

CONFÉRENCE

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“Metabolic control by the LKB1-AMP-activated protein kinase (AMPK) pathway: implications for immunity.”

The ability of a cell to successfully grow and divide depends on the cell having enough energy to complete the task. However, part of the challenge that faces proliferating cells, such as antigen-stimulated T cells, is that they require increased amounts of fuel and energy to meet the demands of uncontrolled growth. T cells unable to adapt to these changes undergo apoptosis. Our laboratory is focused on identifying the molecular mechanisms by which T cells control their metabolism in response to activation and stress. Through this we have identified a novel role for the LKB1-AMP-activated protein kinase (AMPK) signaling pathway in the control of T cell development, activation, and metabolism. AMPK is a highly conserved serine/threonine kinase complex that functions to tether the bioenergetic state of a cell to numerous critical biological functions including translational control, mitochondrial function, cell polarity, and proliferation. T cells lacking the upstream kinase for AMPK, LKB1, display dramatic defects in thymocyte development, peripheral T cell homeostasis, and proliferation. Notably, the ability of T cells to regulate their normal cellular metabolic programs is disrupted in the absence of LKB1 or AMPK. We will discuss these results and the implications of LKB1-AMPK signaling for proper energy homeostasis and lymphocyte function.

Jeudi 3 février 2011 à 11 h 30
Pavillon Claire McNicoll, salle Z-300

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