Investigating the Unexplored Genome: Evaluating the Role of the Long Non-Coding RNA (lncRNA) Morrbid in the Development of Inflammatory Bowel Disease (IBD)

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Inflammatory Bowel Disease (IBD) is an autoimmune disease that develops in humans resulting from inflammation of the gastrointestinal tract. This is due to an overabundance of myeloid cells involved in innate immunity such as monocytes and other immune cells such as lymphocytes. Researchers have turned to genetics, specifically the parts of the genome that do not encode proteins or non-coding RNAs, in search of a cure. While long-noncoding RNAs (lncRNAs) have been shown to regulate immune cells, their role in the context of disease development remains unexplored. One particular lncRNA of interest is myeloid RNA regulator of Bim-induced death or Morrbid. Morrbid blocks expression of a gene called Bim that induces apoptosis of cells. Recruited for phagocytosis and cytokine secretion, myeloid cells are important components of innate immunity and thus worthy of exploration. This study focuses in particular on the function of Morrbid in a mouse colitis disease model: Dextran Sulfate Sodium Salt (DSS), utilizing two cell types of myeloid lineage: monocytes and macrophages. We also examined the expression level of Morrbid in colonic monocytes and macrophages in the same mouse model with RNA sequencing. We found that Morrbid levels are elevated, leading to lower levels of Bim in colonic monocytes and macrophages from mice with colitis. Further, through qPCR, we investigated Morrbid and Bim expression levels in whole colon cells isolated from DSS-treated mice compared to untreated mice. Collectively, this data showed Morrbid expression level changes to be prominent in myeloid cells, which begins to address how altered expression of Morrbid may contribute to IBD manifestation. Ultimately, this research could also lead to the development of a new treatment for autoimmune diseases.