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DNA methylation biomarkers and their utility for solid cancer diagnostics.

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Abstract

Cellular DNA undergoes profound changes in methylation during cancer development, with hypermethylation occurring in specific gene promoters, amidst a backdrop of generalized hypomethylation. DNA methylation in cancer often causes the silencing of tumor suppressors and other genes important for cellular growth, regulation and differentiation. Over the past two decades, there have been thousands of publications describing the methylation status of hundreds of genes in cancer, with numerous associations with clinical states, disease outcomes and therapeutic responses being reported. New methods for DNA methylation fingerprinting have emerged, allowing for the exponential growth of "epigenomic" information. Despite this wealth of data, only a handful of methylated genes are utilized as cancer biomarkers in the clinical laboratory. A literature review centered on DNA methylation in six solid cancers was performed, including colorectal, pancreatic, prostate, bladder, breast and ovarian. Commonly methylated genes in the six cancer types were identified and catalogued, and could serve in the future as tissue-based biomarkers or as part of cancer-specific panels. Perhaps more importantly, this endeavor has also focused on methylated genes that appear to be unique to particular cancers. These genes may be more versatile for clinical use, with blood or urine-based cancer screening becoming a reality.