Brain Mapping Center

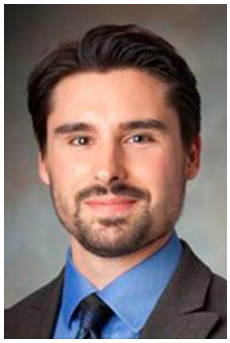
SEMINAR SERIES

Sponsored by the UCLA Brain Mapping Center Faculty

The focus of these talks is on advancing the use of brain mapping methods in neuroscience with an emphasis on contemporary issues of neuroplasticity, neurodevelopment, and biomarker development in neuropsychiatric disease.

Hosted By: Marco Iacoboni, MD, PhD, Psychiatry and Biobehavioral Sciences, UCLA

Towards Computational Neuropsychiatry by Combining Neuroimaging, Pharmacology & Computation



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Neuropsychiatric disorders such as schizophrenia profoundly alter the structure and function of distributed neural networks and present a massive health and economic burden. Non-invasive functional neuroimaging tools have evolved sufficiently to allow reliable characterization of large-scale distributed neural systems in humans. Such approaches have been applied to better understand large-scale neural network disturbances in neuropsychiatric disease, particularly using task-based and resting-state techniques. However, human neuroimaging does not yet allow the evaluation of individual neurons within local circuits, where pharmacological treatments ultimately exert their effects. This limitation constitutes an important obstacle to the effort to translate findings from animal research to humans and from healthy humans to patient populations. Integrating new neuroscientific tools may help to bridge these gaps. Two complementary approaches are discussed in the context of understanding cognitive deficits in schizophrenia - namely computational modeling and pharmacological neuroimaging. First, we discuss behavioral and neuroimaging studies that combine causal pharmacological manipulations in healthy volunteers and neurobiologically grounded computational models. Specific focus is placed on neuroimaging studies using the NMDA receptor antagonist, ketamine, to probe glutamate synaptic dysfunction associated with schizophrenia. Second, we discuss the extension of the models to help understand pharmacological task-based findings in the context of working memory. Finally, clinical resting-state neuroimaging findings across schizophrenia illness stages are discussed in relation to pharmacological effects and computational models extended to the level of large-scale networks. In summary, the argument is presented that linking experimental neuroimaging studies in humans with causal pharmacological manipulations that can be incorporated into computational models, may advance the effort to bridge levels of analysis in clinical neuroscience.

October 1, 2015 11:00 am - 12:00 pm

Neuroscience Research Building (NRB 132)
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