Subcellular localization of beta catenin in colorectal non neoplastic and neoplastic lesions

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Abstrak

Loss of adenomatous polyposis coli (APC) function is typically an early event in sporadic colorectal cancer (CRC) pathogenesis. The key tumor suppressor function of the APC protein lies in its ability to destabilize free cytoplasmic beta catenin. This lead to the accumulation of nuclear beta catenin, and together with the DNA binding protein Tcf-4, function as a transcriptional activator. Accumulation of stabilized free â-catenin is considered as an early event and perhaps initiating the process in intestinal tumorigenesis. Neoplastic transformation in the CRC associated chronic colitis is considered similar to the adenoma-carcinoma sequence in sporadic CRC. The distinguish feature from the CRC-related colitis is the difference in time and frequency changes. Loss of APC function, regarded as the beginning of a very common event in sporadic CRC, but the CRC associated chronic colitis generally occurs at the end of the dysplasia-carcinoma sequence. This research was conducted to determine the subcellular location of beta catenin expression in chronic colitis, colorectal adenomas and carcinomas that were evaluated by immunohistochemical staining. It can be concluded that beta-catenin is a component that plays a role in the development of the CRC and the subcellular location of beta-catenin can describe its oncogenic activity.