Abstract

The mTOR Complex 1 (mTORC1) signaling pathway has evolved to sense and respond to cellular energy status, nutrient availability and surrounding oxygen concentrations. In addition, mTORC1 can be further activated by mitogen-and hormone-activated AGC kinases including Akt and RSK, and suppressed by S6K1 via a variety of negative feedback loops. The integration of these multiple inputs controls the strength and duration of downstream signaling, which is important in differentially regulating mTORC1-dependent processes such as protein synthesis and cellular metabolism.

Dr Blenis will discuss how mTORC1 and S6K1 regulate aspects of nutrient metabolism, mRNA metabolism and protein production - processes critical to the control of cell growth. He will also provide evidence that targeting nutrient metabolism in cancer cells with activated mTORC1 may provide an important therapeutic opportunity.

About the Presenter

Professor Blenis’ research is defining how altered cell communication networks promote carcinogenesis. He has made seminal contributions to our understanding of the cell signalling pathways that control cellular growth, metabolism and movement and how these pathways are perturbed in the development and progression of human cancers. Among his many achievements, he has established that activation of the serine/threonine kinase S6 is blocked by the mTOR inhibitor rapamycin, a drug that is now in several clinical trials for cancer.

His honors include ACS Junior Faculty award, AHA Established Investigator award, the LAM Foundation Established Investigator award and the NIH/NCI MERIT award. Dr Blenis is the newly appointed Kellen Professor of Cancer Research in the Department of Pharmacology at the Meyer Cancer Center, Weill Cornell Medical College.

This Biomedicine Discovery Lecture is part of the Cancer Cell Signalling Mini-Symposium.

For more information and free registration: www.med.monash.edu/research/mini-symposium.html

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