Colorectal Cancer: Vitamin D Receptor Deficiency Upregulates Expression of Claudin 5

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In this project, the relationship between vitamin D receptor (VDR) and Claudin 5 with regards to colorectal cancer is investigated. I will compare the expression levels of the Claudin 5 protein and gene in wildtype mice and mice with the vitamin D receptor gene knocked out (no expression of vitamin D receptors) to support the hypothesis that vitamin D receptor deficiency induces increased expression of the Claudin 5 protein. Procedures: Immunofluorescence: I Deparaffinized colon sections, stained them with antibodies, and observed and compared expression levels of Claudin 5 in different sample groups. Western Blot: I culture cells, lysed them, and loaded samples into gel and run gel electrophoresis, transferred protein onto nitrocellulose membrane, incubated in antibody, and detected the protein. Real-time polymerase chain reaction (qPCR): I extracted RNA, ran reverse transcription PCR, and ran qPCR. Results showed that Claudin 5 exists in higher amounts inside mice colon belonging to VDR knockout mice compared to wildtype based off of immunofluorescence results. Results also showed that the Claudin 5 protein and gene is found to be expressed in significantly higher amounts in mouse embryonic fibroblast cells when the VDR gene is knocked out compared to wildtype mouse embryonic fibroblast cells ($p = 0.03$ for Western Blot, $p=0.008$ for qPCR). Claudin 5, which promotes the metastasis of colorectal cancer cells, is upregulated by low expression levels of VDR. It is also possible for Claudin 5 to be upregulated by low levels of VDR in colorectal cancer patients. Further research in the future concerning VDR and Claudin 5's mechanism in colorectal cancer may be done in order to eventually find a treatment and alleviate the pain of so many people in the world today.