

## Poster 17 Comparison of IBD-associated proinflammatory fibroblasts with cancer-associated Fibroblasts in CRC and IBD-associated colorectal carcinomas

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### Background

Colorectal carcinomas (CRC) and chronic inflammatory bowel diseases (IBD), such as Crohn's disease (CD) and ulcerative colitis (UC), are characterized by an altered tumor and inflammatory microenvironment. In particular, cancer-associated fibroblasts (CAFs) and proinflammatory fibroblasts play a central role in tissue remodeling and inflammatory responses and may share a common role in tumor progression.

This study aims to investigate the properties and tumorigenic potential of proinflammatory fibroblasts in IBD and their similarity to CAFs in CRC and IBD-associated CRC. The goal is to characterize these cell populations regarding their proliferation and fibroblast marker expression.

### Methods

We will analyze a set of patient tissue (49 specimens), provided by the HTCR/Biobank, which includes samples from CD (n=7), UC (n=5), IBD-CRC (n=8), and CRC (n=6) patients. The tissue samples can be further grouped into "normal" (n=24), "inflamed" (n=15), and "tumor" (n=10). These samples as well as samples from the Institute of Pathology, which include IBD-CRC, matched CRC, and inflammatory human UC samples, will be analyzed by IHC staining. For an initial characterization H&E and Sirius Red staining are performed, which assesses the inflammation status, tumor localization/morphology, differentiation, and degree of fibrosis for each sample. Expression of a well-established CAF marker,  $\alpha$ SMA (smooth muscle marker), is used to assess the differentiation status of CRC samples as  $\alpha$ SMA expression increases with tumor dedifferentiation from adenoma to adenocarcinoma. The proliferative capacity of CAFs is then compared to inflammatory fibroblasts in IBD by Ki-67 staining. Fibroblast subpopulations will be grouped according to the expression of following markers: Vimentin, CD90, PDGFR $\alpha$ , and Podoplanin.

### Results

This staining strategy will help to characterize and differentiate CAFs from proinflammatory fibroblasts. We anticipate that  $\alpha$ SMA expression will be increased in pathologically altered tissue (CRC, IBD) compared to healthy tissue. Furthermore, we expect an increased expression of CD90 in inflammatory and fibrotic tissue. However, a final assessment cannot yet be made as the analysis are ongoing.

## **Outlook**

The results of this study should provide valuable insights into the role of specific fibroblast cell populations in the tumor and inflammatory microenvironment and potentially reveal new diagnostic or therapeutic targets.