Online Proceedings

Poster Session 1

Commons West, Easel 39

11:00 AM to 12:30 PM

El Motor de Conocimiento: Mercedes Sosa, el Conocimiento Indígena y la Política Involucrada

Gabriela (Gaby) Trejo, Senior, Global Studies (Bothell)
Mary Gates Scholar
Mentor: Jos?? Antonio Lucero, International Studies/CHID
Mentor: Carolyn Pinedo Turnovsky, American Ethnic Studies
Mentor: Simon Trujillo
Mentor: Raj Chetty

Grounded in subaltern studies, a body of work that analyzes the viewpoints of those who are outside of the social, political and geographic power structure, this paper strives to illuminate the Nueva Canción, or "New Song" movement. Emerging in Latin America during the 1960s, Nueva Cancion celebrated the lives of indigenous and laboring class communities, but was largely made up of figures from positions of racial and economic privilege within the larger system. Investigating the seemingly contradictory politics of representation present, this paper discusses how the movement sought to champion disenfranchised figures, while simultaneously casting such figures as incapable of representing themselves; thus rendering them voiceless. At the center of my inquiry is Mercedes Sosa, a prominent Nueva Cancion figure of local and international recognition, who, as a woman of mixed European and indigenous ancestry, attempted to traverse the gap between the movement and its sentimentalized subject matter. By using Sosa as a focal point, this paper aims to illustrate the complex dynamics of the social and political climate in Latin America from 1960-1982, that enabled Sosa to function neither as producer or subject, but as what can be referred to as a "knowledge mover," circulating the dominant ideology at the same time as effectively challenging it.

Session 1E

Sensorimotor Neuroscience

Session Moderator: Eric Chudler, Bioengineering

234 MGH

1:15 PM to 2:45 PM

* Note: Titles in order of presentation.

Background Luminance Alters Tracking Performance of Freely Flying Hawkmoths Revealing Rariable Delays in Optomotor Processing

Robert William Hall, Senior, Biology (General)
Initiative for Maximizing Student Development Scholar, Mary Gates Scholar, Undergraduate Research Conference Travel Awardee
Mentor: Simon Sponberg, Biology
Mentor: Tom Daniel, Biology

Hawkmoths, *Manduca sexta*, feed mainly during early morning and late evening in low light conditions by hovering and tracking moving flowers. The variable lighting conditions in which the hawkmoths feed in nature allow for the perfect setting to examine how visual signal acquisition can affect the performance of motor controlled tasks. By varying the luminance levels, it could result in a change in the amount of time it takes for hawkmoths to react to visual stimuli. In other words, motion-sensing tasks, like tracking a moving flower while feeding, may vary with the background sensory environment. We tested our hypothesis with freely flying moths feeding from a robotically actuated artificial flower at a low luminance level of .3 lux and a high luminance level of 300 lux. Because the flower motion was composed of the superposition of multiple sine waves (0.2-20 Hz), we were able to examine how moths responded at different frequency levels, making it possible to reconstruct a performance pattern. The flower's movement was done in both the vertical and horizontal axes. By calculating the coherence at each frequency, gain, and phase delay, we discovered that moths reliably track at frequencies exceeding 5 Hz. As predicted, we perceived much larger processing delays from the moth's response to the flower's movement at lower luminance levels than higher. This processing delay corresponds to moths being able to perceive and react to visual stimuli 16ms faster at high luminance levels than low luminance levels. At low luminance levels, moths actually overcorrected by overshooting the flower's position at peak tracking frequencies (1-2Hz), possibly due to longer integration delays. Future experiments involve integrating two degrees of freedom by combining multiple axes. Background sensory environment significantly alters the performance of an ecologically-relevant tracking behavior as predicted from sensory neurophysiological mechanisms.

SESSION 1G

ONLINE SPACES: THE EPHEMERA AND RITUAL, THE ARTISTIC AND SUBVERSIVE

Session Moderator: Kristin Gustafson, School of Interdisciplinary Arts and Sciences, UW Bothell

242 MGH

1:15 PM to 2:45 PM

* Note: Titles in order of presentation.

NSFW: The Subversive Cultural Logic of Anonymous Online Culture

Jordan Taylor Augustine, Senior, English
Mary Gates Scholar
Mentor: Jos?? Antonio Lucero, International Studies/CHID
Mentor: Carolyn Pinedo Turnovsky, American Ethnic Studies
Mentor: Raj Chetty
Mentor: Simon Trujillo

NSFW ("Not Safe For Work") is an acronym used to indicate that a website, a picture, or a link contains inappropriate (usually pornographic) content. One could say that all of 4chan.org is NSFW. 4chan is an imageboard where communication takes place in the form of pictures and images posted by anonymous users and deleted minutes after they appear online. This anonymity and ephemerality of communication unleashes levels of racism, sexism, homophobia, and general malevolence unseen in Social Web communities like Facebook. However, 4chan operates with virtually no advertisement (or profit-motive) while Facebook is now a publicly traded corporation, with a mandate to generate revenue for its stockholders by monetizing its assets, which happen to be the words, pictures, jokes, and plans of its more than 1 billion users. Discourses surrounding the Social Web speak in terms of "openness," "connectivity," and "transparency," promising a more civil online community. However, these trends make personal data and communication susceptible to appropriation by capital. How has 4chan remained a largely non-commodified space in the midst of the corporate colonization of the Social Web, and what do we make of its sociopathic ethos? I studied the community and signifying practices of 4chan's random board /b/, finding that although the fundamental anonymity and ephemerality of communication on the board free up participants to be racist, sexist, homophobic, and generally malevolent to degrees unseen in the Social web, these same conditions create a space for truly free speech (in both the monetary and behavioral sense), creating a novel mode of signification and a space for subversive political consciousness in the process.

SESSION 1U

GENOTYPIC AND PHENOTYPIC CONSEQUENCES OF NUTRIENT AVAILABILITY

Session Moderator: Celia Payen, Genome Sciences

175 JHN

1:15 PM to 2:45 PM

* Note: Titles in order of presentation.

Treatment of a Mitochondrial Disease Mouse Model with the Dietary Restriction Mimetic Rapamycin

Maya Sangesland, Senior, Biology (Molecular, Cellular & Developmental)
Amgen Scholar, Mary Gates Scholar
Mentor: Matt Kaeberlein, Pathology
Mentor: Simon Johnson, Neurology

Leigh syndrome is a clinically defined collection of diseases comprised of multiple genetic defects related to mitochondrial function. This syndrome results in early death in children typically due to respiratory failure as a consequence of progressive neuropathy, and currently no viable treatment options exist. Leigh syndrome can result from mutations in several different mitochondrial genes, one of which encodes the NDUFS4 subunit of complex I in the mitochondrial electron transport chain. As the NDUFS4 knockout mice phenocopy human patients with Leigh syndrome remarkably well, this model was utilized for the purpose of this study. Dietary restriction (DR) was identified as a possible treatment for mitochondrial deficiencies through a yeast genetic screen, which found that mitochondrial gene mutants generally received a robust lifespan benefit from DR. Thus, the primary focus of our research is to determine the efficacy of rapamycin (a DR mimetic) as an intervention in the NDUFS4 -/- mitochondrial disease mouse model. Rapamycin functions as a DR mimetic through its inhibition of the mechanistic target of rapamycin (mTOR) signaling pathway, which is responsible for modulating a variety of metabolic and cellular processes. Our model suggests that the increased NADH/NAD ratio resulting from a complex I deficiency in these knockout mice is sensed as energy abundance and results in systemic metabolic derailment. We propose that inhibiting mTOR signaling addresses this defect by altering intracellular energy sensing and modifying metabolic status. In order to test the efficacy of rapamycin treatment, NDUFS4 -/- mice were treated with daily injections of rapamycin or vehicle. Lifespan data for each treatment and additional assays for health related parameters were conducted to determine the response to the treatment regime. Investigating Leigh syndrome in this context will allow us to better understand the underlying mechanisms of pathogenesis, and directly test novel interventions aimed at

attenuating this disease.

SESSION 2B

THE AMERICAS REVISITED: LOCAL VOICES CHALLENGING AND OCCUPYING CONSTRUCTED NATIONAL IDENTITIES

Session Moderator: Julie Villegas, English
171 MGH
3:45 PM to 5:15 PM

* Note: Titles in order of presentation.

Old War, Nuanced Soldiers: 'Generational Borderland' Catalysts of Chilean University Movement
Sara Jane Alstrom, Senior, International Studies
Mary Gates Scholar
Mentor: Jos?? Antonio Lucero, International Studies/CHID
Mentor: Carolyn Pinedo Turnovsky, American Ethnic Studies
Mentor: Raj Chetty
Mentor: Simon Trujillo

The infamous dictator Augusto Pinochet came to power through a bloody coup on September 11, 1973 and drastically changed the face of the Chilean government. His regime instituted strict policies of neoliberalism that led to the privatization of the university system. Forty years after the coup, many of these policies remain in place, untouched and accepted as the reasons behind Chile as the 'economic miracle' of South America. However, tensions under these neoliberal policies, specifically in the university system, fomented a recent eruption of political activism in May of 2011. Why is it that Chile, one of the most economically 'successful' countries in South America and the 'pride' of the IMF and World Bank models, is seeing the rise of a powerful student movement against neoliberal legacies? My research juxtaposes the master narrative of Chile as a 'model country', in terms of hegemonic modernity, against the experiences of the Chilean university students who have fought to challenge it. I argue that the catalyst behind the student movement can be in part explained by the legacies of Pinochet's repression and the political struggles of past generations. This contradictory temporal space forms a 'generational borderland'. These generational ruptures combined with the legacies of repression, have led to the emergence of new forms of innovative and marketable protest, cultivated longevity for the movement through the mistrust of politicians, and inspired a reinvigoration of the Communist Party of Chile.

POSTER SESSION 3

Commons East, Easel 46
2:30 PM to 4:00 PM

Rapamycin as a Novel Intervention in Leigh Syndrome
Jessica May (Jessica) Hui, Senior, Neurobiology
Mentor: Matt Kaeberlein, Pathology
Mentor: Simon Johnson, Neurology

A replicative lifespan (RLS) screen in yeast (*Saccharomyces cerevisiae*) was recently performed in the Kaeberlein lab to determine how different mutants respond to caloric restriction (CR). The results of this screen indicated that many mutants lacking genes encoding proteins related to mitochondrial function were rescued to wild-type lifespan by CR. The beneficial effects of CR in both yeast and mammals are mediated by reduced signaling through the mechanistic target of rapamycin (mTOR) pathway and can be mimicked by the drug rapamycin, a specific inhibitor of mTOR. Based on the findings from the yeast screen, we decided to investigate the effects of rapamycin on mitochondrial disease in a mammal. Leigh syndrome is clinically defined as a collection of genetically heterogeneous human mitochondrial diseases. These diseases result from mutations in genes encoding mitochondrial proteins and are characterized by progressive neurological degeneration resulting in early childhood mortality. To test rapamycin as a therapeutic intervention for Leigh syndrome, we are using a mouse line carrying a heterozygous loss of the electron transport chain complex I subunit Ndufs4, a direct homolog of the human NDUFS4 gene whose mutation causes Leigh syndrome. Mice homozygous for loss of NDUFS4 provide a robust phenocopy of the human disease, including neurological degeneration and early life mortality. Our results so far show that rapamycin dramatically increases lifespan and rescues the Ndufs4-/- mice for several health-related parameters, such as general mobility, visual ability, and grip strength. We are working towards determining the mechanistic basis for these effects, and we hypothesize that they may result from a metabolic shift toward increase fat metabolism in the liver. Understanding how rapamycin rescues Leigh Syndrome mice has clear translational implications, as rapamycin derivatives are FDA approved and patients with Leigh syndrome and other human mitochondrial diseases currently have limited treatment options.