

Pacific Northwest Chapters Meeting of the Society for Neuroscience



Organized by the Pacific Cascades and British Columbia Chapters of SfN

Supported by:

*Society for Neuroscience
UBC Brain Research Center
WWU College of Humanities and Social Sciences
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Schedule of Events

Morning Session

- 8:30 – 9:30** **Registration & Mentor-mentee introductions (AIC West, 2nd floor foyer)**
Poster setup (AIC Skybridge)
- 9:30 – 9:45** **Opening remarks (AIC West, room 204)**
Dr. Janet M. Finlay, Director, WWU Behavioral Neuroscience Program
- 9:45 – 11:00** **Trainee Talks I (AIC West, room 204)**
- 9:45 *Effects of NR1 gene deletion in the mouse prefrontal cortex and hippocampus on working memory and sustained attention*
Gabe S. Puttrese, Janet M. Finlay, Robbie W. Greene
- 10:00 *The ability of estrogens to upregulate cell proliferation in the hippocampus of older females is dependent on reproductive experience*
Cindy K. Barha, Liisa A.M. Galea
- 10:15 *Prenatal exposure to stress and ethanol produce sex-specific changes to long-term potentiation in the dentate gyrus of rat*
Andrea K. Titterness, Brian R. Christie
- 10:30 *Elevated corticosterone levels in stomach milk, serum, and brain of male and female offspring after maternal corticosterone treatment*
Susanne Brummelte, Kim L. Schmidt, Kiran K. Soma, Liisa A.M. Galea
- 10:45 *Investigating the effects of prenatal alcohol exposure and stress on the adult brain in male and female rats: implications for mechanisms underlying depression and addiction*
Kristina Uban, Liisa A.M. Galea, Joanne Weinberg
- 11:00 – 11:30** **Coffee break**
- **Posters (AIC Skybridge)**
 - **Exhibits (AIC West 3rd floor foyer)**
 - **Presentation - *Strategies for Increasing Public Understanding & Support for Biomedical Research* - Susan Adler, Executive Director, NWABR (11:10 – 11:25, AIC West, room 304)**
- 11:30 – 12:30** **Keynote Address I (AIC West, room 204)**

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| <p>Dr. Eberhard Fetz Professor, Physiology & Biophysics at the University of Washington <i>The Adaptive Self and Recurrent Brain-Computer Interfaces</i></p> |
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Afternoon Session

12:30 – 1:30

Lunch (AIC 3rd floor foyer)

- **Panel discussion** - “Life Outside of Academia” Elaine Jones (Allen Institute), Carol Thompson (Allen Institute), Peter Chen (Noldus), Ron Gordon (Elekta), Susan Adler (NWABR) **(12:45-1:15, AIC West, room 304)**
- **Tour of WWU Neuroscience Facilities (12:45-1:15, AIC 5th floor)**

1:30 – 2:45

Trainee Talks II (AIC West, room 204)

- 1:30 *Palmitoylation of the Kv4.2 voltage-gated potassium channel*
Hamed Nazzari, Rujun Kang, Yvonne Cheng, David Fedida, Eric Accili
- 1:45 *Abstinence-dependent transfer of lithium chloride-induced sucrose aversion to a sucrose-paired cue in rats*
John H. Harkness, Sierra Webb, Jeffrey W. Grimm
- 2:00 *Sterol regulatory binding protein-1 (SREBP1) a novel death signal in neuronal excitotoxic diseases: A target for therapeutic intervention*
Changiz Taghibiglou, Henry S. Martin, Ted Weita Lai, Yu Tian Wang, Neil Cashman
- 2:15 *I can play that: Iconic images activate the human MNS*
Lawrence Behmer, Michelle Callero, K.J. Jantzen
- 2:30 *B-catenin stabilization regulates the form and function of hippocampal synapses*
Fergil Mills, Thomas Bartlett, Aiza Waheed, Yu Tian Wang, Shernaz Bamji

2:45 – 3:15

Coffee break (location)

- **Posters** (AIC SkyBridge)
- **Exhibits** (AIC West 3rd floor foyer)
- **Presentation** - *Video Tracking in Action*, Peter Chen, Noldus (AIC East, room 537)

3:15 – 4:15

Keynote Address II (AIC West, room 204)

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| <p>Elaine Jones COO, Allen Institute for Brain Science <i>Changing the World Through Nonprofits</i></p> |
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4:15 – 6:30

Posters and social (AIC SkyBridge)

Awards for best talks and posters

6:30

Dinner

POSTERS
11:00 – 11:30

Board #

- 1 *Impact of social housing and exercise on cellular proliferation in the aged dentate gyrus*
Timal Kannangara, Melanie Lucero, Joana Mohapel-Gil, Rob Drapula, Jessica Simpson, Henrietta van Pragg and Brian R. Christie
- 2 *Chemosensory context conditioning in *Caenorhabditis elegans**
H. Lee Lau and Catharine Rankin
- 3 *Evaluation of neurogenesis in the YAC128 transgenic mouse model of Huntington's Disease*
Jessica Simpson, Joana Gil-Mohapel, Mahmoud Pouladi, Michael Hayden and Brian R. Christie
- 4 *Activity-Induced Palmitoylation of Delta-Catenin enhances clustering with cadherins in hippocampal neurons*
G. Stefano Brigidi and Shernaz X. Bamji
- 5 *Insulin reduces somatodendritic dopamine concentrations*
Dmitry Mebel and Stephanie L. Borgland
- 6 *Characterization of the rat intubation model of Fetal Alcohol Syndrome (presenting w/ Leah Kainer)*
Fanny Boehme, Leah Kainer and Brian R. Christie
- 7 *Characterization of the rodent neurogenesis quiescent zone*
Joanna Gil-Mohapel, Jessica Simpson, Andrea Titterness and Brian R. Christie
- 8 *Insulin-induced LTD of AMPAR-mediated synaptic transmission in dopamine neurons of the VTA*
Labouebe Gwenael and Borgland Stephanie
- 9 *Synaptic vesicles are recruited to nascent synapses by polymerized actin clusters*
Yu Sun and Shernaz X. Bamji
- 10 *Synapse formation is induced through functional interactions between Nlg1 and N-cadherin*
Mytyl Aiga, Joshua Levinson, Eileen Yoshida and Shernaz X. Bamji
- 11 *Automated recognition of brain region mentions in neuroscience literature*
Leon French, Suzanne Lane, Lydia Xu and Paul Pavlidis
- 12 *What can we learn about the plasticity of nervous systems by studying partially overlapping neural circuits?*
Andrew Giles and Catharine Rankin
- 13 *An in vivo study of the role of adenylyl cyclase in dendritogenesis*
Blair Duncan and Kurt Haas
- 14 *Chemical exposure during development results in selective attraction in adult *C. elegans**
Bobby Bragg, Jonathan Adams-Moore and Jackie Rose
- 15 *Effect of ethanol exposure during specific periods of brain development on adult hippocampal neurogenesis*
Anna Patten, Adrian Cox, Leah Kainer, Fanny Boehme, Joana Gil-Mohapel and Brian R. Christie

- 16 *PGRN deficiency linked to FTD decreases neural connectivity but enhances synaptic efficacy*
Lucia Tapia, Aobo Guo, Eileen Yoshida, Fergil Mills, Nan Yang, Cristina Vasuta, Austen Milnerwood, Ian MacKenzie, Lynn Raymond, Max Cynader, William Jia and Shernaz X. Bamji
- 17 *Developmental regulation of synapse elimination by semaphorin 5B*
Lucia Tapia, Katie Cockburn, Wenyan Wang, Erin Currie, Timothy O'Connor and Shernaz X. Bamji
- 18 *Effects of embryonic ethanol exposure in C-elegans*
Conny Lin, Yun Li and Catharine Rankin
- 19 *Disruption of circadian rhythms by constant light does not inhibit cell proliferation in the hippocampus of adult rats*
Anka Mueller, RJ Mear and Ralph E. Mistlberger
- 20 *Increased excitation in mice over-expressing Neuroligin-1 associated with impaired long-term potentiating and learning*
Brennan Eadie, Timal Kannangara, Regina Dalhaus, Rochelle Hines, Yu-Tian Wang, Alaa El-Husseini and Brian R. Christie
- 21 *D1 receptors are responsible for modulating synaptic transmission in distinct regions of the orbitofrontal cortex*
Jennifer Thompson and Stephanie L. Borgland
- 22 *An RNAi screen to identify components of the sydn-1 / pfs-2 synapse and axon development*
Julia Cochran, Ben Rollins and Heather Van Epps
- 23 *Characterization of an endogenous hemi-gap-junctional current in Xenopus laevis oocytes*
Maggie Fuqua, Bonnie McKinney, Thomas Jordan, Chris Scodeller, Hamid Samie and Jose Serano-Moreno
- 24 *Abstinence and accumbal subregion-dependent attenuation of sucrose cue reactivity by SCH23390*
John Harkness, Cristine Ratliff, Addison Tice, Kindsey North and Jeff Grimm
- 25 *In vivo microdialysis of catecholamines in the mouse prefrontal cortex*
Jonathan Bale, Mitchell Wold and Janet Finlay
- 26 *B-catenin stabilization regulates development results in selective attraction in adult C. elegans*
Fergil Mills, Thomas Bartlett, Aiza Waheed, Yu Tian Wang and Shernaz Bamji
- 27 *Chronic fluoxetine exposure does not alter responsiveness to acute exposure following washout*
Rusty Mann, Chris Buchanan and Kara Gabriel
- 28 *Response properties of V1 neurons at chromatic detection threshold*
Charlie Hass and Greg Horowitz

TRAINEE TALKS I 9:45 – 11:00 am**9:45 – 10:00**

Effects of NR1 gene deletion in the mouse prefrontal cortex and hippocampus on working memory and sustained attention

Gabe S. Puttrese, Janet M. Finlay, and Robbert W. Greene

Dysfunction of glutamate N-methyl-D-aspartate (NMDA) receptors may contribute to cognitive deficits in schizophrenia. We examined whether a localized deletion of a functionally requisite exon for the NMDA receptor NR1 subunit in the prefrontal cortex (PFC) and/or CA3 subregion of the hippocampus disrupts working memory and sustained attention (n=9-13/group). Localized gene deletions were induced using an AAV-Cre vector together with floxed NR1 transgenic mice (a gift from Tonegawa lab). Working memory was assessed using a T-maze delayed-response task. Mice reached a criterion performance level of >80% correct responses under conditions of no delay and were subsequently tested under 10, 20, and 30 s delay conditions. Relative to controls, mice previously sustaining CA3 and PFC + CA3 gene deletions exhibited a reduction in percent correct responses under conditions of 10-s delays (82±2, 65±5, and 60±3, respectively) and 20-s delays (80±3, 62±3, and 63±4, respectively). In mice previously sustaining gene deletions of the PFC, percent correct responses under 10- and 20-s delays (71±4 and 70±3, respectively) tended to be lower than control values, however this effect was not statistically significant. Sustained attention was assessed using a visual 5-choice serial reaction time task. Mice were trained to achieve a criterion level of performance of <20% omissions when tested using a stimulus duration (SD) of 5.0 seconds. Probe trials using SDs of 2.5, 1.75, and 1.25 were then introduced. Mice with prior gene deletions of the PFC and PFC + CA3 exhibited significantly higher percent omissions under probe trial conditions than controls or those previously sustaining gene deletions of CA3 alone (89±13, 85±14, 60±8, and 60±7, respectively). Deficits in attention were present in the absence of any change in accuracy. Localized deletion of the NR1 gene was confirmed by riboprobe in situ hybridization. The present findings suggest that NMDA receptor dysfunction in the PFC and CA3 differentially impairs sustained attention.

10:00 – 10:15

The ability of estrogens to upregulate cell proliferation in the hippocampus of older females is dependent on reproductive experience

Cindy K. Barha and Liisa A.M. Galea

Estrogen has been implicated as a possible therapeutic agent for improving cognition in postmenopausal women and has been linked to neurodegenerative disorders such as Alzheimer's disease. Despite the many different forms of estrogen most research has focused on estradiol. Estradiol is the more common estrogen in young women, while estrone is more common in older women. The most commonly prescribed hormone replacement therapy (HRT) consists primarily of estrone, and does not have as many cognitive-enhancing benefits as HRTs that consist primarily of estradiol. Furthermore, there are two naturally occurring isomers of estradiol, 17β-estradiol and 17α-estradiol. Although 17α-estradiol is a putative ligand for the estrogen membrane receptor, most research conducted with estradiol has used 17β-estradiol. We have previously shown that all of these estrogens promote hippocampal neurogenesis in a dose-dependent manner in young, virgin ovariectomized female rats. The aim of the present study was to determine the acute effects of 17β-estradiol, 17α-estradiol, and estrone on hippocampal neurogenesis in aged ovariectomized female rats and to determine whether effects are dependent on previous reproductive experience. Middle-aged 13 to 14 month old retired breeder female rats and aged-matched virgin female rats were injected subcutaneously with vehicle (sesame oil), or 10μg dose of 17β-estradiol, 17α-estradiol, or estrone. Rats were then injected with bromodeoxyuridine, a marker of DNA synthesis, and perfused 24 hours later. We have found that all estrogens increase cell proliferation in retired breeder female rats but not in virgin female rats. Therefore, previous reproductive experience may make the older brain more responsive to estrogens later in life. Findings from this study will advance our understanding of how different forms of estrogen mediate hippocampal neurogenesis in aged rats, which may ultimately lead to the development of new therapeutic advances in the treatment of symptoms associated with menopause.

10:15 – 10:30

Prenatal exposure to stress and ethanol produce sex-specific changes to long-term potentiation in the dentate gyrus of rats

Andrea K. Titterness and Brian R. Christie

Animal models of fetal alcohol syndrome (FAS) have shown that prenatal ethanol exposure (PNEE) can impair long-term potentiation (LTP), a putative mechanism behind learning and memory, in the hippocampus of adult males. Interestingly, prenatal stress, like PNEE, can also reduce the capacity for LTP in male offspring. Surprisingly, it is unknown how either PNEE or prenatal stress affect LTP in the hippocampus of females. The goals of the current study were to 1) investigate how PNEE affects LTP in female offspring and 2) determine if prenatal stress compounds the effects of PNEE on long-term potentiation in male and female offspring. **METHODS:** Pregnant Sprague-Dawley rats were assigned to one of three feeding conditions on gestation day 1 (GD1): 1) ethanol (E): ad libitum access to a liquid diet containing 35.5% ethanol derived calories; 2) pairfed (PF): liquid diet similar to E dams containing maltose-dextrin instead of ethanol; dams received the same amount of food in g/kg/day as matched E dam; 3) control (C): ad libitum access to standard rat chow. E, PF and C dams were further separated into a prenatal stress treatment (3, 45-min restraint sessions/day during GD12-21) or non-stress treatment (remained undisturbed in home cage). LTP was assessed in the dentate gyrus of male and female offspring between postnatal days 30-35. **RESULTS:** PNEE reduced LTP in male offspring compared to C males but enhanced LTP in female offspring compared to C females. Surprisingly, prenatal stress did not reduce LTP in male offspring, regardless of prenatal diet. In contrast, prenatal stress did reduce LTP only in females following PNEE. **CONCLUSIONS:** These results indicate that hippocampal function in males and females is differentially affected by PNEE. Furthermore, females, but not males, are sensitive to the deleterious effects of prenatal stress on LTP.

10:30 – 10:45

Elevated corticosterone levels in stomach milk, serum, and brain of male and female offspring after maternal corticosterone treatment

Susanne Brummelte, Kim L. Schmidt, Kiran K. Soma and Liisa A.M. Galea

Early influences such as maternal stress affect the developmental outcome of the offspring. We created an animal model of postpartum depression based on giving high levels of corticosterone to the dam during pregnancy or the postpartum, which resulted in behavioral and neural changes in the offspring. The current study investigated whether elevated levels of corticosterone during pregnancy or the postpartum are transferred from the mother to her offspring by examining corticosterone levels in the stomach milk, serum and the brain of offspring using solid phase extraction and radioimmunoassay. Dams received daily injections of corticosterone (40mg/kg) or oil (control) either during pregnancy (gestational day 10-20) or during the postpartum (day 2-18). Results revealed that pups exposed to high gestational maternal corticosterone had higher serum levels of corticosterone on postnatal day (PND) 1, but there were no significant differences in corticosterone concentrations in the stomach milk or brain. However, if pups were exposed to elevated maternal corticosterone postpartum, they showed significantly higher corticosterone levels in their stomach milk and brain on PND7, but not in serum. On PND18, serum corticosterone levels were significantly higher in the postnatally corticosterone-exposed pups compared to controls. However, 24 hours after weaning this effect was gone. Thus corticosterone given to the dam postpartum is transferred to the offspring via breast milk which results in elevated corticosterone in the brain and serum of these offspring. Developmental exposure to high corticosterone could reprogram the HPA axis and contribute to the observed behavioural and neural changes seen in adult offspring.

10:45 – 11:00

Investigating the effects of prenatal alcohol exposure and stress on the adult brain in male and female rats: implications for mechanisms underlying depression and addiction

Kristina Uban, Liisa A.M. Galea and Joanne Weinberg

Children exposed prenatally to alcohol may present with a broad range of problems, referred to as fetal alcohol spectrum disorder (FASD). Neurodevelopmental deficits in these children include several problems in a number of life domains, including cognition, communication, learning, memory and behavior, and an increased prevalence of mental health issues such as depression, anxiety and addiction. One of the most consistently described biological abnormalities in depression and addiction is an alteration in hypothalamic-pituitary-adrenal (HPA) activity.

Importantly, data indicate that alcohol exposure in utero reprograms the developing fetal HPA axis leading to alterations which persist into adulthood, and these alterations may contribute to the increased prevalence of mental health problems later in life. Using our model of prenatal alcohol exposure (PAE), key brain regions implicated in depression and addiction were investigated in males and females following exposure to chronic mild stress (CMS) in adulthood. Male and female offspring were either left undisturbed or were subjected to a 10 day CMS regimen. All animals were sacrificed on day 11 under basal conditions, and brains were analyzed for mRNA levels of corticotropin-releasing hormone (CRH). Preliminary evidence suggests that CRH mRNA levels in the paraventricular nucleus of the hypothalamus did not differ among groups. However, CRH mRNA levels were decreased in the amygdala (CeA) of prenatal alcohol exposed females compared to control females, while no significant differences were seen in males. Interestingly, different alterations in glucocorticoid receptors (MR and GR) mRNA were observed in males and females, with a trend for decreased MR in non-stressed E females. In summary, region- and sex-specific alterations in CRH mRNA following CMS were observed, with differential effects among prenatal treatment groups. These findings will add to our understanding of brain damage specific to prenatal alcohol exposure and stress that may play an important role for mechanisms underlying depression and addiction.

TRAINEE TALKS II 1:30 – 2:45 pm

1:30 – 1:45

Palmitoylation of the Kv4.2 voltage-gated potassium channel

Hamed Nazzari, Rujun Kang, Yvonne Cheng, David Fedida and Eric Accili

Potassium channels play a critical role in regulating excitability and synaptic plasticity in neurons. The Kv4.2 voltage-gated ion channel subunit underlies a transient A-type potassium current that modulates dendritic function and action potential firing in neurons. The proper functioning of these channels is dependent on their ability to traffic to the cell surface and interact with the lipid bilayer. To date, little is known about the molecular mechanisms that contribute to this dynamic regulation. Palmitoylation refers to the reversible addition of the fatty acid palmitate to cysteine residues located in cytoplasmically accessible regions of the protein. Here, we demonstrate the Kv4.2 is palmitoylated in adult rat cortical neurons as well as when overexpressed in COS-7 cells, the first time such a modification has been shown in this channel isoform and the first demonstrating palmitoylation of a voltage-gated potassium channel in vivo. By serine mutagenesis of cysteine residues and biochemical assay, we have localized the site of palmitoylation to the intracellular C-terminus. Examination of Kv4.2-expressing COS-7 cells by patch clamp electrophysiology revealed no change in the current density or opening and closing properties of serine-substituted mutant channels that lack palmitoylation, suggesting that the abolition of palmitate attachment does not occur secondarily to alterations in folding, assembly or trafficking of the channel to the cell surface. Given the presence of Kv4.2 in dendrites, it is tempting to speculate that the addition of palmitate regulates its localization to synapses in the central nervous system.

1:45 – 2:00

Abstinence-dependent transfer of lithium chloride-induced sucrose aversion to a sucrose-paired cue in rats

John H. Harkness, Sierra Webb and Jeffrey W. Grimm

Rationale: Responding for drug- and sucrose-paired cues increases over forced abstinence (incubation of craving). If the incentive value of the cue is tied to the primary reward, it should be possible to reduce cue reactivity by devaluing the primary reward. **Objectives:** We investigated whether conditioned taste aversion (CTA) to sucrose would transfer to a sucrose-paired cue after 1 or 30 days of forced abstinence and whether CTA after 1 d of forced abstinence would affect incubation of craving. **Materials and methods:** Rats self-administered 10% sucrose paired with a tone + light cue for 10 days. After 1 (Exp.1) or 30 (Exp.2) days of forced abstinence, rats received two home-cage pairings of sucrose with either LiCl (65 mg/kg, IP) to produce CTA, or saline as a control. Two days later, rats responded for the cue alone. The following day, sucrose consumption was assessed in the same operant conditioning chamber. Exp.1 rats were tested again one month later to determine if CTA would affect incubation of craving. **Results:** Exp.1: CTA after 1 d of forced abstinence did not attenuate cue reactivity when tested immediately after CTA nor did the

treatment affect incubation of craving or incubation of sucrose consumption. Exp.2: CTA after 1 month of forced abstinence resulted in a significant reduction in cue reactivity. Conclusion: The incentive value of sucrose and the conditioned representation of sucrose become greater over an extended period of forced abstinence. This incubation enhances the transfer of an aversion to the primary reward to the conditioned cue.

2:00 – 2:15

Sterol regulatory binding protein-1 (SREBP1) a novel death signal in neuronal excitotoxic diseases: A target for therapeutic intervention

Changiz Taghibiglou, Henry S. Martin, Ted Weita Lai, Yu Tian Wang and Neil Cashman

Neuronal excitotoxicity or overactivity of glutamate receptors is believed to be a common feature of most neuropathological conditions causing neuronal injury such as stroke, brain trauma, amyotrophic lateral sclerosis (ALS), Alzheimer's, Huntington and Parkinson diseases. Excitotoxicity caused by overactivation of the N-methyl-D-aspartate subtype of glutamate receptors (NMDARs) is thought to be one of the primary contributors in neuronal injury. However, several large scale clinical trials have failed to find the expected efficacy of NMDAR antagonists in reducing the brain injuries of patients. Thus, novel NMDAR-based therapies for neuronal excitotoxicity are urgently needed. Here, we demonstrate that in primary cortical neuronal cultures, and in the rat transient middle cerebral artery occlusion (MCAo) model of focal ischemic stroke, excitotoxic neuronal death is associated with a NMDAR- and time-dependent activation of SREBP-1, a transcription factor that regulates the expression of genes involved in maintaining lipid homeostasis. Degradation of Insig-1, an ER membrane resident protein, is a prerequisite for SREBP-1 activation. We have established a direct linear correlation between excitotoxicity-induced neuronal cell death and SREBP-1 activation. We demonstrate that Indip, a TAT fused peptide which blocks SREBP1 activation via stabilizing Insig-1, efficiently protects neurons against excitotoxicity-induced death. We also investigated SREBP1 activation in ALS and found that in spinal cords of the murine transgenic G93A model of ALS and post-mortem specimens of human ALS (FALS and SALS) Insig-1 was drastically degraded leading to significant SREBP-1 activation. Again, blocking SREBP1 activation by Indip was significantly neuroprotective against glutamate induced excitotoxicity in both wild type mouse and G93A cultured spinal cord neurons. Our study identifies activation of SREBP1 as a novel step in signaling cascades leading to excitotoxic neuronal death.

2:15 – 2:30

I can play that: Iconic images activate the human MNS

Lawrence Behmer, Michelle Callero and K.J. Jantzen

The human sensorimotor cortex generates EEG waves in the alpha and beta spectrum, referred to as the "mu-band," and is suppressed when the motor cortex is engaged during execution of a movement or when an individual simply observes a movement. It has been posited that this desynchronization in the mu-band represents activation of the mirror neuron system (MNS). The MNS, first found in the macaque monkey and later demonstrated to have a human homologue, has been shown to facilitate an implicit understanding by the observer of the meaningful actions of others. This same system has also been shown to be active when presented with abstract stimuli such as sounds, action language, and communicative hand gestures. The ability to "map" observed actions or meaningful stimuli onto one's own MNS requires the observer to have an existing motor repertoire of what is being observed, and is a key requirement that allows for the action to be understood or imitated. Diverse audio visual stimuli and meaningful biological actions have been shown to activate the MNS in monkeys and humans, but no study has examined whether or not abstract, iconic images might activate the MNS of individuals who can understand them. We measured mu desynchronization in the rolandic region of trained musicians and non-musicians using EEG, while presenting them with novel music stimuli. Mu ERD was significantly greater in the alpha and beta bands when musicians observed sheet music and musical performances. We believe this rolandic desynchronization is indicative of downstream IFG activity which is consistent with MNS activation measured by EEG and MEG in previous studies. This study shows that iconic images that are capable of being translated into meaningful motor activity activate the MNS in individuals who can understand them. This research has implications for sensorimotor learning, language acquisition, and artificial intelligence.

2:30 – 2:45

B-catenin stabilization regulates the form and function of hippocampal synapse

Fergil Mills, Thomas Bartlett, Aiza Waheed, Yu Tian Wang and Shernaz Bamji

Recent evidence suggests that disruption of synapse formation and function is the major correlate of AD dementia in Alzheimer's Disease (AD), and temporally precedes out-and-out neuronal loss. It is therefore crucial to understand the basic biology of synapse sprouting, maintenance and elimination and how these processes are affected in AD patients. β -catenin, the intracellular partner of the cadherin family of cell adhesion molecules, may play a key role in the synaptic pathology of AD. The cadherin/ β -catenin complex is essential to the formation and remodelling of synapses, and both molecules associate with presenilin-1 (PS1), a protein which targets β -catenin for degradation. Mutations in the PS1 gene account for close to 70% of early-onset familial Alzheimer's disease (FAD) cases, and many of these key PS1 mutations disrupt β -catenin degradation, resulting in an intracellular accumulation of β -catenin. We hypothesize that increases in β -catenin levels contribute significantly to the synaptic pathology observed in AD cases caused by mutations in the PS1 gene, and that stabilization of β -catenin levels will perturb synapse efficacy and plasticity by the aberrant maintenance of strong cadherin-based adhesion. To test this hypothesis, we have generated a transgenic mouse line expressing a conditionally-stabilized form of β -catenin in the hippocampus. We are currently characterizing the synaptic, physiological and behavioural consequences of this stabilization and have found significant changes in the morphology and plasticity of hippocampal synapses in these animals, including increased numbers of synaptic vesicles per synapse, decreased paired pulse facilitation (PPF), and impaired responses to 100 Hz high-frequency stimulation (HFS) and 14 Hz prolonged repetitive stimulation (PRS). These results demonstrate that chronic stabilization of β -catenin may have effects in the adult CNS which are different from its previously characterized roles regulating development and dendritic morphology.

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PNW Chapters Meeting of SfN: Directory of Attendees

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