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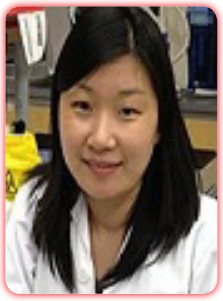

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Understanding RNA editing in human cancer: Causes and functional consequences

Conventionally, cancer is driven by a clonal accumulation of somatic mutations, referred to as driver mutations, conferring a selective growth advantage to cancer cells. RNA editing, is an epigenetic mechanism, introduces changes in the RNA sequences encoded by the genome, contributing to editing/epigenetic mutations. In humans, the most frequent type of editing is the conversion of adenosine to inosine (A-to-I), which is catalyzed by ADAR (Adenosine Deaminase Acting on RNA) proteins, ADAR1 and ADAR2. Inosine (I) essentially mimics guanosine (G), therefore ADAR proteins actually introduce a virtual A-to-G substitution in transcripts. Such changes can lead to specific amino acid substitutions, alternative splicing, altered microRNA seeds or targets, or changes in transcript localization, expression and degradation. Up to now, changes in the information are being investigated almost exclusively at the DNA level. Using integrative genomic approaches, our previous study highlighted a link between a disrupted RNA editing balance and cancer development. We recently place focus on understanding the regulators of A-to-I RNA editing and their role in cancer development.

Biography

Polly Leilei Chen is an Assistant Professor in the Department of Anatomy and Principle Investigator at the Cancer Science Institute of Singapore, National University of Singapore where she directs a research lab studying human cancers, particularly liver cancer. Her current focus is on the identification of key RNA editing events and translating these findings into diagnoses and even treatment. In addition, she has collaboration with local and international researchers to study the roles of RNA editing enzyme ADARs and their substrates in different types of human cancers, such as esophageal squamous cell carcinoma (ESCC), gastric cancer and acute myeloid leukemia.

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