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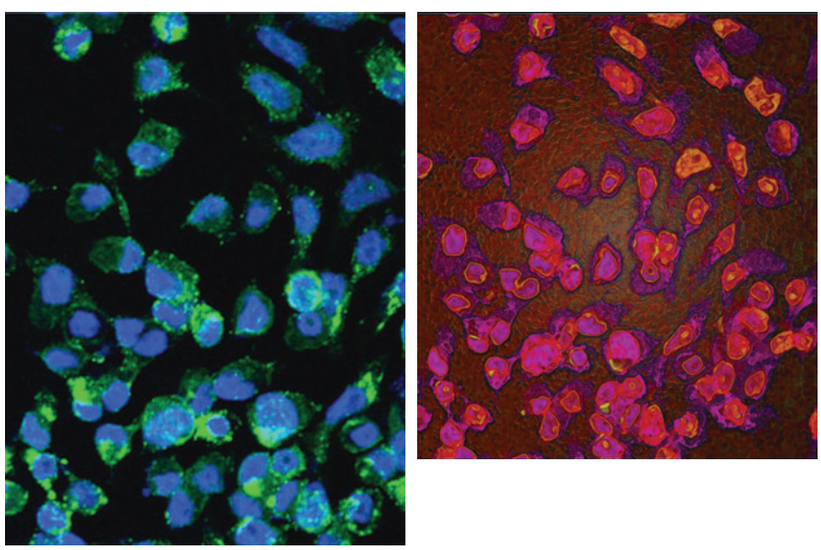
ABOUT THE COVER

Colon cancer can be initiated by certain types of chronic gut inflammation, such as ulcerative colitis. During inflammation, the glycosylation patterns of various proteins, such as the glycoprotein Mucin-1 (MUC1), in epithelial cells are stimulated to change. This altered glycosylation contributes to the development and progression of colon cancer. Kvorjak et al. investigate what causes these different glycosylation patterns and find that myeloid cells contribute to this phenomenon. M2-like macrophages produce IL13 and CCL17, stimulating colon cancer cells to express ST6GALNAC1, a glycotransferase that subsequently alters the glycan composition of MUC1. Utilizing a computational model, the authors demonstrate that IL13 inhibition is a possible therapeutic avenue for colon cancer patients by hindering glycosylation-dependent tumorigenesis. To read more, Kvorjak et al. begins on page 167. This work is also highlighted "In the Spotlight" by Vincenzo Bronte on page 160. Original immunofluorescence imaging of MUC1 on HT-29 cells cocultured with M2 macrophages by the Cascio laboratory. Artwork by Lewis Long.



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