gutflix
GI learning, personalised

Summary

IL-33 ACTIVATES CD73-EXPRESSING CELLS PROMOTING TUMORIGENESIS DURING COLITIS-ASSOCIATED COLORECTAL CANCER

Fabio Cominelli

UEG Week Copenhagen 2023 | Poster

IBD

October 15, 2023

In a mouse model of colitis-associated colorectal cancer, genetic deletion of IL-33 or its receptor ST2 significantly reduced polyp number and size, with RNA sequencing suggesting involvement of the CD73/adenosine pathway.

KEY POINTS

- AOM/DSS-treated Il33-/- and T1/St2-/- mice showed significantly decreased polyp number and size compared to wild-type mice, with colonoscopy revealing protruding lesions with abnormal vascular patterns in wild-type but not deficient mice.
- IL-33 and ST2 mRNA and protein expression were dramatically elevated in AOM/DSS-treated wild-type mice versus controls, with immunofluorescence localizing IL-33 to colonic epithelium and lamina propria cells consistent with stromal and mast cells.
- RNA sequencing identified significant reduction of Nt5e (CD73) and adenosine pathway targets in Il33-/- and T1/St2-/- versus wild-type mice, confirmed by qPCR on isolated polyps.
- Genetic deletion of Nt5e or therapeutic CD73 inhibition in AOM/DSS-treated mice produced significantly decreased polyp number and size versus controls.
- This experimental research may interest investigators studying inflammatory bowel disease-associated colorectal cancer pathogenesis and adenosine signaling in tumorigenesis.

VIEW THIS CONTENT ON GUTFLIX

https://gutflix.eu/search/d/c0a26d0c-743c-11ee-aecb-0242ac140004

AI-generated summary

This summary was generated by an AI large language model based on the content transcript. It is for informational purposes only and should not be considered a substitute for clinical judgment. Always rely on your professional expertise and the full clinical context when making clinical decisions.