Increasing Coding Complexity of Human Genome at RNA Level

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Abstract
The number of human coding genes is surprisingly small given the biological complexity of the organism, suggesting additional layers of regulation to increase its coding complexity. We found that at the coding complexity of human genome can be increased by multiple pathways at RNA level. First, the majority of human genes undergo alternative splicing to produce multiple isoforms with distinct functions, which is a major mechanism to increase proteome complexity of human genome. This process is tightly regulated, and misregulation of splicing is closely associated with various human diseases. We have been focused on systematic study of splicing regulation, as well as the dysregulation of alternative splicing in cancers. We identify and study the regulatory cis-elements and trans-acting splicing factors, and seek to assemble such information into a predictive “splicing code” to help us understand how the alternative splicing is controlled in different cell types and different disease stages. In addition, the mRNA translation can be regulated to produce multiple peptides from single mRNA. By using circRNA as a model, we have studied the non-canonical RNA translation and the regulation and new function of circular RNAs. We found that a large fraction of circular RNAs can function as mRNA to code for proteins, and the translation of circRNA can be driven by diverse sequences to produce additional translation isoforms. These findings provided molecular mechanisms to expand the coding capacity of human genome at RNA levels.

About the Speaker
Zefeng Wang received his bachelor degrees with double major in Biological Science and Computer Technology from Tsinghua University and master degree in Molecular Biology from Institute of Biophysics, CAS. He received his PhD degree at Johns Hopkins Medical School, US, and subsequently worked a Damon Runyon fellow at Massachusetts Institute of Technology. He became an assistant professor at University of North Carolina at Chapel Hill in 2007 and was promoted as associate professor with tenure in 2013. In 2015, he moved to Shanghai and became the director of Partner Institute for Computational Biology, a partner institute jointly run by Chinese Academy of Science and Max-Planck society. Zefeng’s research focuses on the regulation of gene expression in RNA level. His lab has developed a series of genomic approaches to study RNA splicing regulation in a systematic fashion, and developed artificial proteins to specifically manipulate RNA metabolism. He has patented the technology of the “artificial site-specific RNA endonuclease”, which can be used as a research tool or potential therapeutic reagents. He has published >60 research papers that were cited >6000 time, and his work was recognized by many research awards, including RNA Society/Scaringe Young Scientist Award, Alfred Sloan Research Fellow, Beckman Young Investigator, Kimmel Scholar, Jefferson-Pilot Fellowships in Academic Medicine, Max-Planck Fellow, Mercator Fellow, CAS pioneer hundred talents program (type A), etc.

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