Are Micrornas the Answer to Colorectal Cancer's Big Questions?

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Introduction

The question, "What determines whether a polyp will become cancerous?" was recently declared one of the five central puzzles critical to advancing colon cancer treatment [1]. The benign to cancerous transition is a critical intervention stage as tumors diagnosed in subsequent non-localized and malignant stages are exponentially more difficult to treat successfully. The decreasing mortality rate is largely credited to increased screening with sigmoidoscopy, colonoscopy, and fecal occult blood tests that result in early detection of precancerous polyps or early stage colon cancer. Despite the increased preventive screening, colorectal cancer remains a deadly disease, with an estimated 136,830 new cases and 50,310 deaths in the United States in 2014 [2]. There is both a critical need and opportunity for novel treatment strategies that either halt or prevent the transformation from polyp to cancer.

MicroRNA (miRNA) dysregulation is an established feature of colon cancer progression but it has not been clear whether this dysregulation is driving transformation and progression, or whether

has been postulated that this is due to diets dominated by processed foods that are higher in fat and sodium. High-fat diet-induced obesity increases miR-21 expression in adipose tissues where is controls the adipogenic differentiation of mesenchymal stem cells [12]. miR-21 is also one of the most consistently upregulated miRNAs across many different tumor types; its expression level correlates with cancer progression and patient prognosis [13]. It is intriguing to think that this is not mere coincidence and that it may in fact provide a direct link between the two conditions. Future studies should continue to explore the critical conserved roles that miRNAs such as miR-182 and miR-503 play in cancer development and progression. In addition, miRNAs also play a major in non-cell autonomous functions, it will be critical to evaluate the gene networks and signaling pathways that are regulated in the tumor microenvironment.

References

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