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Summary

# OBEFAZIMOD INCREASES MIR-124 EXPRESSION TO CONSISTENTLY MODULATE INFLAMMATION AND FIBROSIS ACROSS PRECLINICAL MODELS

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**Obefazimod, an oral miR-124 enhancer with demonstrated efficacy in ulcerative colitis, shows consistent anti-inflammatory and anti-fibrotic activity in preclinical models of intestinal fibrosis relevant to Crohn's disease.**

## KEY POINTS

- In the chronic TNBS colitis model, obefazimod significantly reduced collagen deposition by approximately 52% with preventive dosing (day 5) and 42% with early intervention dosing (day 20), and reduced serum PRO-C6 by approximately 30% in the preventive arm ($p<0.05$).
- In human intestinal fibroblasts, obefazimod consistently reduced PRO-C3 and α-SMA expression (both $p<0.0001$), indicating inhibition of fibroblast activation and collagen production.
- RNA-seq analysis showed that TGF-β1 receptor expression, which was increased 52% under inflammatory conditions, was reduced 50% with obefazimod treatment toward control animal levels, suggesting rebalancing rather than suppression of TGF-β1 signaling.
- Anti-fibrotic effects were observed both under preventive conditions and after inflammation and fibrosis were established, indicating activity beyond inflammation control and extending to modulation of fibroblast-driven tissue remodeling.
- The findings position miR-124 as a central regulatory node linking inflammatory responses and tissue remodeling, potentially addressing fibrosis as a major unmet need in Crohn's disease where effective anti-fibrotic therapies are lacking.

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