NATIONAL NETWORK *of* DEPRESSION CENTERS

NNDC Annual Conference
September 24-25, 2019
Ann Arbor, MI

MOOD DISORDERS IN TRANSFORMATION:
NOVEL APPROACHES TO BIOLOGY, PSYCHOLOGY, AND ACCESS TO CARE

Poster and Travel Award Application Guidelines

Poster Submission Guidelines

The 11th Annual NNDC Conference will be held September 24-25 at the Sheraton Ann Arbor in Ann Arbor, Michigan. The Conference Program Committee welcomes all conference attendees to submit a poster related to depressions and bipolar illnesses, focusing on our theme of "Mood Disorders in Transformation: Novel Approaches to Biology, Psychology, and Access to Care" 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 **https://form.jotform.com/91125761755157**. Abstracts must be formatted appropriately (see last page of this document for examples) and be submitted by 5**:00 pm ET on Friday, August 16, 2019** 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 Tuesday, September 24 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 Tuesday 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 Tuesday, September 24. You may leave your poster up for the remainder of the conference or take it down Tuesday night. Please note that any posters left up after the conference adjourns (Wednesday, September 25 at 12:30 pm ET) will be discarded.

Travel Award Eligibility

The Conference Program Committee is pleased to offer up to 10 Emerging Scholar Travel Awards of $1,000 each to eligible conference attendees who present a poster at the 11th Annual NNDC Conference. This award can be used to request reimbursement for travel costs assocated with attending the conference, including airfare, hotel, ground transportation, parking, and mileage, as appropriate.

Who is eligible to apply for a Travel Award?

To be eligible to apply for a Travel Award, you must satisfy the following criteria:

1. Be the first or last author on the poster that you are presenting at the Annual Conference
2. Be a graduate student, fellow, post-doctoral fellow, resident, or junior faculty within 5 years of fellowship or post-graduate appointment at one of the 25 NNDC Member Centers

Applicants from under-represented minority groups are particularly encouraged to apply for a Travel Award.

How do I apply for a Travel Award?

To apply for a Travel Award, you must submit a letter of recommendation from your program director, faculty advisor, or NNDC Board Member representing your site in addition to your half-page abstract. Please submit all required information at **https://form.jotform.com/91125761755157**.

When are applications due?

Complete applications (including poster abstract) must be submitted for consideration by **5:00 pm ET on Friday, August 16, 2019**.

How do I claim my reimbursement?

Submit receipts no later than 5:00 pm ET on Friday, October 25, 2019 to Rachel Skylis (rskylis@nndc.org) or mail them to:

NNDC
2350 Green Road Suite 191
Ann Arbor, MI 48105

A reimbursement request form will be provided to you prior to the conference.

Other questions?

Please contact Rachel Skylis (rskylis@nndc.org).

Abstract Formatting Examples

Example 1: Identification of a genome-wide significant association with depression under stress – A GWAS analysis from Intern Health Study

Yu Fang MSE[1*],Laura Scott PhD[2], Margit Burmeister PhD[1], Peter Song[2] & Srijan Sen MD PhD[1]
1. Molecular and Behavioral Neuroscience Institute, University of Michigan, Ann Arbor, MI 48109
2. Department of Biostatistics, School of Public Health, University of Michigan, Ann Arbor, MI 48109
* presenting author, contact: yfang@umich.edu

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.

[1] Major Depressive Disorder Working Group of the PGC, et al. "Genome-wide association analyses identify 44 risk variants and refine the genetic architecture of major depression." (2017): preprint
[2] Hyde, Craig L., et al. "Identification of 15 Genetic Loci Associated with Risk of Major Depression in Individuals of European Descent." Nature Genetics 48.9 (2016): 1031-036
[3] CONVERGE consortium. "Sparse Whole-genome Sequencing Identifies Two Loci for Major Depressive Disorder." Nature 523.7562 (2015): 588-91
[4] Yang, Nan, et al. "ACTN3 Genotype Is Associated with Human Elite Athletic Performance." The American Journal of Human Genetics 73.3 (2003): 627-31

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 Tarrasch 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

received open label ketamine. Subject were evaluated pre-trial, after 4 hours and on days 3, 7, 14, and 21 as well as safety assessment on day 28. In order to predict remission at day 21 discriminant analyses were performed using MADRS at time 4 hours as a predictor. **Result:** In phase 1, 22 subjects were randomized to the ketamine group, and 18 to the placebo group. In phase 2, 11 subjects received open label ketamine. For phase 1: the reduction in MADRS at day 21 was 12.09 points in the ketamine group versus 1.50 in the placebo group (p=0.05). In phase 2, the reduction was 14.33. No serious side effects were documented. Based on the response to the first dosage of ketamine (phase 1&2), we correctly categorized 90.9% of all patients (100% out of 26 non-remising, and 57.1% out of 7 remising) (Wilks' Lambda= 0.72, chi(1)=10.03, p=.002). **Conclusion:** Repeated administration of oral ketamine produced rapid and sustained amelioration of depressive symptoms, and was well tolerated. Based on the response to the first dose, we manage to predict who will achieve remission in 90.8%. Notably, all the patients (100%), who did not response to the first dosage, did not eventually achieve remission. Our result suggests that once a patient did not respond to the first ketamine administration, further administrations might be futile.

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.[1,3,4,*], Jane E. Persons, Ph.D.[2,*], Michael J. Ostacher, M.D., M.P.H.[4,5], Peter Zandi, M.H.S., M.P.H., Ph.D.[6], Po W. Wang, M.D.[5], Caleb Adler, M.D.[7], Shervin Assari, M.D., M.P.H.[8], Wendy Marsh, M.D., M.S.[9], Michael E. Thase, M.D.[10], Mark A. Frye, M.D.[11], William Coryell, M.D.[1]

of the National Network of Depression Centers Bipolar Disorders Interest Group

[1]Departments of Psychiatry and Internal Medicine; [2]Roy J. and Lucille A. Carver College of Medicine; [3]Department of Epidemiology, College of Public Health; [4]François M. Abboud Cardiovascular Research Center, Iowa Neuroscience Institute, Obesity Research and Education Initiative, The University of Iowa, Iowa City, IA

[4]Bipolar Disorder & Depression Research Program, VA Palo Alto Health System, Palo Alto, CA

[5]Department of Psychiatry and Behavioral Sciences, Stanford University School of Medicine, Stanford University, Palo Alto, CA

[6]Department of Mental Health and Epidemiology, Johns Hopkins Bloomberg School of Public Health, Department of Psychiatry, Johns Hopkins Medicine, Baltimore, MD

[7]Department of Psychiatry and Behavioral Neurosciences, University of Cincinnati, Cincinnati, OH

[8]Department of Psychiatry, University of Michigan, Ann Arbor, MI

[9]Department of Psychiatry, University of Massachusetts Medical School, Worcester, MA

[10]Department of Psychiatry, Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA

[11]Department of Psychiatry & Psychology,Mayo Clinic, Rochester, MN

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