Role of SRPK1-modulated Alternative Splicing in Cisplatin Resistance in Breast Cancer Cells

For PhD Qualifying Examination (PQE) “Oral Component” – Open Seminar

THURSDAY
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10AM – 10.45AM

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Chemo-drugs such as Cisplatin usually becomes ineffective after the prolonged use in initially responsive patients through initiating cellular defense mechanisms that reduce the cytotoxicity. A growing body of evidence has suggested that aberrant alternative splicing could lead to chemoresistance. Serine/Arginine-rich splicing factors (SRSFs) and the upstream kinases, SRPKs could be involved in the acquired chemoresistance to cisplatin in cancer cells. In addition to their expression levels, it was noticed that post-translational modifications (PTMs) of SRPKs and SRSFs, including phosphorylation and acetylation presented to be important determinants of specific relevant splicing events. Therefore, this study aims to elucidate how PTMs of SRPKs and SRSFs modulate pro-apoptotic and anti-apoptotic alternative splicing events that could confer drug resistance to cancer cells.