10th Annual Conference
October 17-19
Baltimore, MD
More details at:
nndc.org/annual-conference
Poster Submission Guidelines

The 10th Annual NNDC Conference will be held October 18-19 at the Baltimore Marriott Waterfront in Baltimore, Maryland. The Conference Program Committee welcomes all conference attendees to submit a poster related to depressions and bipolar illnesses, focusing on our theme of "Clinical Innovation, Collaboration, and Computational Psychiatry: Leveraging Networks" in the categories of Basic Science, Clinical Programs, and Task Group Work.

Posters will apply to one of the following categories:

Basic Science
All areas of research are encouraged, but posters dealing with approaches (both biological and psychosocial) to understanding mood disorders, new or repurposed treatments and technologies, or monitoring strategies with the potential to improve patient outcomes are particularly welcome.

Clinical Programs
Unified efforts are the best way to expedite technological innovation, dissemination of new information, and translation of research into clinical practice. Posters might showcase multidisciplinary programs or demonstrate the efficacy of collaborations beyond academic boundaries.

Task Group Work
Reports on study progress or programs developed by NNDC Task groups, or as a result of previous work of NNDC Task Groups, are encouraged.

A $500 prize will be awarded to the best poster in each category.

Frequently Asked Questions

How do I submit a poster?
Please submit a half-page abstract for the poster you would like to present at the Annual Conference at surveymonkey.com/r/NNDC2018Posters. Abstracts must be submitted by 5:00 pm ET on Friday, August 17, 2018 to be considered.

What are the physical requirements for posters?
Posters must be no larger than 36 inches high by 48 inches wide. Pinboards and pushpins will be available for you to hang your poster.

When is the poster session?
The poster session is scheduled for Thursday, October 18 at 1:00 pm ET, during a dessert reception following lunch. You may hang your poster at any time before that - pinboards will be available first thing Thursday morning, so it is recommended that you hang your poster during breakfast (7:30-8:30 am). Awards for each category will be announced at dinner on Thursday, October 18. You may leave your poster up for the remainder of the conference or take it down Thursday night. Please note that any posters left up after the conference adjourns (Friday, October 19 at 1:00 pm ET) will be discarded.
Example 1: Identification of a genome-wide significant association with depression under stress – A GWAS analysis from Intern Health Study
Yu Fang MSE1, Laura Scott PhD2, Margit Burmeister PhD1, Peter Song2 & Srijan Sen MD PhD1
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**Background:** Major depressive disorder (MDD) is one of the most prevalent mental illness and world's second leading cause of disability. Unfortunately, genome-wide association studies (GWAS) have been unsuccessful in identifying genetic variants associated with MDD unless recruiting massively increased size of samples [1,2] or rigorously assessed samples with homogeneous phenotypes [3].

**Methods:** The Intern Health Study (IHS) is a longitudinal cohort study that assesses stress and mood in medical interns around the country. The 9-item Patient Health Questionnaire depressive symptom score (PHQ-9) prior to internship, and quarterly during internship year, along with DNA samples, were collected from the first-year residents. The longitudinal design and the focusing on current depression, which can be assessed much more accurately than lifetime history of depression, help to improve phenotypic accuracy. Additionally, IHS focuses only on depression that develops in response to internship stress, reducing the heterogeneity in pathways to depression present in general population samples. Taking these advantages, a GWAS of PHQ-9 change under internship stress was conducted, aiming to target variants associated with depression under stress.

**Results:** After quality control, population stratification and imputation, 3825 individuals with European ancestry and 12 million SNPs were included in the GWAS. One loci at chromosome 11 (ACTN3 gene, top SNP=rs2229456, MAF=0.18, p=1.8e-8, minor allele beta= -0.17, meaning carriers of minor allele have more resilience under stress) exceeded genome-wide significance in association with depression under stress. 555 individuals with East Asian ancestry and 436 individuals with South Asian ancestry were included in the trans-ethnic meta-analysis together with the European ancestry subjects. The same loci reached genome-wide significance after the meta-analysis (log10BF=6.8). The total heritability explained by all common SNPs is 17% (SE=0.09, P<0.05).

**Conclusions:** Our findings may provide a new angle to investigate the genetic basis of depression and its development. The result indicates the involvement of ACTN3 gene in depression under stress. ACTN3 gene is known to encode alpha-actinin-3 protein in skeletal muscle and associate with athletic performance [4]. The mechanism underlying its association with depression is yet to be studied. It is also expected that as the Intern Health Study going on, larger sample size will help to identify more loci associated with depression under stress.


Example 2: Single dosage of ketamine as a response predictor to a 21 days oral ketamine treatment. Two-phases control trial.
Yoav Domany MD, Haggai Sharon, MD, Ricardo Tarassch PhD, Roi Meidan MD, Shaul Schreiber MD, Miki Bloch MD, Talma Hendler MD PhD and Maya Bleich-Cohen PhD

**Background:** Ketamine is a rapid acting antidepressant. Treatment response, however, might be limited. We hypothesis that using the ketamine's rapid therapeutic response on the first administration, may predict treatment remission to a 21 days of oral ketamine trial.

**Methods:** In a two-phase trial; phase 1- a double-blind, placebo-controlled phase, 40 depressed subjects, were randomized to oral ketamine treatment or placebo. In phase 2, the placebo-receiving subjects
Example 3: Do mixed symptoms carry an increased risk for suicidal ideation and behavior beyond that attributable to depressive components?

Jess G. Fiedorowicz, M.D., Ph.D., Jane E. Persons, Ph.D., Michael J. Ostacher, M.D., M.P.H., Peter Zandi, M.H.S., M.P.H., Ph.D., Po W. Wang, M.D., Caleb Adler, M.D., Shervin Assari, M.D., M.P.H., Wendy Marsh, M.D., M.S., Michael E. Thase, M.D., Mark A. Frye, M.D., William Coryell, M.D.

Objectives: We sought to assess whether the risk of suicidal ideation or behavior during mixed states exceeds that attributable to the depressive components alone.

Methods: We utilized real-world clinical data collected on 290 patients with bipolar disorders (bipolar I, bipolar II, and bipolar not otherwise specified (NOS)) from the National Network of Depression Centers (NNDC) Clinical Care Registry (CCR) followed for a mean of 27.5 weeks. Depressive symptoms were measured with the Patient Health Questionnaire-9 (PHQ-9), manic symptoms with the Altman Self-Rating Mania (ASRM), and suicidal ideation and behavior with the Columbia-Suicide Severity Rating Scale (C-SSRS), obtained as part of the routine, measurement-based care provided across the NNDC. The relations between depressive symptoms, manic symptoms, and the interaction thereof (mixed symptoms) on suicidal ideation and behavior were modeled in generalized linear mixed models.

Results: Depressive symptoms, as measured by the PHQ-9, were strongly associated with suicidal ideation and behavior (p<0.0001), while there was no significant association with manic symptoms as measured by the ASRM or the interaction between depressive and manic symptoms. Similar results were observed when the outcome was restricted to suicidal behavior and when mood was modeled categorically. There was no evidence of moderation by gender or bipolar subtype.

Limitations: Diagnoses were based on clinician assessment and not structured interview. Suicidal ideation was more frequently observed than suicidal behavior (23/272 visits where outcome positive).

Conclusions: Depression represents the primary mood state accounting for suicide risk in bipolar disorder. Co-occurring symptoms of mania (mixed symptoms) do not appear to convey an elevated risk for suicidal ideation or behavior beyond that explained by the depressive symptoms alone.