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Summary

SARCOPENIA AND IBD: MOLECULAR AND MICROBIAL PATHWAYS AND POTENTIAL PHARMACOLOGICAL MODULATORS IN EXPERIMENTAL MODELS OF CHRONIC INFLAMMATORY BOWEL DISEASE

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A DSS-induced colitis mouse model and in vitro myoblast studies demonstrate that gut dysbiosis, LPS, and inflammatory cytokines contribute to sarcopenia in IBD through activation of muscle atrophy pathways, with JAK inhibitors showing partial protective effects.

KEY POINTS

- The colitic mouse model exhibited reduced body weight and muscle performance, increased expression of muscle atrophy markers (myostatin and Murf-1), elevated STAT3-pSTAT3 pathway expression, decreased pAKT, gut dysbiosis, and increased serum LPS.
- In vitro treatment of C2C12 myoblasts with Upadacitinib and Tofacitinib in the presence of inflammatory cytokines (TNF-α, IFN-γ, IL-6) decreased expression of muscle atrophy pathways (STAT3, pSTAT3, NFkB) and increased pAKT expression.
- When myoblasts were treated with LPS, NFkB expression did not decrease with drug addition, suggesting that LPS activates NFkB independently of the JAK/STAT pathway and is only partially controlled by these drugs.
- The authors propose that microbiota and LPS should be considered alongside inflammatory cytokines in the pathogenesis of sarcopenia in IBD, representing a gut-muscle axis warranting further investigation.

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