

Undergraduate Research Symposium May 19, 2017 Mary Gates Hall

Online Proceedings

POSTER SESSION 2

Balcony, Easel 108

1:00 PM to 2:30 PM

AKS Primality Test: A Deterministic Primality Algorithm in Polynomial Time

Daria Micovic, Senior, Mathematics

Blanca Vina Patino, Junior, Pre-Major (Arts & Sciences)

Bryan Tun Pey (Bryan) Quah, Junior, Pre-Major (Arts & Sciences)

Rohan Koosha Hiatt, Junior, Mathematics

Mentor: Amos Turchet, Mathematics

Mentor: Travis Scholl, Mathematics

Prime numbers are fascinating objects in mathematics, fundamental to number theory and cryptography in particular. A primality test takes an integer as input and outputs whether that number is prime or composite. Most practical applications require primality tests to be efficient. Today, the largest known prime number has over twenty million digits. Proving that a number of this size is prime can be computationally expensive. Most tests are probabilistic and do not guarantee that any given prime number is in fact prime. Other tests fail to filter pseudo-primes, like Carmichael Numbers, that are in fact composite integers. In 2002, Agrawal, Kayal, and Saxena published the first deterministic primality test that also runs in polynomial time relative to the binary representation of the input. Although their algorithm represents an important breakthrough in the field of computational number theory, it is seldom used in practice. Our objective was to determine why this idealized algorithm is not practical enough compared to other primality tests. We have re-created the algorithm and optimized our initial naïve implementation by studying each step to achieve optimal complexity using Fermat's Little Theorem, modular arithmetic, and the Fast Fourier Transform for multiplicative efficiency. To confirm that probabilistic methods are still preferred, we compared execution times and accuracy of other tests to our own results.

POSTER SESSION 3

MGH 241, Easel 138

2:30 PM to 4:00 PM

Diversity of Human T Cell Receptors Specific for Mycobacterial Sulfolipid Antigens

Lalita Saraswathi (Lalita) Narayanan, Senior, Biology (Molecular, Cellular & Developmental)

Mentor: Chetan Seshadri, Medicine

Mentor: Krystle Quan

Mycobacterium tuberculosis (M.tb) is the causative agent of tuberculosis, an infectious disease responsible for nearly two million deaths around the world every year. Mycobacteria express a number of cell wall lipids that activate our immune system. For example, T cells are activated by lipids when bound to highly conserved CD1 molecules on the surface of antigen-presenting cells. T cells express a unique T cell receptor (TCR) that is the result of genetic recombination and mutation. Thus, the DNA sequence of a TCR is specific for the antigen recognized by a T cell. Virulent M.tb strains contain sulfolipids (SGLs), but the diversity of SGL-specific T cells has not been determined. This information could be helpful in distinguishing infection from exposure or vaccination. The objective of this work is to determine the diversity of TCRs expressed by SGL-specific T cells. We isolated mRNA from SGL-specific T-cells that were derived from a patient with a latent M.tb infection and used template-switched PCR to amplify the TCR sequences. We will present data summarizing the diversity and conservation of sequence motifs required for recognizing SGLs. The identification of conserved sequences could be utilized to develop novel TCR sequence based diagnostics for M.tb.

POSTER SESSION 3

MGH 241, Easel 137

2:30 PM to 4:00 PM

Optimization of Optogenetic Stimulation Methods for Spinal Cord Injury Rehabilitation

Benjamin David (Benjamin) Pedigo, Senior, Bioengineering

Mary Gates Scholar

Mentor: Chet Moritz, Physiology & Biophysics

Mentor: Sarah Mondello, Rehabilitation Medicine

Recent studies have shown that electrical stimulation of the spinal cord can manipulate spinal cord plasticity and may be effective in recovering motor function after spinal cord injury. The emerging field of optogenetics allows researchers to change a cell's membrane potential using light. Cells are made to express light-dependent ion channels (channel-

rhodopsins) which cause a cell to depolarize or hyperpolarize after being triggered by specific light wavelengths. Our lab has shown that optogenetics can be used to elicit forelimb movements in rats by stimulating the cervical spinal cord. Long-term methods for providing light stimulation *in vivo* are needed to explore the treatment potential of optogenetics. Initial experiments by our group using a LED implant stimulator demonstrated that long-term optogenetic stimulation of the spinal cord results in increased GAP-43 staining. GAP-43 staining highlights areas of new neuronal growth, suggesting an increase in neuronal plasticity in optogenetically stimulated rats. Animals in this study, however, also exhibited unusual tissue morphology around the site of implant. Current research is working to understand the possible sources of this tissue disruption, including heat production from the LED and inflammation induced by LED stimulator implantation near a spinal cord injury. To investigate the possibility of heat production, I developed a new implant prototype that incorporates a thermistor to monitor temperature changes during long-term light stimulation. I tested this implant design in anesthetized and freely-moving rats to investigate how different light stimulation parameters affected implant temperature. I then performed histological tissue analysis at the site of the implants to assess the effects these temperature changes had on tissue condition. The results from this work will inform the next generation of light stimulation implants, and help to improve function following spinal cord injury via optogenetic activation of neural tissue.

ceive an epidural implant for stimulating the spinal cord. All of the rats will perform the former tasks—IBB and LUAT—once a week to track their recovery. Half of the rats will be stimulated via the epidural implant while performing the automatic-pellet-reaching task and Lever task in the hopes of inducing Hebbian plasticity to enhance functional recovery. We are currently testing different epidural implants to identify a method that provides robust, long-term EES. Post-injury performance will be compared to the rat's pre-injury performance. Subsequently, the rats are perfused for tissue analysis. We are optimizing the tissue analysis protocols for identifying GAP-43 immunoreactivity to determine if there was an increase in plasticity and axonal growth. Cresyl violet and myelin staining will also be used to measure the magnitude of the injury by staining the spared neurons and myelin.

POSTER SESSION 3

MGH 241, Easel 136

2:30 PM to 4:00 PM

Epidural Stimulation for Rehabilitation

*Josephine Cordelia (Josephine) D'angelo, Senior,
Neurobiology*

Mary Gates Scholar

Mentor: Chet Moritz, Physiology & Biophysics

Mentor: Sarah Mondello, Rehabilitation Medicine

Spinal cord injury (SCI) disrupts the communication between the body and the brain. As a result, people with SCI typically have significant sensorimotor deficits leading to a lower quality of life. Electrical epidural stimulation (EES)—that is, the stimulation of the spinal cord from the surface of the cord—has shown promise for enhancing recovery of the hindlimbs when applied to the thoracic spinal cord. The goal of this study is to determine whether this same therapy applied to the cervical spinal cord in rats with cervical injuries can improve forelimb function. Prior to injury, the rats perform multiple tasks: Irvine, Beatties and Bresnahan (IBB) task, Limb-use Asymmetry Test (LUAT), Automatic-pellet-reaching task, and a Lever task. Next, rats will receive a unilateral C4 hemicontusion and below the injury the rats will re-