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CpG island methylator phenotype in adenocarcinomas from the digestive tract: Methods, conclusions, and controversies

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Abstract

Over the last two decades, cancer-related alterations in DNA methylation that regulate transcription have been reported for a variety of tumors of the gastrointestinal tract. Due to its relevance for translational research, great emphasis has been placed on the analysis and molecular characterization of the CpG Island Methylator Phenotype (CIMP), defined as widespread hypermethylation of CpG islands in clinically distinct subsets of cancer patients. Here, we present an overview of previous work in this field and also explore some open questions using cross-platform data for esophageal, gastric, and colorectal adenocarcinomas from The Cancer Genome Atlas. We provide a data-driven, pan-gastrointestinal stratification of individual samples based on CIMP status and we investigate correlations with oncogenic alterations, including somatic mutations and epigenetic silencing of tumor suppressor genes. Besides known events in CIMP such as *BRAF* V600E mutation, *CDKN2A* silencing or *MLH1* inactivation, we discuss the potential role of emerging actors such as Wnt pathway deregulation through truncating mutations in *RNF43* and epigenetic silencing of *WIF1*. Our results highlight the

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