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Inhibition of LIM kinase inhibits cancer growth

Juliana Antonipillai[1, 2], **Rong Li1** and **Ora Bernard**[1]
[1]St Vincent Medical Research Institute, Australia
[2]Victoria University, Australia

LIM kinase (LIMK) regulates actin cytoskeleton via inactivation of an actin depolymerising factor cofilin and involves in cell motility, invasion and migration. Metastasis is the major cause of death in cancer patient and the factors that regulate migration and invasion considered to be a good therapeutic target for metastatic disease. We investigated the consequences of LIMK inhibition on growth and metastasis of humans and mouse cell lines and tissues with number of LIMK inhibitors. LIMK activity was reduced in tumor cells by expression of dominant-negative LIMK1 by RNA interference or with a selective LIMK inhibitor. The extent of phosphorylation of the LIMK substrate, cofilin of proliferation and invasion in 2D and 3D culture and of tumor growth and metastasis in mice were tested. Inhibition of LIMK activity efficiently reduced the pro-invasive properties of tumor cells in vitro. Tumors expressing dominant-negative LIMK1 grew more slowly and were less metastatic in mice. Our findings with number of cell permeable LIMK specific inhibitors and SiRNA suggest that LIM kinase functions as a signaling node that controls actin dynamics. LIM kinase may therefore represent a targetable enzyme for cancer treatment.

Biography

Juliana Antonipillai is a lecturer in the College of Health & Biomedicine. She specialises in cancer and cardiovascular biology. Prior to joining Victoria University, Juliana was an academic researcher for 12 years at several medical research institutes and hospitals in Melbourne and the USA. In the last five years her research has focused mainly on how LIMK regulates platelet function in humans and mice, and on the role of LIMK in cancer metastasis, using a variety of small molecule LIMK inhibitors. She demonstrated that these inhibitors inhibit the proliferation of human breast cancer cells in 2D and 3D cultures. She also contributed to a collaborative study showing the effect of a new LIMK inhibitor drug, Pyr1, on cancer cell growth in vitro and in mice.

Juliana.Antonipillai@vu.edu.au

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