Department of Biology presents a seminar

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"Bcl-2 family proteins: a direct regulatory link between cell survival and metabolism?"



It is now well established that shifts in energy metabolism are associated with cancer development and progression. The most studied of these phenomena is the Warburg effect, which corresponds to an increase of anaerobic glycolysis *vs* mitochondrial oxidative phosphorylation to produce energy for cellular processes. However, the mechanisms related to these metabolic switches are still a matter of debate. Bcl-2 family proteins contain both pro-(e.g. Bax), and anti-apoptotic (e.g. Bcl-2 and Bcl-xL) members which are respectively encoded by tumor suppressors and proto-oncogenes. Up-regulation of the anti-apoptotic proteins Bcl-2 has been associated with Non-Hodgkin's lymphoma; and certain studies suggest that Bcl-2 plays a role in the regulation of energy metabolism. However, the molecular players involved in this regulation are still to be defined. We recently observed that Bcl-2 or Bcl-xL overexpression led to a significant increase of lactate production rates in a mouse pro-lymphocyte B cell line. This phenomenon was associated with a stimulation of the lactate dehydrogenase (LDH) enzyme specific activity; and an increase of the expression of the LDH-A subunit. Also, this phenotype was strongly attenuated if a Bcl-2 mutant of interaction with Bax (Bcl-2-G145E) was overexpressed instead of native Bcl-2. These data suggest that Bcl-2/xL expression levels may play an active role in stimulation of lactic fermentation commonly observed in blood cancer cells; and that this effect may depend on the ability of these anti-apoptotic proteins to physically interact with Bax.

## Friday, May 1, 2015 3:00 – 4:00 PM Science 2, room 109

For further information: <u>www.csufresno.edu/biology</u>. If you need a disability-related accommodation or wheelchair access, please contact Lindasue Garner at the Department of Biology at 278-2001 or e-mail <u>lgarner@csufresno.edu</u> (at least one week prior to event).