THE ROLE OF MFSD2A IN THE RESOLUTION OF COLORECTAL CANCER-PROMOTING INFLAMMATION: IMPLICATIONS FOR INNOVATIVE THERAPIES

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Endothelial MFSD2A expression in colorectal cancer promotes resolution of tumor-associated inflammation through specialized pro-resolving mediators and reduces cancer cell growth in vitro.

KEY POINTS

- Transcriptomic and lipidomic analysis of human intestinal microvascular endothelial cells from CRC patients showed increased MFSD2A transcript levels, activation of resolution phase-related biological processes, increased specialized pro-resolving mediator release, and reduced pro-inflammatory lipids compared to healthy controls.
- MFSD2A silencing in CRC endothelial cells switched them to a pro-inflammatory phenotype with activation of pro-inflammatory biological processes and increased pro-inflammatory lipid release.
- Enhancement of MFSD2A expression in vitro reduces cancer cell growth in co-culture experiments with human epithelial adenocarcinoma cells.
- The hypothesis tested is that defective MFSD2A-dependent pathways in endothelial cells may fail to resolve tumor-associated intestinal inflammation, promoting CRC development, growth and metastasization.
- The findings suggest MFSD2A may offer novel therapeutic insights for CRC patient management by contrasting tumor-associated inflammation and balancing pro-inflammatory and pro-resolving environments.

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