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Genetic alterations of histone methylation modifiers and their significance in breast cancer

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Methylation of lysine and arginine residues on histones and non-histone proteins plays critical roles in chromatin function, transcriptional regulation, genomic stability, cell differentiation and survival. These epigenetic methylations are mediated by antagonistic sets of enzymatic complexes—the methyl transferases, which catalyze methylation in a site-specific manner and the demethylases, which remove the methylation marks. Such methylation marks are interpreted by "reader" proteins that specifically bind to the modified histone. The largest and most diverse set of reader proteins includes the Tudor domain and plant homeodomain (PHD)-containing proteins. Accumulating evidence suggests that histone methylation pathways play an integral role in the sequential progression of cancer. It is shown that many histone methyl transferases/demethylases and "readers" are targeted for mutation and deregulation in cancer patients. However, the genomic landscape and clinical significance of these histone methylation modifiers in breast cancer remain poorly characterized. Here, we performed an integrated genomic and transcriptomic analysis of histone methyl transferases/demethylases and Tudor-containing "reader" proteins in breast cancer. We identified associations among recurrent copy number alterations, gene expressions, clinicopathological features and survival of patients. Furthermore, we interrogated cancer genomics data and functional small-interfering RNA (siRNA) screens to pinpoint potential oncogenes and novel targets, focusing on histone methylation modifiers in breast cancer. Integrative analysis identified a subset of histone methylation modifiers that are dysregulated by genetic alterations, classifying them as candidate therapeutic targets. Together, our findings provide a strong foundation for further mechanistic research and therapeutic options that target these histone methylation modifiers to treat breast cancer.

Biography

Zeng-Quan Yang has completed his PhD from Tokyo Medical and Dental University and Post-doctoral studies from University of Michigan School of Medicine. Currently, he is an Associate Professor at the department of oncology, Wayne State University. His research interest is focused on investigating the genetic and epigenetic bases of human breast cancers and identifying novel therapeutic targets for cancer treatment. He has published more than 30 papers in reputed journals.

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