Singapore Immunology Network
SIgN

SIgN SEMINAR

Hosted by Prof LAM Kong Peng

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Decoding TNFR-driven Long Non-Coding RNA Functions to Design New Therapeutics for Treatment-Refractory Cancers

Inflammation-driven signalling networks reprogram the epigenome and transcriptional states of cancer and various diseases. Among these pathways, tumour necrosis factor receptor (TNFR) signalling—particularly the TWEAK/Fn14 axis and NF-κB pathway—emerge as potent drivers of tumour aggressiveness, therapeutic resistance, metabolic dysfunction, and metastasis. Yet, the molecular effectors that translate TNFR signalling into disease-specific gene expression programs remain poorly understood. Increasing evidence suggests that long non-coding RNAs (*lncRNAs*) and their associated RNA-binding proteins, as well as epigenome reprogramming play critical roles in shaping these pathogenic transcriptional states. We have identified an oncogenic *lncRNA*, *PLUM*, which is activated by NF-κB through the epigenome and mediates chemoresistance in multiple myeloma by interacting with EZH2, the catalytic subunit of Polycomb Repressive Complex 2. Interestingly, a distinct malignant plasma cell population displaying elevated *PLUM*-regulated gene signature is observed in relapsed multiple myeloma patients. These plasma cells display enhanced oxidative phosphorylation, metabolic and stress-adaptation programs, along with the suppression of interferon and inflammatory responses. Our findings support a model in which *PLUM*-associated programs contribute to a sustained, therapy-resistant malignant cell state. Hence, targeting *PLUM*-EZH2 interactions may represent a clinically potent strategy for the treatment of relapsed, refractory cancers harbouring aberrant EZH2 activity.

15 January 2026 (Thursday)
10 – 11 AM (Singapore Time)
SIgN Seminar Room
8A Biomedical Grove, Immunos, #04-06
Singapore 138648

Seminar is open for all to attend.

Registration is not required.