NOTCH PATHWAY: THERAPEUTIC TARGET IN INTESTINAL FIBROSIS ASSOCIATED WITH CROHN'S DISEASE

Dolores Ortiz-Masia

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A study investigating Notch signaling in Crohn's disease-associated intestinal fibrosis found elevated expression of Notch pathway markers and senescence proteins in affected tissue, with DLL3 appearing to activate senescence in fibroblasts.

KEY POINTS

- In intestinal tissue from CD patients with complicated lesions, expression of DLL4/3, NOTCH4 and HES1 was significantly higher compared to control patients, as were levels of senescence and apoptotic markers BCL2, MCL1, P16 and P53.
- The senescence marker P16 correlated positively and significantly with DLL3/4, NOTCH3 and HES1 in intestinal tissue samples.
- NOTCH3 localized preferentially to muscular areas including muscularis mucosa, endothelium and muscularis externa, while NOTCH4 was found more specifically in mucosal crypts and lamina propria cells.
- In vitro experiments showed that DLL3, but not DLL4, significantly increased protein expression of BCL2 and P53 in intestinal fibroblasts.
- The authors conclude that the Notch pathway is involved in cellular processes essential for intestinal fibrosis pathogenesis in CD, with DLL3 having a relevant role in fibroblast senescence activation.

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