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Jiangwen Zhang

University of Hong Kong, Hong Kong

Identification of DNA methylation of distal regulatory regions with causal effect on tumorigenesis

Aberrant promoter methylation is a common mechanism for tumor-suppressor inactivation in cancer. However, the exact role of DNA methylation at enhancers remains to be elucidated. We have developed a set of tools to genome-wide identify DNA methylation in distal regions with causal effect on tumorigenesis. Novel oncogenes/tumor-suppressors and their putative enhancers can be identified together based on this strategy. Many predictions were directly demonstrated by dCas9-based epigenetic editing with strong evidence to support the accuracy and efficiency of our tool. Our study reveals the prevalent regulation of genome-wide putative enhancers by DNA-methylation with causal effect on cellular malignancy and patient survival. Mechanistically, oncogenic and lineage-specific transcriptional-factors aberrantly shaped the methylation landscape with diverged tumor-subtype core regulatory circuitry. Notably, the gene regulatory networks orchestrated by enhancer methylation across different cancer types converged on a common architecture, highlighting general organization principle for such networks regulated by DNA methylation of distal regulatory regions.

Biography

Jiangwen Zhang graduated from Johns Hopkins University with a PhD. He has worked at Harvard University Genome Centre as Senior System Biologist for years before joining the University of Hong Kong in 2013. His lab has broad interest in genetic and epigenetic regulation in development and diseases. Currently, his lab is focusing on epigenetic regulation of tumorigenesis. His lab employs high through-put "omics" assays and large scale computation to dissect the gene regulatory network and signaling pathways involved in oncogenesis.