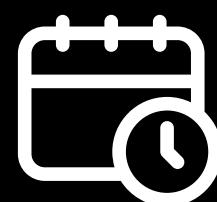


# **MASSIVE SWITCH IN ALTERNATIVE POLYADENYLATION (APA) AS A NOVEL REGULATORY LAYER OF INNATE IMMUNITY**



**Monday 5 August 2024**  
**10.30am (SGT , GMT+8)**



**Via Zoom**



## **About the speaker**

Xiang-Dong Fu received his B.S. degree of Virology from Wuhan University in 1982. He was the first class of the CUSBEA (China-United States Biochemistry Examination and Application) for graduate training. He obtained his Ph.D. degree from Case Western Reserve University in 1988 and completed postdoctoral training at Harvard in 1988. In 1992, he joined the faculty at University of California, San Diego and rose through the rank. He became a Distinguished Professor of Cellular and Molecular Medicine in 2018 at UC, San Diego. Since January 2023, he joined Westlake University, Hangzhou, China as Chair Professor in RNA Biology and Regenerative Medicine.



**Dr. Xiang-Dong Fu**  
Chair Professor in RNA Biology and  
Regenerative Medicine  
Westlake University

## **About the seminar**

Alternative polyadenylation (APA) is a pervasive mechanism to generate mRNA isoforms that have been linked to diverse biological processes. To understand how APA is regulated in mammalian cells, we perform a high throughput screen by high throughput sequencing (HTS2) based on hundreds of endogenous APA events in HeLa cells. This systematic effort reveals key roles of specific cleavage factors (CFs) and cleavage and polyadenylation specificity factors (CPSFs), but not cleavage stimulating factors (CstFs) in APA regulation. We also detect broad participation of specific transcription factors, splicing factors, and RNA export factors in APA regulation, suggesting strong influence of both upstream and downstream gene expression processes on the selection of alternative APA sites. Most strikingly, we found that a large of genes subjected to APA regulation are part of the innate immunity, suggesting that APA regulation serves a key regulatory strategy to fine-tune the immune response to pathogen infection without causing adverse autoimmunity.

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