Fusobacterium nucleatum as a Marker for Epithelial to Mesenchymal Transition in Colorectal Cancer

Huang, Angela (School: Sebring High School)

Colorectal cancer is the second most common cause of cancer death in the US, but it is curable and preventable if diagnosed early. Following the recent emphasis on the human microbiome's implications in cancer, a microbial diagnostic marker, Fusobacterium nucleatum, was investigated. CRC originates as a polyp that may undergo biochemical changes through the epithelial to mesenchymal transition (EMT), enabling it to metastasize. F. nucleatum, an abundant bacterium, is often found in gut and oral cavities and has been shown to promote colorectal carcinogenesis. F. nucleatum binds to the E-Cadherin epithelial cell surface adherence protein through adhesin fadA, which initiates EMT, inflammatory, and oncogenic responses. This project sought to assess F. nucleatum and EMT biomarkers in CRC. De-identified formalin fixed paraffin embedded tissue samples of different stages in CRC were immunostained for E-Cadherin and Vimentin as markers for epithelial and mesenchymal cells. Adhesin fadA was used as a marker for F. nucleatum. Normal tissue specimens were found to express E-Cadherin exclusively in mucosal epithelial cells, while mesenchymal cells in the stroma exclusively expressed Vimentin. Further, some precancerous and cancerous specimens showed Vimentin-positive foci within epithelial cells lacking E-Cadherin expression. Partial EMT was noted when E-Cadherin was downregulated in the tissue specimens. There was also presence of F. nucleatum secreted fadA, which plays a key role in transforming F. nucleatum from commensal to pathogenic. Therefore, its detection in the colon may aid in early CRC diagnosis and provide an opportunity for targeted intervention and personalized treatment plans to provide more predictive and detailed analysis of cancer initiation and progression.

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