Undergraduate Research Seminar
Wednesday, June 4th, 2014 5:30 p.m.
Leigh 309

Brendan Regnery

"A Study to Enhance the Sensitivity for the Discovery of the Higgs Boson Coupling to Dimuons"

On July 4th 2012, the Higgs boson was discovered at the Large Hadron Collider (LHC) at the European Organization for Nuclear Research (CERN). The University of Florida is a participant in the Compact Muon Solenoid (CMS) Experiment—one of the two experiments at CERN credited with the discovery. Now that the Higgs has been discovered, researchers must confirm if it behaves exactly as predicted in the Standard Model of particle physics. We are contributing to that process by searching for the Higgs decay to dimuons, with associated production of a W or Z vector boson. If discovered, the measurement of the branching ratio for this decay can confirm the predicted Higgs coupling to dimuons. In order to optimize the search for this channel, we improved upon the current Higgs to dimuons analysis of CMS by adding additional discrimination criteria: invariant mass of the dijet decay of the W or Z vector boson, dimuon transverse momentum, the angle between the dimuon and dijet systems in the transverse plane, and missing transverse momentum. Our overall improvement in the sensitivity for this process (calculated using a signal/sqrt(background) ratio) was estimated to be a factor 1.8. In this seminar, I will explain what the Standard Model is, what the Higgs boson is, how the LHC works, how we obtained our results from simulated samples, and how our results will be used.

Brandon Lam

"Dysregulated Cytokine Production by Leukocytes, Mediated by SOCS1 Deficiency, is Correlated to Skin Pathology: Implications for Therapeutic Targeting"

Although dysregulated immune cell activation and cytokine signaling are known to contribute to autoimmune disease, ways to target and prevent these abnormalities are limited. Notably, an intracellular protein, Suppressor of Cytokine Signaling-1(SOCS1) is important in the regulation of both immune cell activation and cytokine signaling. Mice lacking expression of SOCS1 (SOCS1-/-) exhibit dysregulated immune function characterized by enhanced pro-inflammatory cytokine production and decreased lifespan. It has been demonstrated that treating these mice with a peptide that mimics SOCS1 function, SOCS1-KIR, significantly improved survival. To add, in vitro studies show that pro-inflammatory cytokine production by immune cells in SOCS1 deficient mice was also ameroilated upon SOCS1-KIR treatment alone. One regulatory mechanism by which SOCS1-KIR extended survival of SOCS1-/- mice is through the down regulation of interferon-gamma (IFN□) production. To further investigate the role of IFN□ in this process, SOCS1 x IFN□ knockout mice were studied. In comparison to SOCS1-/- mice, SOCS1-/- IFN□-/- mice do not develop lethal inflammatory disease. However, SOCS1-/+ IFN□-/- mice display dysregulated immune function in the form of skin pathologies, and dysregulated IL-17 and IL-2 cytokine production. Preliminary hematoxylin and eosin (H&E)

stains of skin pathologies reveal increased numbers of neutrophils, mast cells, and hemorrhage points in the area. In addition, hyperproliferation and accumulation of lymphocytes and splenocytes are noted in vitro. Following treatment with SOCS1-KIR, flow cytometry, direct count, and ELISA demonstrated restored IL-2 production, and reductions in proliferation. Together these results implicate an important role of SOCS1 in the regulation of immune responses. Moreover these results suggest that targeting SOCS1 deficiency, by a mimetic of SOCS1, may have implications in the regulation of skin pathologies mediated by immune dysregulation.