEPENDENT SARCOPENIA

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A murine DSS-colitis model demonstrates that intestinal dysbiosis, elevated serum LPS, and inflammatory cytokines contribute to sarcopenia through STAT3 pathway activation, while JAK inhibitors and probiotics show potential to modulate muscle atrophy pathways in vitro.

KEY POINTS

- DSS-induced colitis in mice produced decreased body weight and muscle function, increased muscle atrophy markers (myostatin and Murf-1), upregulation of the STAT3-pSTAT3 pathway, and decreased pAKT expression.
- Colitic mice exhibited intestinal dysbiosis with reduced Roseburia and increased Sutterella, Turicibacter, and Bacteroides, alongside elevated serum LPS levels.
- In C2C12 muscle cells exposed to inflammatory cytokines, Upadacitinib and Tofacitinib reduced muscle atrophy pathways (STAT3, pSTAT3, NFκB) and increased pAKT expression, but did not suppress LPS-induced NFκB activation.
- Probiotic treatment (L. plantarum, L. rhamnosus) reduced STAT3 and pSTAT3 signals in vitro, suggesting potential positive modulation of muscle atrophy pathways.
- The authors conclude that the gut-muscle axis represents a promising research area for managing sarcopenia in IBD, with microbiota and LPS warranting consideration alongside inflammatory cytokines.

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