INVESTIGATING THE E-CADHERIN/β-CATENIN INTERACTION IN COLON CANCER: A PROXIMITY LIGATION APPROACH

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Background The investigation of molecular interactions in cancer pathogenesis has provided valuable insights into the underlying mechanisms driving disease progression. Among these interactions, the dynamic interplay between β-catenin and E-cadherin holds significant relevance in colon cancer. Dysregulation of the β-catenin/E-cadherin complex has been implicated in aberrant Wnt signaling, leading to uncontrolled cell proliferation, epithelial-to-mesenchymal transition, and metastasis. In this study, we aimed to investigate the relevance of the β-catenin/E-cadherin interaction in colon cancer in situ and its potential implications for disease progression.

Methods Formalin-fixed, paraffin-embedded (FFPE) tissue sections from a cohort of colon cancer patients were subjected to staining using specific antibodies against β-catenin and E-cadherin. We employed the Naveni™ proximity ligation method, a powerful technique for detecting protein-protein interactions in situ, to examine the β-catenin/E-cadherin interaction in colon cancer. This technique relies on the proximity of target molecules, allowing for the formation of a detectable signal only when they are in close proximity.

Results A variable staining pattern for the β-catenin/E-cadherin interaction among different patients was revealed. In some cases, a strong and distinct colocalization of β-catenin and E-cadherin was observed at the cell membrane, indicating a preserved interaction and intact adherens junctions. Conversely, other patients displayed a weaker or diffuse staining pattern, suggesting a disruption or reduction in the β-catenin/E-cadherin interaction. The loss or altered localization of β-catenin and E-cadherin at the cell membrane could potentially disrupt cell adhesion and promote tumor progression, proliferation, invasion, and metastasis. Furthermore, the diversity in staining pattern suggests the existence of distinct molecular subtypes of colon cancer, each with its unique characteristics and clinical outcomes.

Conclusions These findings highlight the complex nature of the β-catenin/E-cadherin interaction in colon cancer and its potential implications for disease progression. The observed heterogeneity among patients underscores the need for personalized approaches in targeting this pathway for therapeutic interventions. Understanding the dynamics of the β-catenin/E-cadherin interplay in colon cancer may pave the way for the development of personalized treatment strategies and improved patient outcomes. Future studies should focus on correlating the staining patterns with clinicopathological parameters and patient outcomes to further elucidate the clinical relevance and prognostic value of the β-catenin/E-cadherin interaction in colon cancer.

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