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NOVEL DNA METHYLATION IN GBM

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Genetic drivers of cancer can be disregulated through epigenetic modifications of DNA. Although the critical role of DNA 5-methylcytosine (5mC) in the regulation of transcription is recognized, the functions of other non-canonical DNA modifications remain obscure. Here, authors report the identification of novel N6-methyladenine (N6-mA) DNA modifications in human tissues and implicate this epigenetic mark in human disease, specifically the highly malignant brain cancer glioblastoma. Glioblastoma markedly up regulated N6-mA levels, which co-localized with heterochromatic histone modifications, predominantly H3K9me3. N6-mA levels were dynamically regulated by the DNA demethylase ALKBH1, depletion of which led to transcriptional silencing of oncogenic path ways through decreasing chromatin accessibility. Targeting the N6-mA regulator ALKBH1 in patient-derived human glioblastoma models inhibited tumor cell proliferation and extended the survival of tumor bearingmice, supporting this novel DNA modification as a potential therapeutic target for glioblastoma. N6-mA also response to hypoxia stress and hypoxia respond genes were regulated by ALKBH1. Collectively, author's results uncover a novel epigenetic node in cancer through the DNA modificationN6-mA.

BIOGRAPHY

Tao P Wu has completed his PhD in 2008 from University of Chinese Academy of Sciences, China. He is the Assistant Professor of Baylor College of Medicine, USA. He has over 20 publications including one Nature article and one Cell article and his publication H-index is 9.

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