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Cellular and molecular changes in response to Interleukin-1 β in an African American colon cancer cell line

Colorectal cancer is the third most common cancer and the third leading cause of cancer death among African Americans (AAs). When compared to Caucasian Americans (CAs), AAs present higher incidence and mortality rate, as well as worse response to 5-FU treatment and immunotherapies. Our previous findings from human genomic analysis suggest that this might be due to an increased expression of genes involved in the inflammatory response, such as Interleukin-1 β (IL-1 β). Therefore, we investigated the role of IL-1 β in promoting cell proliferation, cell migration and activation of specific inflammatory pathways in an AA colon cancer cell line (CHTN-06) and how these responses compare to an established CA colon cancer cell line (HT-29).

Methods: We analyzed cell proliferation in response to IL-1 β by MTS assay, as well as activation of transcription factors by Western Blot. A transwell assay was used to assess cell migration after IL-1 β stimuli. We analyzed cell viability following 5-FU treatment and IL-1 β stimuli to evaluate the effect of inflammation on 5-FU response.

Results: IL-1 β treatment significantly increased cell proliferation in CHTN-06 compared to HT-29. The migration rate differed between CHTN-06 and HT-29, with the CA cell line being more migratory even without stimuli; however, migration was increased by IL-1 β for both cell lines. Cell viability following 5-FU differed between the two cell lines, even after IL-1 β stimuli.

Conclusions: Our findings demonstrated an increase in cell proliferation for AA colon cancer cell line compared to CA colon cancer cell line following stimuli with IL-1 β which revealed an important role for the pro-inflammatory cytokine in driving race-specific cell growth. The transwell assay showed an increase in migration for both cell lines in response to IL-1 β stimuli. In conclusion, our results show distinct responses of colon cancer cell lines to inflammatory stimuli and illustrate a possible new target to exploit with immunotherapy.

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