Sixth Annual Neuropsychology Research Day
September 19, 2008

9:00-9:10 AM	Welcome Joshua Brumberg, Ph.D. Founder and co-organizer Neuropsychology Research Day
Session I: Moderator:	Janine Flory, Ph.D. Director of Clinical Training Neuropsychology Department of Psychology, Queens College, CUNY
9:10-9:25	**Feeding elicited by GABA-B Receptor agonists in the nucleus accumbens and ventral tegmental area: intersite inhibitory interactions by GABA-A and GABA-B antagonists** Pat Miner (Bodnar Lab, Queens College)
9:25-9:40	**The Impact of Antidepressant Treatment on Cognitive Functioning of Depressed Older Adults** Michelle Culang (Sneed Lab, Queens College)
9:40-9:55	**NMDA Antagonism in the VTA impairs Reward-Related Learning** Karen Kest (Ranaldi Lab, Queens College)
9:55-10:10	**Reaction Time Differences in Preschool Children with ADHD: The influence of cuing and feedback** Agnieszka Mlodnicka (Halperin Lab, Queens College)
10:10-10:25	**Tobacco Use in College Students: Associations with Trait Measures of Impulsiveness** Samantha Weltz and Naftali Wein (Flory Lab, Queens College)
10:25-10:45	Questions
10:45-11:00	Coffee Break

Session II:

Moderator: Joshua C. Brumberg, Ph.D.
Program Head, Neuropsychology Ph.D. Subprogram
Department of Psychology, Queens College, CUNY

11:05-11:20 **Sensitivity of Tests of Balance and Weight Transfer to MCI and Early Alzheimer's Disease**
Richard Mariani (Kluger Lab, Lehman College)

11:20-11:35 **Behavioral modification of adult neurogenesis**
Carole Parent (Pytte Lab, Queens College)

11:35-11:50 **Morphine-6-glucuronide rapidly increases pain sensitivity independently of opioid receptor activity in mice and humans**
Amanda Waxman (Queens College, Kest Lab, College of Staten Island)

11:50-12:00 PM Questions

12:00-1:00 PM Lunch

Session III: Keynote Speaker

1:00-2:30 Words of Welcome:
Richard Bodnar, Ph.D.
Acting Dean of Research and Graduate Studies
Queens College, CUNY

Introduction of Keynote Speaker:
Joshua Brumberg, Ph.D.
Program Head, Neuropsychology Ph.D. Subprogram
Department of Psychology, Queens College, CUNY

Keynote Address: **Dynamic auditory processing, musical experience and language/reading development**
Nadine Gaab, PhD.
Assistant Professor of Pediatrics
Harvard Medical School
Member of the Faculty of Education
Harvard Graduate School of Education

Children's Hospital Boston
Department of Medicine
Division of Developmental Medicine
Laboratories of Cognitive Neuroscience

2:30-2:35 Break

Session IV:

Moderator:	Claudia Brumbaugh, Ph.D. Department of Psychology, Queens College, CUNY
2:35-2:50	**Morphological Heterogeneity of Layer VI Neurons in Mouse Barrel Cortex** Chia-Chien Chen (Brumberg Lab, Queens College)
2:50-3:05	**Feedback from securely and insecurely attached potential partners: Will flattery get you everywhere?** Peryl Grossman (Brumbaugh Lab, Queens College)
3:05-3:20	**Verbal Fluency Performance in Amnestic MCI and Older Adults with Cognitive Complaints** Kate Nutter-Upham (Rabin Lab, Brooklyn College)
3:20-3:35	**Interchannel Associations Among Emotion Perception Tasks Increase in Older Age** Katie Finley (Borod Lab, Queens College)
3:35-3:50	**Three-dimensional shape from second-order orientation flows** Carole Filangieri (Li Lab, Queens College)
3:50-4:05	**Signaling pathways of VEGF-mediated neuroprotection following status epilepticus** Elisa Salerni (Croll Lab, Queens College)
4:05-4:20	Questions

Abstracts

Session I

Feeding elicited by GABA-B Receptor agonists in the nucleus accumbens and ventral tegmental area: intersite inhibitory interactions by GABA-A and GABA-B antagonists. Pat Miner (Bodnar Lab, Queens College)

Understanding the neurochemistry of food intake is a significant step in understanding obesity and its treatment. The ventral tegmental area (VTA) and nucleus accumbens (NAC) are critical structures in this feeding circuit, and elicit feeding following GABA-A (muscimol) and GABA-B (baclofen) administration. Pretreatment with GABA-A (bicuculline), but not GABA-B (saclofen) antagonists in the same site blocks muscimol-induced feeding, indicating within-site receptor selectivity. Correspondingly, saclofen, but not bicuculline in the same site blocks baclofen-induced feeding. Regional and reciprocal feeding interactions between the VTA and NAC have been observed for opioid-induced feeding in which general and selective antagonists administered in one site blocks opioid-induced feeding in the second site. The present preliminary study examined whether GABA-B agonist (baclofen)-induced feeding elicited from the NAC was blocked by VTA pretreatment with either GABA-A (bicuculline) or GABA-B (saclofen) antagonists, and whether baclofen-induced feeding elicited from the VTA was blocked by NAC saclofen. Baclofen (200 ng) elicited potent (3-4 fold) increases in feeding following NAC and VTA treatment. NAC baclofen-induced feeding was dose-dependently reduced by VTA pretreatment with either saclofen (0.5-5 ug) or bicuculline (7.5-150 ng). Correspondingly, VTA baclofen-induced feeding was dose-dependently reduced by NAC pretreatment with saclofen. These data indicate a reciprocal regional interaction between the VTA and NAC in which both GABA-A and GABA-B receptors in the second site participate in mediating feeding responses elicited by GABA-B agonists elicited from the first site. These preliminary data are being replicated, and we are extending this analysis to the mediation of feeding elicited by GABA-A agonists.

The Impact of Antidepressant Treatment on Cognitive Functioning of Depressed Older Adults. Michelle Culang (Sneed Lab, Queens College)

Cognitive impairment is common in late-life depression and can be extremely debilitating for elderly patients. The literature regarding the effect of antidepressants on cognition is inconsistent and there have been few placebo-controlled trials investigating this issue in depressed older adults. In this talk, I present some work aimed at examining the impact of placebo versus medication treatment on change in cognitive functioning with the use of neuropsychological data collected as part of a large (n=174), randomized, double-blind, placebo-controlled trial of citalopram in depressed older adults. To our knowledge, this is the first attempt to approach these issues using a placebo-controlled trial of an SSRI in an old-old (>75 years old) depressed population.

NMDA Antagonism in the VTA impairs Reward-Related Learning. Karen Kest, (Ranaldi Lab, Queens College)

Recent studies in our lab have shown that NMDA receptors in the ventral tegmental area (VTA) are necessary for the acquisition, but not expression, of instrumental learning. Whether

the same neural mechanisms underlie Pavlovian learning remains to be elucidated. In the present study, we hypothesized that NMDA receptor stimulation in the VTA is necessary for the acquisition, but not expression, of conditioned approach behavior. Male rats were prepared with indwelling cannulae positioned to allow bilateral microinjections of AP-5 (an NMDA receptor antagonist; 0.0, 0.25 or 0.5 μg/0.5 μl). Animals were exposed to either an acquisition (5 sessions) or expression (9 sessions) paradigm in which random presentations of a discrete light stimulus were paired with food pellet deliveries. In the acquisition study, animals received intra-VTA microinjections of AP-5 before sessions 1-3 and intra-VTA microinjections of saline before session 5. In the expression study, animals received intra-VTA microinjections of AP-5 before session 9. A drug related effect was observed in the acquisition study. The high dose of AP-5 (0.5 μg/0.5 μl) significantly impaired the acquisition of conditioned approach whereas the low dose of AP-5 (0.25 μg/0.5 μl) did not and appeared similar to vehicle (0.0 μg/0.5 μl). In the expression study, intra –VTA microinjections of AP-5 did not impair the expression of conditioned approach after it was already learned. These finding support our hypothesis that NMDA transmission in the VTA is necessary for the acquisition, but not expression, of Pavlovian learning.

Reaction Time Differences in Preschool Children with ADHD: The influence of cuing and feedback. Agnieszka Mlodnicka (Halperin Lab, Queens College)

Background: Increased intra-subject variability on reaction time (RT) tasks is emerging as one of the most consistent findings in the literature on cognition in ADHD and has led some investigators to posit that attentional fluctuations represent a core deficit in this disorder. Two approaches to "improving" attention have been through the use of warning signals/cues and/or rewarding feedback. Objectives: This study examined the differential impact of cues and feedback on RT in typically-developing (TD) preschool children and a well-matched group with ADHD to determine their differential impact during the early manifestations of the disorder. Method: Child-appropriate versions of computerized simple RT (SRT), cued RT (CRT), and feedback (FB) tasks were administered to a sample of 74 preschool children (36 TD, 38 ADHD) between three and five years of age. Children were digitally visually recorded during the administration of the task. These recordings were later coded for off-task behaviors likely to interfere with valid responding during the tasks; all invalid trials were excluded from the analyses. The effects of CRT and FB, relative to SRT were quantified by calculating difference scores from SRT. Two-way (Group x Task) analyses of variance were conducted to examine differences in performance with mean RT and intra-subject RT variability (RTSD) serving as the dependent measures. Results: For mean RT, a significant interaction emerged such that cuing improved performance of children in the TD group, but not in those with ADHD; both groups had a robust positive response to FB. With regard to RTSD, similar, although not identical results emerged. Cuing substantially reduced RTSD in the TD group, but had no impact on those with ADHD. In contrast, RTSD was greatly reduced in those with ADHD following FB. Conclusion: These data suggest that preschool children with ADHD are quite sensitive to and have a robust response to rewarding feedback, but unlike their TD counterparts, the implementation of cues or warnings prior to stimulus onset has little impact on their behavior. Implications for treatment as well as the possible underlying causes of ADHD will be discussed.

Tobacco Use in College Students: Associations with Trait Measures of Impulsiveness. Samantha Weltz and Naftali Wein (Flory Lab, Queens College)

Varying aspects of impulsive personality have been associated with tobacco use in cross-sectional and prospective studies, including reward seeking and disinhibition. Neurobiological theories of addiction have posited that while initial drug use is associated with reward seeking and increased dopaminergic activity in the mesolimbic reward system, repeated or chronic use results in a loss of inhibitory control (i.e., compulsive drug taking) that is due, in part, to neuroplastic changes to the frontostriatal system. An alternative view is that the inability to inhibit drug use and drug seeking behavior represents individual differences in trait measures of disinhibition and frontal cortex functioning that may predate the initiation of drug use. This view, that disinhibition is "trait" rather than "state" related, may help to explain why some people who initiate tobacco use do not go on to become addicted. The prevalence of tobacco use in students enrolled in Queens College introductory psychology courses (most of whom are non-smokers or "triers"), will be presented, followed by data showing associations between tobacco use and trait measures of reward seeking and disinhibition. In addition to self-reported measures of impulsivity, researchers often measure impulsiveness using standardized behavioral tasks, but the retest reliability of these measures has not been established. We will describe a planned investigation to evaluate retest reliability of commonly used self-report and behavioral measures of impulsiveness. As the above mentioned alternative theories of addiction indicate, it is important to establish the stability of behavioral measures of impulsiveness in order to use them in prospective investigations of the pathway from initiation to addiction.

Session II

Sensitivity of Tests of Balance and Weight Transfer to MCI and Early Alzheimer's Disease.
Richard Mariani (Kluger Lab, Lehman College)

Studies identifying behavioral measures that are sensitive to the earliest manifestations of dementia such as mild cognitive impairment (MCI) and mild Alzheimer's disease (AD) are of critical importance for the early diagnosis and prognosis of these conditions. This study examined three groups of elderly: cognitively normal individuals (NL; N = 44) and cases with either MCI (N = 28) or mild AD (N = 26). MCI is a designation for elderly in between normal aging and early AD. MCI cases are at heightened risk for future decline to AD, compared to NL elderly. The 98 individuals in this study ranged in age from 54 to 86 years (mean = 72 years) and were evaluated at the Silberstein Aging & Dementia Research Center at the NYU School of Medicine. All subjects were assessed on computerized tests of balance and weight transfer along with standard tests of verbal recall of paragraphs and a measure of global cognitive and functional ability, the Global Deterioration Scale (GDS). The balance and weight transfer tests were performed both with and without the presence of concurrent visual feedback. The three elderly groups had similar ages and gender distributions but differed modestly in levels of education. Analyses of covariance (adjusting for the possible effects of education) indicated the MCI (GDS 3) and AD (GDS 4) groups performed more poorly on tests of balance and weight transfer than the NL (GDS 1 - 2) group. These findings indicate that tests of balance and weight transfer may be useful in the early detection of dementia. Recent evidence suggests that tests of motor function show lower associations with level of education than more traditionally used tests of verbal recall. Thus motor tests of balance and weight transfer may prove to be more accurate than verbal tests in detecting the early signs of dementia among elderly with low levels of education.

Behavioral modification of adult neurogenesis. Carole Parent (Pytte Lab, Queens College)

Neuron replacement occurs in regions of the adult zebra finch telencephalon which are used in song learning and song production. Regeneration of these new neurons may allow fine-tuning of the song motor pattern, and newborn neuron survival could be a function of the accuracy of song production. To test this idea, we altered sensory feedback during singing and assessed new neuron retention in the neural substrates underlying song behavior. In the first treatment, we injected the vocal muscles with botulinum toxin (botox) which resulted in a functional muscle paralysis and altered auditory and sensorimotor feedback during song production. In a second treatment, we deafened birds, thereby removing auditory feedback while leaving sensorimotor feedback intact. New neuron quantification was done using fluorescent antibody labeling to the mitotic marker bromodeoxyuridine (BrdU) as well as the neuron specific protein Hu. We report that deafening leads to a decrease in new neuron retention in NCM, the avian analog of the auditory cortex, without any effect on song production regions. Botox treatment results in a *decrease* in new neuron retention in HVC, a premotor nucleus which also contains neurons tuned to the bird's own song. In the same birds, botox treatment resulted in an *increase* in early neuron retention in a nucleus of avian basal ganglia (Area X) which functions in adult song maintenance and fine-tuning of the motor pattern. These results suggest that the regulation of new neuron survival and replacement may be influenced by the sensory information to which those neurons are tuned.

Morphine-6-glucuronide rapidly increases pain sensitivity independently of opioid receptor activity in mice and humans. Amanda Waxman (Kest Lab, College of Staten Island)

Acute doses of morphine-6-glucuronide (M6G), a morphine metabolite with analgesic properties, have been associated with a paradoxical increase in pain sensitivity in mice and humans. Here, we tested whether this M6G hyperalgesia in mice and humans requires opioid receptor activity and a contribution from NMDA receptors. Using the tail withdrawal test in mice, an acute injection of M6G (10 mg/kg) produced hyperalgesia in CD-1 mice implanted with pellets containing the general opioid antagonist naltrexone. Continuous M6G infusion (1.6 mg/kg/24 h) also produced hyperalgesia within 24 h that lasted for a minimum of 6 days, irrespective of the presence or absence of concurrent opioid receptor blockade. The NMDA receptor antagonist MK-801 (0.05 mg/kg) blocked and reversed hyperalgesia after the acute injection and continuous infusion of M6G, respectively. In healthy volunteers, M6G (0.2 mg/kg) injection increased pain sensitivity for at least 6 hours. This M6G hyperalgesia persisted despite the simultaneous continuous infusion of a high dose of naloxone (3 mg loading dose followed by 3 mg/h). In contrast to M6G, a morphine (0.15 mg/kg) injection caused analgesia only. Collectively, these data indicate that acute and chronic M6G exposure causes hyperalgesia independent of prior or concurrent opioid receptor activity or analgesia. In mice, a causal role for the NMDA receptor is also indicated. Since M6G concentrations rise during chronic morphine treatment, the present data suggest that M6G may play an appreciable role in morphine-induced hyperalgesia. Furthermore, the use of M6G as an option in opioid-based pain management may not preclude development of hyperalgesia.

Session III

Keynote Address. Nadine Gaab, Ph.D., Harvard University.

Session IV

Morphological Heterogeneity of Layer VI Neurons in Mouse Barrel Cortex. Chia-Chien Chen (Brumberg Lab, Queens College)

Understanding the basic neuronal building blocks of the neocortex is a necessary first step towards comprehending the composition of cortical circuits. Neocortical layer VI is the most morphologically diverse layer, and plays a pivotal role in gating information to the cortex via its feedback connection to the thalamus and other ipsilateral and callosal cortico-cortical connections. The heterogeneity of function within this layer is presumably linked to its varied morphological composition. However, so far very few studies have attempted to define cell classes in this layer using unbiased quantitative methodologies. Utilizing the Golgi staining technique, we reconstructed over N=220 cortical neurons from layer VI of mouse barrel cortex. Morphological analyses were performed by quantifying somatic and dendritic parameters, and using principal component and cluster analyses, we quantitatively categorized neurons into six distinct morphological groups. Systematic replication on a separate population of neurons yielded similar results, demonstrating the consistency and reliability of our categorization method. Subsequent post-hoc analyses of dendritic parameters further supported our neuronal classification scheme. Characterizing neuronal elements using unbiased quantitative techniques provides a framework for better understanding structure-function relationships within neocortical circuits in general.

Feedback from securely and insecurely attached potential partners: Will flattery get you everywhere? Peryl Grossman (Brumbaugh Lab, Queens College)

When asked whether secure or insecure partner prototypes are more attractive, research has shown that people tend to select secure individuals as their first choice. Despite this pattern, in reality, not everyone selects secure partners. The objective of this study was to examine whether flattery from potential partners plays a role in insecure mate choice. Participants were exposed to insecure targets who gave positive feedback, and to secure targets who gave neutral feedback. We found that flattery substantially increased insecure targets' attractiveness. These results suggest that people may compromise their relationship standards for the satisfaction of feeling good about themselves, regardless of the partners' negative qualities and the possible repercussions of those qualities.

Verbal Fluency Performance in Amnestic MCI and Older Adults with Cognitive Complaints. Katherine E. Nutter-Upham, Laura A. Rabin, Andrew J. Saykin, & Robert M. Roth; Brooklyn College and The Graduate Center of CUNY, Brain Imaging Laboratory, Dartmouth Medical School

Verbal fluency tests are employed regularly during neuropsychological assessments of older adults, and deficits are a common finding in patients with Alzheimer's disease (AD). Little extant research, however, has investigated verbal fluency ability and subtypes in preclinical stages of neurodegenerative disease. We examined verbal fluency performance in 107 older adults with amnestic mild cognitive impairment (MCI, n = 37), cognitive complaints (CC, n = 37) despite intact neuropsychological functioning, and demographically-matched healthy controls (HC, n = 33). Participants completed fluency tasks with letter, semantic category, and semantic switching constraints. Both phonemic and semantic fluency were statistically (but not clinically) reduced in amnestic MCI relative to cognitively intact older adults, indicating subtle

changes in both the quality of the semantic store and retrieval slowing. Investigation of the underlying constructs of verbal fluency yielded two factors: Switching (including switching and shifting tasks) and Production (including letter, category, and action naming tasks), and both factors discriminated MCI from HC albeit to different degrees. Correlational findings further suggested that all fluency tasks involved executive control to some degree, while those with an added executive component (i.e., switching and shifting) were less dependent on semantic knowledge. Overall, our findings highlight the importance of including multiple verbal fluency tests in assessment batteries targeting preclinical dementia populations and suggest that individual fluency tasks may tap specific cognitive processes.

Interchannel Associations Among Emotion Perception Tasks Increase in Older Age. Katie Finley [1], Joan C. Borod[1], Adam M. Brickman[2], J. Michael Schmidt[2], Stephanie Assuras[1], Susan J. Hall[3], Lawrence H. Pick[3], & Martin Sliwinski[4]; [1]City University of New York -- The Graduate Center & Queens College; [2]Columbia University College of Physicians and Surgeons; [3] Independent Practice, New York, NY; [4]Syracuse University

Previous research on emotional communication has revealed a general decline in the processing of emotional stimuli with age, but very few studies have examined multiple channels in the same subjects. Research on the effects of aging on *cognitive* processes has revealed that correlations among different cognitive measures increase with age (Baltes & Lindenberger, 1997). In the current project, we investigated the effects of aging on the perception of emotional stimuli in three channels (i.e., facial, prosodic, and lexical) across the adult life span. A unique feature of this study was an examination of interchannel associations across the decades. Our sample included 103 healthy adults divided into three age groups (Young: 20-39; Middle-Aged: 40-59; Older: 60-81). Participants were screened for cognitive and basic perceptual functioning, and matched across groups for gender, education, occupational level, ethnicity, handedness, and general intellectual functioning. Experimental procedures included discrimination and identification tasks from the New York Emotion Battery (Borod, Welkowitz, & Obler, 1992), and control procedures included perceptual measures using nonemotional stimuli. Correlations among channels were analyzed to explore whether older individuals demonstrate less specialization for emotional perceptual abilities than healthy younger cohorts. As hypothesized, the correlations among channels for emotional tasks increased steadily as a function of age, with the Older group median correlation significantly higher than the Young group median correlation for identification tasks. This effect is similar to age-related increases in correlations reported among cognitive domains. Implications of these findings are discussed within the context of aging hypotheses, including the hemispheric asymmetry reduction (HAROLD) model (Cabeza, 2002).

Three-dimensional shape from second-order orientation flows. Carole Filangieri (Li Lab, Queens College)

Input into the visual system is 2-dimensional, and yet we convert this 2-dimensional input effortlessly into percepts of the 3-dimensional world around us. How does the visual system accomplish this, and where might it happen along the visual pathway? We have used the psychophysical technique of selective adaptation using images of textured 3-D surfaces to help localize the neural processing of 3-D shape information from 2-D retinal images. This talk will summarize findings in our lab that point to the existence of neurons located beyond striate cortex (V1) that are selective for particular 3-D shapes. Shape percepts and negative shape aftereffects elicited by luminance-modulated (LM) orientation flows can be explained by responses of

neurons in V1 that largely only respond to LM contours. We show that orientation flows formed by contrast-modulated contours (CM) and illusory contours (IC), that have been shown to activate neurons in V2 and beyond, convey 3-D shape and elicit negative shape aftereffects. In addition, adaptation to LM orientation flows induce robust 3-D shape aftereffects on CM and IC test stimuli. These results cannot be easily explained by responses of V1 neurons alone. Rather, our results can be explained by the adaptation of 3-D shape-selective neurons beyond V1 that respond to LM, CM, and IC orientation flows. The transfer of aftereffects across these pattern types strengthens the current evidence for pattern-invariant, 3-D shape-selective mechanisms located in extra-striate regions.

Signaling pathways of VEGF-mediated neuroprotection following status epilepticus. Elisa Salerni (Croll Lab, Queens College)

Past research in our lab has demonstrated that intrahippocampal administration of Vascular Endothelial Growth Factor (VEGF) rescues neurons from death during pilocarpine-induced status epilepticus. Because VEGF is a large protein, it does not cross the blood-brain barrier, which limits its therapeutic utility in human epilepsy. If the receptor mediating VEGF's neuroprotective effect could be identified, it would be possible to develop small molecule reagents specific to the target receptor to serve as neuroprotective agents. VEGF's neuroprotective effects in vitro have previously been shown to be mediated via the signaling activity of one of its receptors, VEGFR2 (Flk-1 or KDR), either through the Akt and/or Erk1/2 protein kinase signaling pathway(s). To establish whether in vivo VEGF-induced neuroprotection is mediated by VEGFR2 receptor following status epilepticus, rats received SU1498, (a VEGFR2 blocker) during VEGF treatment in pilocarpine-induced status epilepticus. Hippocampal cell loss 24-hours after status epilepticus was determined using a subjective rating scale of Nissl stained brain tissue. Unexpectedly, animals treated with the SU1498 VEGFR2 blocker demonstrated increased protection following status epilepticus than control animals. Such results may indicate that the neuroprotective effects of VEGF do not function via VEGFR2, as is conventionally observed in cell culture. Instead, VEGF-mediated protection may function via VEGFR1 or via alternative compartmentalization of VEGFR2. That is, blockade of VEGFR2 may result in changes in receptor profiling, perhaps augmenting VEGF's effects via VEGFR1 located on glia or via neuropilin-1, which is heavily expressed on hippocampal pyramidal neurons.

Neuropsychology Ph.D. Subprogram Colloquia Fall 2008

Monday	August 25	**Welcome New Neuropsychology Graduate Students**
Friday	September 19	6th **Annual Neuropsychology Research Day** (Rosenthal Library 230, 9:00 AM – 5:30 PM) **Nadine Gaab, Ph.D.** Harvard University *Dynamic auditory processing, musical experience and language/ reading development*
Wednesday	September 24	**6th Annual Alumni Lecture** **Linda Friedman, Ph.D.** New York College of Osteopathic Medicine *Long term consequences of early-life seizures on neurotoxicity and neurogenesis "role of glutamate receptors"*
Monday	October 6	**Marla Hamburger, Ph.D.** Columbia University *Cortical representation of naming in epilepsy*
Wednesday	October 15	**Jeffrey Goodman, Ph.D.** SUNY Downstate Medical Center *Developmental Hypothyroidism Induces a Cellular Malformation in the Corpus Callosum of the rat*
Wednesday	October 29	**Marom Bikson, Ph.D.** City College of New York, CUNY *New technology for non-invasive electrical treatment of brain disorders: High-Density transcranial Direct-Current Stimulation*
Monday	November 3	To Be Determined
Monday	December 8	**Eric A. Fertuck, Ph.D.** Columbia University *Social Neuroscience of Borderline Personality Disorder*

Seminars are open to the public and held at 12:15PM in the Razran Room (SB E-308).
For further information contact Joshua C. Brumberg, Ph.D., joshua.brumberg@qc.cuny.edu.