

ORAL PRESENTATIONS

Seal Herpes Impacts on the Diving Physiology of the Harbor Seal, *Phoca vitulina*

Primary Contributor: Pamela Allen, Department of Zoology

Faculty Sponsor: Wayne Goodey, Department of Zoology

Location: Room 1330, LSC Time: 1:20pm-1:40pm

Harbor seals depend on their good diving capability to catch food and evade predators. This excellent diving ability is a result of their interesting physiology. A possible challenge to diving, however, may be a disease called phocine herpesvirus. This disease has recently come to the Pacific Coast harbor seal population and can lead to repeated infections in an animal. I hypothesize that harbor seals that have experienced extreme infections may have decreased diving performance, thus limiting their chance at finding food and potentially leading to population decline. By pooling years of scientific research from around the world, I have outlined the major physiological processes that occur in a harbor seal during a dive and hypothesize how these processes may be affected by a herpesvirus infection.

The effect of changing the physico-chemical properties of turret residue 462 on C-type inactivation in hKv1.5.

Primary Contributor: David Chiang, Cellular and Physiological Sciences

Co-Contributor: Cyrus Eduljee, Cellular and Physiological Sciences

Faculty Sponsor: Dr. Steven J. Kehl, Cellular and Physiological Sciences

Location: Room 1330, LSC Time: 1:45pm-2:05pm

This study investigated the importance of the turret (outer pore mouth) of voltage-gated potassium channel, hKv1.5, in C-type inactivation. Using the whole-cell voltage clamp technique, mutant channels hKv1.5 T462C expressed in HEK293 cells were treated with solutions containing varying concentrations of propyl-MTS. Before treatment, the inactivation of T462C was faster ($\tau = 479\text{ms}$; $n=3$) than that of the wild type channels ($\tau = 1183\text{ms}$; $n=3$). After treatment, the rate of inactivation was further increased. Additionally, propyl-MTS irreversibly reduced the maximum current passed by the mutant channels by about 80%. These findings show that changes in the volume of residue 462 speed up inactivation and block current, suggesting that the turret of hKv1.5 plays an important role in C-type inactivation and channel gating in general.

A study to determine the efficiency of wire basket traps for collecting mountain pine beetle (*Dendroctonus ponderosae*) on pesticide-treated trees

Primary Contributor: Sam Coggins, Forest Sciences.

Faculty Sponsor: Dr. John McLean, Forest Sciences.

Location: Room 1330, LSC Time: 2:10pm-2:30pm

Wire basket traps are attached to the lower stem of trees and used to assess beetle mortality when evaluating protective pesticides. A trap with a 20 cm opening was constructed in the laboratory and assessed for catch efficiency. Five groups of fifty beetles were dropped from four sides of the tree at three heights. The results were analyzed using two-way ANOVA and showed high catch efficiency at the lower and mid-heights, becoming less efficient at the tallest height. Any beetles falling outside the trap were counted and their distance measured perpendicular to the trunk. An estimate of the effectiveness of a 30 cm opening was also made, and showed an increase from in catch efficiency from 96% to 98% when compared to a 20 cm opening.

The Role of Integrin-linked Kinase (ILK) in Integrin-Mediated Signaling

Primary Contributor: Christine Parachoniak, Department of Physics and Astronomy

Co-Contributor: Virginia Gray, Cancer Biology, Jack Bell Research Centre

Faculty Sponsor: Dr. Shoukat Dedhar, Department of Biochemistry and Molecular Biology

Location: Room 1330, LSC Time: 2:55pm-3:15pm

Integrins, like their name implies, are important proteins that bind or "integrate" the inside of each cell to its extracellular environment. Integrins, however, are not merely clamps but also act as receptors which are able to communicate information from the outside of the cell inwards or vice versa. One of the intracellular proteins thought to be involved in mediating some of the signaling pathways is a protein called integrin-linked kinase (ILK). Recent evidence indicates that abnormal levels of, or mutations in, ILK may cause cellular transformations contributing to cancer. This study aims to elucidate how ILK interacts with integrins by activating and inhibiting steps in the signaling pathway and monitoring the results.