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Summary

EMERGING ROLE OF IL-33 ACTIVATED CD73-EXPRESSING CELLS IN THE PROMOTION OF TUMORIGENESIS DURING COLITIS-ASSOCIATED COLORECTAL CANCER

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In a mouse model of colitis-associated colorectal cancer, IL-33/ST2 signaling promotes tumor development through CD73 activation, with genetic deletion or pharmacological inhibition of this pathway significantly reducing polyp formation.

KEY POINTS

- AOM/DSS-treated wild-type mice showed dramatically elevated IL-33 and ST2 expression with IL-33 localizing to colonic epithelium, stromal cells, and mast cells within polyps
- Genetic deletion of IL-33 or ST2 in mice resulted in significantly decreased polyp number and size compared to wild-type mice, with absence of tumorous lesions on colonoscopy
- RNA-Seq and qPCR revealed significant reduction of Nt5e (CD73) and adenosine pathway targets in IL-33 and ST2 deficient mice
- Genetic deletion of Nt5e or therapeutic CD73 inhibition produced significant decreases in polyp number and size similar to IL-33/ST2 deletion
- The authors interpret these findings as evidence that IL-33/ST2 axis promotes tumorigenesis in colitis-associated colorectal cancer through CD73 activation

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