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Metabolic changes regulate the protumorigenic effects of mitotic slippage-induced senescence

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Hosted by Dr Andy Tan

Seminar Abstract

Anti-mitotic drugs are the most-commonly utilised class of chemotherapeutic agents that are administered as first-line therapy; however, their clinical success has been impeded by chemoresistance and disease relapse. To formulate a treatment that achieves complete remission of the disease, it is critical to gain a complete understanding of the cellular pathways underlying this escape from antimitotic drug-induced cell death, also known as mitotic slippage. Mitotic slippage describes a phenomenon where cells escape antimitotic drug-induced mitotic arrest/death and "slip" into interphase without proper chromosome segregation and cytokinesis. In this study, we found that multinucleated post-slippage cells undergo senescence and elicit paracrine pro-tumourigenic effects, both *in vitro* and *in vivo*. Further investigation into potential senescence effectors revealed two major metabolic pathways, autophagy and lipid metabolism, that could abrogate the tumour-promoting effects of antimitotic therapies. Our discovery of eliminating post-slippage senescent cells through metabolic alterations could potentially lead to treatment that circumvent antimitotic drug resistance and enhance the treatment efficacy for cancer patients.

About the Speaker

Alex Wong recently graduated from Interdisciplinary graduate school, NTU, where he performs research on cancer biology and therapeutics. Apart from work, Alex likes to spend time with his family and friends to travel around the world. He also enjoys hiking and baking on his free time.