Online Proceedings

Session 1I

Multidisciplinary Approaches to Medical Research

Session Moderator: Gwenn Garden, Neurology
MGH 248
12:30 PM to 2:15 PM
* Note: Titles in order of presentation.

Quantitative Analysis of Microvasculature and Structural Changes in Uveitis using Optical Coherence Tomography
Jasmine Christina Vu, Senior, Bioengineering
Mary Gates Scholar
Mentor: Ruikang Wang, Bioengineering
Mentor: Zhongdi Chu

As one of the five leading causes of blindness, uveitis is the reoccurring inflammation of the uvea, demonstrating pathological changes in the eye. In recent years, optical coherence tomography (OCT) has been recognized as the leading, clinically accepted imaging modality for diagnosing major human optical diseases. Despite this, research on the clinical usage of OCT, primarily spectral domain OCT (SD-OCT) and swept source OCT (SS-OCT), for uveitis diagnosis remains sparse due to the lack of a quantitative-based set of parameters to assist with OCT image analysis. As a result, there is a need to develop an index of parameters that quantifies the microvasculature and structural changes associated with uveitis. To address this need, a novel five parameter quantitative-based metric consisting of distance of retinal detachment, retinal thickness, vessel area density, vessel diameter, and vessel perimeter was evaluated. Through layer segmentation of SD-OCT and SS-OCT scans, application of optical microangiography, and quantitative analysis of structural and microvasculature changes for healthy and uveitis cases, the clinical potential of SD-OCT and SS-OCT for diagnosing uveitis was evaluated. This project introduced a metric for evaluating changes associated with uveitis in a qualitative and quantitative manner to further understand the abnormalities that accompany the disease. Assessing the clinical efficacy of SD-OCT and SS-OCT for uveitis detection can provide insights on the most effective method for diagnosing this disease.

Poster Session 3

MGH 241, Easel 139
2:30 PM to 4:00 PM

Efficacy of Mutant Ad3 Fiber Knob in Opening Cellular Junctions and Oncolytic Virotherapy
Mike (Michael) Liu, Junior, Pre-Health Sciences
Mentor: Hongjie Wang, Medical Genetics
Mentor: Andre Lieber, Medicine

Oncolytic adenoviruses such as serotype 5 adenoviruses (Ad5) selectively infect and destroy cancer cells and are widely used in preclinical and clinical trials for gene therapy. However, Ad5 has many downsides including preexisting anti-Ad5 immunity in humans, and the Ad5 receptor (CAR) is known to be down regulated in many aggressive tumors. Species B adenoviruses which use a different receptor than CAR are an alternative which shows great promise. These vectors avoid the immune response against Ad5 and show different cell tropism. Our lab has identified desmoglein2 (DSG2) as the main receptor for a group of species B adenoviruses, including Ad3. DSG2 is an epithelial junction protein and is highly expressed in malignant tumors. During Ad3 infection, binding of Ad3 fiber to DSG2 triggers the transient opening of these intercellular junctions and thus supports the lateral spread of the progeny virus. Our lab has recently developed an Ad3 fiber knob mutant (Y250F) which has an enhanced affinity to DSG2. We have shown that an increased affinity is correlated with a stronger ability to open these junctions. In this study, we developed an Ad3 based oncolytic vector containing fibers with enhanced affinity to DSG2 ($>$ 400x higher than wild type Ad3.) We developed this vector using PCR site-directed mutagenesis and cloning the recombinant gene in *E. coli*. By inserting our gene into a Ad3 plasmid it allowed us to transfect the plasmid into human embryonic kidney cells (HEK 293 cells) and to amplify and recover the mutant oncolytic virus. We are working on in vitro experiments and in vivo tumor xenograft models to test our hypothesis that the oncolytic virus with an enhanced affinity to DSG2 will be translated to a significant improvement to the spreading of progeny virus, and thus a significant improvement to the effectiveness of oncolytic virotherapy.