

Undergraduate Research Symposium May 18, 2012 Mary Gates Hall

Online Proceedings

POSTER SESSION 1

Balcony, Easel 96

12:00 PM to 1:30 PM

Cytokine Dynamics in Transgenic Tg338 Mice Challenged with Scrapie

Ke (Kate) Fan, Senior, Biochemistry

Mentor: Denny Liggitt, Comparative Medicine

Mentor: Gina Kiske, Comparative Medicine

What the media has termed as Mad Cow disease is a form of Transmissible Spongiform Encephalopathy (TSE). It is also known as a prion disease and occurs when the normal prion protein in the brain misfolds. This is thought to induce other prion proteins around it to misfold, leading to brain degeneration. Scrapie is a form of TSE in sheep; primarily infecting the central nervous and lymphoreticular systems. There is currently little explanation for why the process occurs, although, studies show that cytokines may be involved. Scrapie will not readily cause disease in normal mice. However, transgenic mice can be infected. These transgenic mice have a prion gene knockout with an insertion of the sheep version of the prion gene, which makes them susceptible when directly infected with scrapie prions. To further our understanding of prion diseases, we aimed to learn more about its source material, transmission, host genetic markers, and immune response with the help of a transgenic mouse model. We challenged transgenic mice with scrapie via the intracerebral route and observed them for signs of disease. Certain tissues were collected and analyzed at multiple points of infection for infectivity, potential co-localized proteins, cytokine dynamics, and growth factors. Various cytokine levels could increase, decrease, or stay the same throughout the infection period. This will enable a better characterization of immune response to prion disease and potentially provide a measure of disease progression using blood cytokine levels.

SESSION 2B

HOST AND PATHOGENS

Session Moderator: Geoffrey Gottlieb, School of Medicine

Mary Gates Hall 231

3:30 PM to 5:00 PM

* Note: Titles in order of presentation.

Immune/Lymphatic Interactions in the Lymph Node

Neela Ramanujam, Senior, Biology (Molecular, Cellular & Developmental)

Mary Gates Scholar

Mentor: Alanna Ruddell, Comparative Medicine

The lymphatic system is essential to fluid and cell transport, immune function, and tumor metastasis. Lymph drains from body extremities into lymph nodes, via lymphatic vessels, to present antigens to lymphocytes. This can result in an immune response. The existence of tumor cells in lymph nodes is a sign that cellular metastasis has occurred from the primary tumor. I am studying lymphangiogenesis, a process in which lymphatic vessels and sinuses grow and dilate in response to antigens or tumors, thus increasing the lymph flow and capacity to transport tumor cells in lymph nodes. Recently an antibody, 10.1.1, has been discovered to recognize an antigen that is selectively and highly expressed in lymphatic endothelium. The 10.1.1 antibody specifically induces lymph node lymphangiogenesis. This binding also induces proliferation of an unknown cell type in the lymph node. In comparison to the 10.1.1 antibody, injecting a nonspecific antibody control has no effect on lymphatic sinus growth. I am therefore studying how the 10.1.1 antibody rapidly induces lymphatic sinus growth, as well as what cell types contribute to making the new sinuses. I am using the technique of immunostaining to study the effects of the 10.1.1 antibody. By taking a frozen slice of lymphatic tissue and staining it with various antibodies to detect proliferating cells and their cell type, I can identify the specific kinds of cells that are forming into lymphatic sinuses. An understanding of how these lymphatic sinuses grow could help generate therapeutic strategies for manipulation of the lymphatic or immune responses. By inhibiting sinus growth and thus limiting lymph flow, it could be possible to prevent the metastasis of cancer.