

BRAIN & BEHAVIOR RESEARCH FOUNDATION

2023 ANNUAL REPORT

Awarding research grants to develop improved treatments, cures, and methods of prevention for mental illness.

MISSION

THE BRAIN & BEHAVIOR RESEARCH FOUNDATION IS COMMITTED TO ALLEVIATING THE SUFFERING CAUSED BY MENTAL ILLNESS BY AWARDING GRANTS THAT WILL LEAD TO ADVANCES AND BREAKTHROUGHS IN SCIENTIFIC RESEARCH.

IN 2023 BBRF FUNDED MORE THAN \$10.3 MILLION IN NEW RESEARCH GRANTS ACROSS A BROAD SPECTRUM OF BRAIN ILLNESSES WHICH INCLUDE: ADDICTION, ADHD, ANXIETY, AUTISM, BIPOLAR DISORDER, BORDERLINE PERSONALITY DISORDER, DEPRESSION, EATING DISORDERS, OCD, PSYCHOSIS, PTSD, SCHIZOPHRENIA, AS WELL AS SUICIDE PREVENTION.

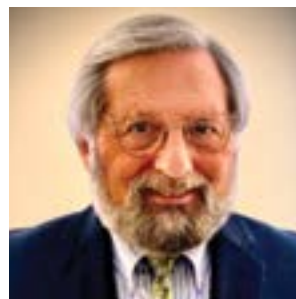
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Jeffrey Borenstein, M.D.
President & CEO



Herbert Pardes, M.D.
President, Scientific Council



Geoffrey Simon
Chair, Board of Directors

The Brain and Behavior Research Foundation (BBRF) is the world's largest private funder of mental health research grants. Over the past 37 years, BBRF has awarded more than \$450 million in research grants to more than 5,400 scientists around the world who are working to find better treatments, cures, and methods of preventing mental illness.

In 2023 BBRF funded \$10.3 million in Young Investigator Research Grants across a broad spectrum of brain illnesses. Support for BBRF is robust and growing. Thanks to the extraordinary financial support of the WoodNext Foundation, BBRF will award ten Distinguished Investigator Grants to senior-level scientists who are conducting innovative projects in neurobiological and behavioral research. The application and selection process began in 2023 and the grants are being given in 2024.

BBRF's 2023 \$1 Million Challenge Match was a tremendous success. We set a high bar to raise \$1 million and we exceeded our goal! At the beginning of the match, we announced that thanks to two very generous family foundations, donations would be matched from new donors, former donors who had lapsed, and current donors who increased their 2023 contribution (the increased amount was matched). In June we achieved and exceeded the \$1 million match. This then prompted the two family foundations that are so passionate about BBRF's vital mission to increase their match by an additional \$1 million. We are delighted to report that at the end of 2023, we again achieved the match goal.

Many significant Foundation-funded research breakthroughs were published in major psychiatric and medical journals during 2023. This annual report features our *Leading Research Achievements by BBRF Grantees, Prizewinners & Scientific Council Members* which illustrate some of the remarkable progress being made by grant-winning investigators. These important advancements, as well as other scientific discoveries, underscores the vital role BBRF plays in helping people who live with mental illness.

In July, we hosted our *Scientific Council Dinner* where we awarded the highly esteemed Klerman & Freedman Prizes. In October, at BBRF's *International Mental Health Research Symposium*, five outstanding achievement award-winning scientists presented their research on schizophrenia, bipolar disorder, pediatric mood and anxiety disorders, and cognitive neuroscience. A presentation was also given by the winner of the Pardes Humanitarian Prize in Mental Health. All presentations from the 2023 Symposium are available to watch online at: www.bbrfoundation.org/event/international-mental-healthresearch-symposium. At our *Annual Awards Dinner* we honored and presented the BBRF Outstanding Achievement Prizes as well as the Pardes Humanitarian Prize in Mental Health.

BBRF's print publication, *Brain & Behavior Magazine*, continues to highlight some of the most important advancements achieved by our grantees. This is supplemented by our weekly email newsletter "eNews," that's available free-of-charge to donors and the public. It features research breakthroughs of BBRF grantees, award-winners, and Scientific Council members.

BBRF also produces the award-winning television series, *Healthy Minds*, which is broadcast on public television stations throughout the U.S. and on pbs.org. The series provides useful information to the public about psychiatric conditions and treatments as well as cutting-edge research advancements. Season 9 is available to watch here: <https://bbrfoundation.org/healthy-minds-tv>.

**While continuous advancements in brain research are being made, more still needs to be done.
As always 100% of your contribution for research directly supports research, as our operating expenses are covered by separate foundation grants.**

Together, we will continue to fund innovative and impactful research that will drive the field forward. Our shared goal of a world free from debilitating mental illnesses relies first and foremost upon you, our donors—in partnership with the scientists selected by the BBRF Scientific Council—who are working to transform your donations into improved treatments, cures, and methods of prevention for our loved ones.

We are inspired by the magnitude and scope of the discoveries that are being made by the scientists we fund together and appreciate your ongoing generous support.

Sincerely,



Jeffrey Borenstein, M.D.
President & CEO



Herbert Pardes, M.D.
President, Scientific Council



Geoffrey Simon
Chair, Board of Directors

2023 **LEADING RESEARCH
ACHIEVEMENTS** BY
FOUNDATION GRANTEES,
PRIZE WINNERS,
& SCIENTIFIC COUNCIL
MEMBERS

Talk Therapy + Brain Stimulation Reduced Suicidal Ideation in Patients with Borderline Personality Disorder and Treatment-Resistant Depression

Next-Generation Therapies: **Borderline Personality Disorder, Suicidality**

Story Highlights:

Individuals with borderline personality disorder (BPD) and co-occurring major depression and suicidal ideation when treated with dialectical behavior therapy (DBT) and concurrent magnetic seizure therapy (MST) experienced a rapid, clinically meaningful reduction in suicidal ideation at 5 weeks that was sustained at 4-month follow-up.

Journal: *Nature Mental Health*
January 19, 2023

About 1.4% of U.S. adults (over 3 million people) experience Borderline Personality Disorder (BPD) in a typical year. But BPD is difficult to treat. Evidence for the effectiveness of medications alone to treat BPD symptoms is limited. A form of talk therapy called dialectical behavior therapy (DBT) originally designed to reduce suicidal behaviors has often been effective in treating people with BPD.

But BPD often co-occurs with other psychiatric illnesses including major depression, and it is associated with an exceptionally high risk of suicidality, especially among patients with comorbid depression. DBT can help many people to reduce their self-harming behaviors, but additional treatment approaches are urgently sought to address the risk of suicidal behavior associated with BPD. It has been estimated that as many as 10% of BPD patients end their lives in suicide, and that over 80% make at least one suicide attempt during their life.

Now, a team of researchers led by 2014 BBRF Young Investigator **Anthony C. Ruocco, Ph.D.**, of the University of Toronto and Centre for Addiction and Mental Health, has reported encouraging results of a small feasibility trial of a new treatment combination for severely suicidal patients with BPD and co-occurring treatment-resistant depression. The team treated 9 such

patients with DBT and “conjoint” magnetic seizure therapy (MST) for 5 weeks, and compared impacts on their symptoms—especially suicidal ideation and depression, as well as cognitive performance—with 10 similar patients who received only DBT for 5 weeks.

Conjoint therapies are those administered in concert. In this case, participants in both groups received 1 hour weekly of individual DBT and 1 hour of weekly DBT skills training focused on distress tolerance. Participants in the “conjoint therapy” group received these DBT treatments plus up to 15 MST treatments (up to 3 per week) over the 5 weeks of the trial. The participants were volunteers who were currently experiencing moderate-to-severe suicidal symptoms, and each was allowed to choose whether to be in the DBT-only or the DBT plus MST group.

MST is a form of non-invasive brain stimulation that has been associated in some trials with significant reduction of suicidal ideation in patients with treatment-resistant depression. It had not been tested previously in BPD. MST uses magnetic pulses to induce a brief seizure in the brain that is intended to have therapeutic effects. In its object, it is similar to electroconvulsive therapy (ECT), but it is designed to act more focally, i.e., in a smaller, targeted area or areas in the brain. ECT is associated with short-term memory



Anthony C. Ruocco, Ph.D., C.Psych.

University of Toronto
2014 BBRF Young Investigator



Jenna M. Traynor, Ph.D.

McLean Hospital / Harvard University
2022 BBRF Young Investigator

loss in some patients, while MST in testing to date has been associated with fewer such cognitive side effects.

BPD's symptoms vary from patient to patient, and are diverse. A diagnosis requires that the patient meet general diagnostic criteria for a personality disorder, as defined in the DSM-5 manual. This means having a disturbance in at least two of four domains: identity, interpersonal functioning, impulse control, and emotion regulation. For BPD specifically, one needs to meet any combination of five of nine potential symptoms. Impulse control symptoms involve things like substance abuse, binge eating, reckless driving, etc. These often go hand in hand with self-harming behavior, which is often related to emotion-regulation abilities and impulse control. People with BPD experience intense emotions and have a difficult time getting back to their baseline level of emotion. Some patients express fears of abandonment; some have chaotic, turbulent, up-and-down relationships with people close to them.

Although the trial led by Dr. Ruocco and colleagues was small, it did generate hopeful results. Combined DBT and MST treatments led to a "rapid, significant, and clinically meaningful reduction in suicidal ideation" at the end of the 5-week study period, the team reported in the inaugural issue of *Nature Mental Health*. This reduction in suicidality was sustained at a 4-month follow-up assessment.

Conjoint DBT + MST was also associated with "significant reductions in depression and BPD interpersonal symptom severity," the team reported, "but neither effect was sustained at the 4-month follow-up. Importantly, there were no observed impacts of MST therapy on cognition, and there were no treatment-related serious adverse effects.

These initial results lead the team to suggest that the DBT + MST combined therapy is "feasible" to offer, and "warrants further exploration" in a larger placebo-controlled clinical trial (with some patients receiving a treatment that sounds and feels like MST but is not delivering actual stimulation to the brain). They also suggest that rTMS, a commonly used form of brain stimulation, might be tested in combination with DBT in suicidal patients with BPD. For now, they said, their results "represent a step toward addressing the long-standing problem of suicidality in BPD."

The researchers note that their results do not suggest that DBT alone is ineffective, rather that the combination of DBT and MST was associated with a more rapid reduction in suicidality compared with 5 weeks of DBT alone. They also note that on the basis of their study it was not possible to determine if improvements in interpersonal BPD symptoms in the combined therapy group "were a cause or consequence of reductions in suicidal ideation." That is a subject for future studies.

The research team also included senior member **Zafiris J. Daskalakis, M.D., Ph.D.**, BBRF Scientific Council, 2008 BBRF Independent Investigator, 2006 and 2004 Young Investigator; co-first author **Jenna M. Traynor Ph.D.**, 2022 BBRF Young Investigator; and **Daniel M. Blumberger, M.D.**, 2010 BBRF Young Investigator. Dr. Ruocco's 2014 BBRF Young Investigator grant, devoted to the work reported in the new paper, was supported by The Families for Borderline Personality Disorder Research.

Four Subtypes of Autism Spectrum Disorder Are Distinguished, Helping to Explain Individual Differences in Symptoms

Diagnostic Tools / Early Intervention: **Autism Spectrum Disorders**

Story Highlights:

Researchers used neuroimaging and genetics data to discover that a cohort of 299 individuals with autism spectrum disorder (ASD) “clustered” in four subgroups, each with distinct patterns of atypical functional connectivity in ASD-related neural networks. The findings promise to advance the search for new treatments.

Journal: *Nature Neuroscience*
March 9, 2023

Researchers led by a BBRF grantee have used a large set of neuroimaging data to identify distinct sets of alterations in functional connectivity that may help explain differences among individuals with autism spectrum disorder (ASD). The finding could have implications for the development of new treatments.

The autism “spectrum” refers to wide variations in the types of symptoms that affect those diagnosed, as well as the degree to which symptoms impact individual function. Social communication and interaction skills are usually affected, although to varying degrees. As noted by the U.S. Centers for Disease Control, people with ASD also may have restricted or repetitive behaviors or interests. In addition, some patients may have delays in acquiring language skills, movement skills, or cognitive and learning skills. Some may exhibit hyperactive, impulsive, or inattentive behavior; or have unusual eating or sleeping habits, gastrointestinal issues, or issues with mood, anxiety or fear.

“Our limited understanding of the neural mechanisms underlying ASD variability has impeded the development of therapeutic interventions,” notes a research team led by 2013 BBRF Young Investigator **Conor Liston, M.D., Ph.D.**, of Weill Cornell Medicine, reporting in *Nature Neuroscience*. Dr. Liston’s team sought to discover consistently identifiable subtypes of ASD as a

way of generating testable theories “about how different biochemical, genetic, and cellular processes may shape” the wide range of ASD’s clinical manifestations.

There was good reason to use neuroimaging data to try to discern ASD subgroups. Past functional magnetic resonance imaging (fMRI) studies have found that impaired social cognition and language processing in ASD are associated with atypical activity in the thalamus and visual areas of the brain, as well as in the salience network, composed of several brain regions that work together to determine which stimuli should command attention. Repetitive and ritualistic behaviors also have been linked in imaging studies with specific brain circuitry.

Dr. Liston and colleagues sought to discover how atypical connectivity contributes to individual differences in ASD symptoms and behaviors. They drew upon two large-scale fMRI datasets curated by the Autism Brain Imaging Data Exchange. The data analyzed was derived from 299 individuals with ASD and 907 neurotypical controls. The analysis enabled the team to relate functional connectivity patterns to three “dimensions” of ASD symptoms—those affecting verbal ability, social affect, and repetitive behaviors and restricted interests.



Conor Liston, M.D., Ph.D.

Weill Cornell Medicine

2013 BBRF Young Investigator

When study subjects with ASD were assessed according to this schema, the team found that they “clustered” in four subgroups, each with distinct patterns of functional connectivity in ASD-related neural networks. The same four subgroups emerged when the team applied the same functional-connectivity analysis to an independent sample of ASD patients.

The next step was to consider the connectivity data for the four ASD subgroups in the light of data on gene expression patterns in the brain. Of the approximately 21,000 human genes, many, but not all, are activated at different moments in brain cells in different regions, and activation patterns vary depending on what tasks the brain is performing. The team hypothesized that distinct genetic pathways may be important in subsets of ASD patients, and may confer risk for specific symptoms by impacting functional connectivity in ASD-related brain networks.

That is what the analysis revealed. Each of the four ASD subtypes was associated with distinct gene expression patterns and the biological processes they affect. This led to a number of interesting observations. Individuals in two of the subgroups, for example, were alike in being “highly impaired” by core ASD symptoms, the team noted, but differed notably in verbal ability, and had dissimilar patterns of atypical connectivity and gene expression. The other two subgroups “had average verbal ability” but differed in the degree to which they were impaired by two of the c

ore ASD symptoms, social affect and repetitive and restricted behaviors.

The four ASD subgroups identified by Dr. Liston and colleagues provide insight into the biological mechanisms “that may regulate changes brain function that lead to ASD behaviors,” the team said. The analysis also makes it possible, they said, to form “multiple testable hypotheses that could be explored in future studies.”

In ASD subgroup 4 for example, which is characterized by strong repetitive and restrictive behaviors and notably diminished “social affect,” i.e., signals to others about how one is feeling, atypical connectivity was linked with decreased expression of a gene called *HTR1A*. That gene encodes a cellular receptor for the neurotransmitter serotonin that has been associated in past research with severe repetitive behaviors and restricted interests. Expression of *HTR1A* is known to be reduced in people with ASD, which in turn is associated with stress and anxiety. Problems with serotonin signaling have also been implicated in altered reward processing in the brain, as well as impairments of the sensorimotor system during development—which contribute to repetitive and restrictive behaviors. These linkages suggest that drugs targeting the serotonin system could potentially be beneficial for reducing these behaviors in some people with ASD, the researchers note.

More broadly, the researchers say their results can generate testable ideas that can be explored in animal models of ASD and in future clinical studies. “They suggest distinct alterations in brain function that could be targeted using circuit-based neuromodulation” such as TMS or other brain-stimulation technologies. They also “predict distinct biological pathways that could help inform studies of drug targets specific to each ASD subtype,” the team said.

Functional Connectivity Brain Changes Involving Links Between Cognition and Motor Function Are Discerned in OCD Patients

Basic Research: **OCD**

Story Highlights:

A large international team found widespread functional connectivity aberrations in people with OCD, notably in the sensorimotor network. These could reflect impairments in suppressing irrelevant sensory, cognitive, and motor information, which may contribute to the inability of patients to inhibit undesired thoughts and images and repetitive behaviors or thoughts.

Journal: *Molecular Psychiatry*
May 2, 2023

The symptoms of obsessive-compulsive disorder (OCD) can be debilitating. Those affected experience intrusive, distressing, and often irrational thoughts (“obsessions”) which are accompanied by repetitive physical or mental acts (“compulsions”). OCD is quite common, thought to occur in 1% to 3% of the global population. It affects both young people and adults, and takes a vast variety of forms.

Efforts have been made to determine the biological underpinnings of OCD symptoms, based largely on functional brain scans in which those with the diagnosis are compared with those who are unaffected. These studies have directed the attention of the research community to a set of brain circuits referred to with the acronym CSTC (cortico-striato-thalamo-cortical). These circuits running between the brain’s cortex, striatum, and thalamus, are complex and involve feedback loops, i.e., flow between regions that are not uni-directional but which “feed back” to affect one another on an ongoing basis. In broad terms, the CSTC circuitry is involved in a wide array of brain processes: cognition, affect, motivation, and motor functions.

Recent research has also suggested involvement in CSTC circuitry of the brain’s sensorimotor circuits, which are involved in the generation and control of motor behaviors and the integration of information from the senses. This circuitry seems “particularly relevant to OCD” given its role in habit formation

and mechanisms that normally serve to inhibit non-essential, unwanted, or irrelevant information, notes a large international research team in a new paper appearing in *Molecular Psychiatry*. Dysfunction in this circuitry, they note, “could be related to the inability of OCD patients to suppress internally triggered repetitive and intrusive thoughts and behavior.”

The team, composed of over 100 investigators, many affiliated with a consortium called ENIGMA-OCD (Enhancing Neuro-Imaging and Genetics through Meta-Analysis), report on their most recent effort to probe functional connectivity in OCD patients, in part to discern how connectivity in those with OCD differs from that in healthy controls, and in part to test for biomarkers which might enable clinicians to distinguish people with OCD from those not affected.

The team’s four senior members included **Odile A. van den Heuvel, M.D., Ph.D.**, 2009 BBRF Young Investigator; **Paul M. Thompson, Ph.D.**, 2017 BBRF Distinguished Investigator; **Dan J. Stein, Ph.D., FRCPC**, 1991 BBRF Young Investigator; and corresponding author Guido A. van Wingen, Ph.D., of the University of Amsterdam, the Netherlands. Willem B. Bruin, Ph.D., was the paper’s first author.

Rather than focus on pre-determined regions of interest in the brain associated with circuitry already



Odile van den Heuvel, M.D., Ph.D.

*Amsterdam University Medical Center,
Netherlands*

2009 BBRF Young Investigator



Paul M. Thompson, Ph.D.

University of Southern California

2017 BBRF Distinguished Investigator

believed to be affected in OCD, the team investigated functional connectivity differences across the entire brain, using MRI data of 1,024 OCD patients and 1,028 healthy controls. Whole brain functional connectivity was assessed at both the regional and network (i.e., spanning regions) levels. The participants were scanned in 28 separate and independent samples within the overall ENIGMA project. The method used by the team is called “mega-analysis,” which involved pooling individual data across the 28 studies. This contrasts with the oft-used “meta-analytic” approach which synthesizes summary statistics from multiple studies to estimate an overall effect.

Most broadly, the mega-analysis revealed “widespread functional connectivity aberrations in OCD patients.” The most pronounced overall change relative to healthy controls was what the team calls “global hypo-connectivity,” with a few instances of hyper-connections. Hypo connections are circuits that show less connectivity than average; hyper connections show greater connectivity than typical.

Most notable among the differences in OCD patients was “significant hypo-connections located within the sensorimotor network,” something that had not been stressed in prior research, moving the team to suggest that on the basis of their analysis (if validated) “neural models of OCD should be revised” to incorporate this feature.

Although there had been some evidence of its involvement in past studies, “the sensorimotor network is often overlooked in OCD studies,” the team notes, and it is not typically included in functional connectivity scans that focus on particular circuits or networks postulated in advance to be involved in OCD. Altered connectivity in OCD patients could reflect impairments in the process (called sensorimotor gating) of suppressing irrelevant sensory, cognitive, and motor information, which may contribute to the inability of OCD patients to inhibit undesired thoughts and images and repetitive behaviors or thoughts.

The team found no aberrations in OCD subjects in what is generally considered the most important part of the CSTC, the frontostriatal loop (pathways that connect the frontal lobe with the basal ganglia, which mediates motor, cognitive, and behavioral functions).

Regarding another of their objectives, the team reported that despite observations of global hypo-connectivity in patients at the group level, their application of machine learning to the results showed that functional connectivity cannot at this time provide an accurate distinction between patients and controls. In other words, the signal is not sufficient, on its own, to serve as a reliable and highly accurate biomarker that clinical use would demand.

The team suggests that the difficulty in applying the widely observed “hypo-connectivity” signal to diagnosis has to do with OCD’s heterogeneity—the considerable variance among different individuals with the disorder, in terms of, for instance, sex, age of onset, severity, medication status, and perhaps most of all, symptoms. “Particular OCD subtypes may be characterized by different neural irregularities and functional changes,” the team said. Machine learning may eventually be able to make headway in defining biomarkers, but only after further studies that incorporate more detailed clinical information about participants and well as observation at different points in life and over the course of years. It is also possible that no biomarker applicable to the total set of OCD patients may be possible to establish, directly reflecting fundamental diversity within the group.

Other BBRF grantees on the team included: **Alan Anticevic, Ph.D.**, 2015 BBRF Klerman Prize winner, 2015 BBRF Independent Investigator and 2012 Young Investigator; **Chiang-Shan R. Li, M.D., Ph.D.**, 2009 BBRF Young Investigator; **Erika L. Nurmi, M.D., Ph.D.**, 2008, 2010 BBRF Young Investigator; **Christopher Pittenger, M.D., Ph.D.**, 2015 BBRF Independent Investigator, 2009, 2007 Young Investigator; **Blair H. Simpson, M.D., Ph.D.**, 2010 BBRF Independent Investigator; **Emil R. Stern, Ph.D.**, 2008 BBRF Young Investigator; **Philip R. Szeszko, Ph.D.**, 2009 BBRF Independent Investigator, 2003, 1998 Young Investigator; **Hein J. F. van Marle, M.D., Ph.D.**, 2022, 2018 BBRF Young Investigator.

In Young Men, Up to 20% of Schizophrenia Might Be Prevented If Cannabis Use Disorder Is Not Present

Basic Research, Diagnostic Tools/Early Intervention: **Substance Use Disorder, Schizophrenia**

Story Highlights:

Using Danish national health records, investigators found that cannabis use disorder (CUD) is contributing to a “steadily increasing proportion” of schizophrenia cases over the last 5 decades—a figure, in young men, they “conservatively” estimate at 15% for 2021. Such cases might have been “preventable” if CUD had not been present, they say.

Journal: *Psychological Medicine*
May 4, 2023

A large study involving the health records of 6.9 million people in Denmark over 5 decades has found “strong evidence” of a linkage between cannabis use disorder (CUD) and schizophrenia, in both men and women. The magnitude of the relationship, the researchers say, is consistently larger among males than females, and much larger in young males, especially those aged 16 to 25.

Using statistical methods based on Danish national health record data, the investigators “conservatively” estimate that in 2021, at least 15% of cases of schizophrenia in males “may have been prevented” if cannabis-use disorder in those individuals had not been present. In females the corresponding number is 4%.

As attitudes about cannabis use have generally become more tolerant, especially in Western nations including the U.S., Canada and Europe, with the steady process of legalization over last decade, so too has the prevalence of impressions of cannabis use as being “harmless,” the researchers note.

They disagree, echoing cautions repeatedly expressed in recent years by **Nora Volkow, M.D.**, a member of BBRF’s Scientific Council who is Director of the U.S. National Institute on Drug Abuse. Dr. Volkow was a member of the team that conducted the Danish study, which appeared in *Psychological Medicine*.

With specific reference to schizophrenia and the contribution of heavy and regular cannabis use coinciding with increased schizophrenia incidence over the last several decades, the researchers write: “Although cannabis use disorder is not responsible for most schizophrenia cases in Denmark [the nation whose population served as the basis for the new research], CUD appears to contribute to a non-negligible and steadily increasing proportion” of schizophrenia cases over the last 5 decades.

In young males, aged 21-30, and possibly up to age 40, the proportion of schizophrenia cases that may now be avoided if CUD is not present “may even be as high as 25% to 30%,” the researchers note. For this reason, the researchers said they consider CUD to be “a major modifiable risk factor for schizophrenia, particularly among males.”

Reasons for this remarkable statistic are not known with certainty, but the researchers propose that increased cannabis usage in the general population, accompanied by increased potency of cannabis products (based on the concentration of active ingredient, THC) “likely” help explain what they found to be the steadily growing portion of new schizophrenia cases that might be prevented in the absence of heavy and regular cannabis use, especially by the young male users.



Nora Volkow, M.D.

Director, National Institute on Drug Abuse/NIH
BBRF Scientific Council

The research team, which was led by Dr. Carsten Hjorthøj, an epidemiologist at Mental Health Services in the Capital Region of Denmark and the University of Copenhagen, and which included collaborators from the U.S. National Institutes of Health including Dr. Volkow, used a nationwide Danish healthcare database consisting of every person in Denmark who was between the ages of 16 and 49 at some point in the years 1972 to 2021. This included the health records of over 6.9 million people, 51% of whom were male. Of that number, a total of 45,327 were diagnosed with schizophrenia at some point in the 5-decade period of the study; 60,563 were diagnosed with cannabis use disorder in the same period, three-fourths of whom were male.

Such a broad-based study, with comprehensive medical follow-up data amounting to, in this case, over 129 million person-years, generates statistical data with a very low potential error rate. The main limitation is that illnesses such as schizophrenia and CUD “appear” in the Danish national records only when actually diagnosed and entered by a medical professional. There are some number of cases of both illnesses that were never diagnosed; hence, the statistical findings of the study tend to err on the low side—they are “conservative.” The study’s findings are associational; they cannot prove a causal impact of cannabis use disorder in the onset of schizophrenia.

“While we cannot be certain of the proportion of [cannabis]-exposed individuals who might have developed schizophrenia even in the absence of CUD, it is unlikely that all of the [observed] associations between CUD and schizophrenia would be explained by confounding facts,” they noted.

Cannabis use disorder is defined by a persistent desire or unsuccessful efforts to cut down or control cannabis use. A great deal of time is spent in activities necessary to obtain cannabis, use cannabis, or recover from its effects. Craving, or a strong desire or urge to use cannabis, is typically present.

Further research is needed, the team says, on the relation of frequency of cannabis use as well as THC concentration in both males and females with the association with schizophrenia, as well as on potential causal linkages. They are clear, however, in their conclusion: while noting “significant differences between the sexes in response to the acute and long-term effects of cannabis,” they say their results suggest “a relationship between intense use of cannabis and risk of developing schizophrenia,” at the level of entire populations. At the level of individual cannabis users, “risk occurs in both sexes but especially appears higher in young males.”

It is the team’s belief that “an increasing proportion of cases with schizophrenia may be avertible by preventing CUD.” They express the hope that their findings will encourage health-care providers to effectively screen for and treat cannabis use disorder, generally, and particularly in young males who may be using cannabis regularly. They, it appears, are most vulnerable to the as yet unexplained impact of cannabis—most of all, highly potent and regularly used cannabis—on the genesis of psychotic disorder.

“The entanglement of substance-use disorders and mental illnesses is a major public health issue, requiring urgent action and support for people who need it,” noted Dr. Volkow. “As access to potent cannabis products continues to expand, it is crucial that we also expand prevention, screening and treatment for people who may experience mental illness associated with cannabis use. The findings from this study are one step in that direction and can help inform decisions that health care providers may make in caring for patients, as well as decisions that individuals may make about their own cannabis use.”

Brain Changes Related to Adolescent Cannabis Use Are Found to Persist Into Young Adulthood

Basic Research, Diagnostic Tools/Early Intervention: **Substance Use Disorder, Schizophrenia**

Story Highlights:

In an analysis of brain scans of 799 teens made 5 years apart, researchers found cannabis use was associated with accelerated thinning of the prefrontal cortex, a key aspect of brain development.

Journal: *Molecular Psychiatry*
June 28, 2023

More than a third of 12th graders report having used marijuana, also called cannabis. And 78 percent of first-time marijuana users are between the ages of 12 and 20 years. Concerningly, cannabis use during adolescence has been linked in some studies to long-term detrimental effects on impulse control and executive functioning, such as planning and decision-making. Cannabis-related brain changes are not well studied, but new research mapping such changes in 799 adolescents suggests how cannabis use in middle to late adolescence may alter development of the brain's cerebral cortex.

A team led by 2020 BBRF Young Investigator **Matthew Albaugh, Ph.D.**, of the University of Vermont, conducted a large longitudinal brain imaging study (following the same individuals over a period of time) of adolescent cannabis use. They used magnetic resonance imaging (MRI) brain scans from 14-year-olds in Europe who reported having never used cannabis, and did follow-up scans 5 years later, when the participants were about 19 years old. Their data showed significant brain changes in those who reported using cannabis in the interval between the scans compared to those who said they did not. The findings were reported in *JAMA Psychiatry*.

In the brain, a signaling system called the endocannabinoid system contributes to the regulation of stress response, anxiety, memory, pain, and motivated behavior. Additionally, endocannabinoid signaling plays a key role in the development, maturation, and sculpting of neural circuits, processes that continue through adolescence. The system includes a number of molecules, including anandamide and 2-AG (2-arachidonoylglycerol), which activate two cannabinoid receptors in the brain, called type 1 (CB1) and type 2 (CB2).

Cannabis engages with the endocannabinoid system: THC, the main psychoactive component, binds tightly to the CB1 receptor. This interaction activates the endocannabinoid system, and may be detrimental during adolescence, when the system is still developing.

"The potential association of cannabis use with adolescent development represents an increasingly relevant public health issue, particularly given evidence of increased problematic cannabis use among adolescents in areas where recreational cannabis use has been legalized," the team noted in their paper.



Matthew D. Albaugh, Ph.D.

University of Vermont

2020 BBRF Young Investigator

Comparing MRI scans at baseline, when participants were 14, and 5 years later, the team found a negative correlation between self-reported cannabis use and prefrontal cortex thickness. The prefrontal cortex is involved in executive functioning and is one of the last parts of the brain to mature—it may not be fully developed until around age 25. This part of the brain normally thins with age, but the study found that on average, cannabis-related thinning was greater in cortical regions that normally show the most significant age-related thinning.

Additionally, the data indicated that teens who used more cannabis had thinner prefrontal cortices than those who used less, indicating that the brain changes are dose-dependent. The team found no association between baseline prefrontal cortex thickness and cannabis use at the 5-year follow-up, suggesting that preexisting anatomical differences did not predict who would later use cannabis.

To find out whether the brain regions with accelerated thinning were associated with the location of CB1 receptors, the team used data from positron emission tomography (PET) scans, which can map CB1 receptors in the brain. This data came from a separate group of young men because, the team pointed out, PET is an invasive test and not ethical to perform on minors. They confirmed that the pattern of cannabis-related thinning was particularly associated with brain areas containing large numbers of CB1 receptors.

To determine whether the prefrontal cortex changes were associated with behavior changes at the 5-year follow-up, they tested three aspects of impulsiveness: attentional, non-planning, and motor. Thinning in the prefrontal cortex was significantly associated with more attentional impulsiveness at the 5-year follow-up, controlling for sex, baseline age, baseline brain volume, baseline pubertal development, verbal IQ, and performance IQ. Other cognitive measures did not change with cannabis use.

In rats, past research has shown that THC exposure disrupts normal brain development by inducing premature pruning of the synaptic connections between nerve cells and degeneration of the complex branching structure of nerve cells in early adulthood. Dr. Albaugh and colleagues hypothesize that the cannabis-related thinning shown in their study may be due to a similar mechanism. However, more research is needed to determine the exact mechanisms by which cannabis leads to the observed changes in the prefrontal cortex of teens.

It remains to be determined how much cannabis use is problematic for adolescents, and whether the observed changes are linked to long-term psychological or cognitive effects. But overall, “the findings underscore the importance of further longitudinal studies of adolescent cannabis use, particularly given increasing trends in the legalization of recreational cannabis use,” the team concludes.

The team also included: **Deepak D’Souza, M.D.**, 2013 BBRF Young Investigator; **Henrik Walter, Ph.D., M.D.**, 2017 BBRF Distinguished Investigator; **Robert Whelan, Ph.D.**, 2015 BBRF Young Investigator; and **Alexandra Potter, Ph.D.**, 2009 BBRF Young Investigator.

Gliosis, an Immune Response to Brain Injury, Is Found in Brains of Recovered COVID Patients With Lasting Depression and Cognitive Symptoms

Basic Research: **Long COVID**

Story Highlights:

Researchers studying recovered COVID patients with lingering depression and cognitive symptoms (COVID-DC) found evidence of gliosis, an immune response to brain injury. Most affected were two brain regions: the ventral striatum and dorsal putamen. Higher readings in the latter area also were found to correlate with motor slowing. Tests of drugs to reduce gliosis may now follow in COVID-DC patients.

Journal: *JAMA Psychiatry*
May 31, 2023

In the weeks, months, and even years following a COVID infection, it is not uncommon for those who have recovered from the virus's acute symptoms to be dogged by persistent depressive symptoms. Often, such depression is accompanied by cognitive symptoms; sometimes the latter can occur in the absence of depression symptoms. Researchers have been trying to get a fix on what causes these post-infection brain-based symptoms, and a new study suggests one possible source.

Scientists believe that a majority of the global population has by now experienced at least one acute episode of COVID illness. Mild to moderate symptoms, predominantly respiratory, but which often include flu-like fever, headache, and/ or muscle pain or weakness and fatigue, are also commonplace, occurring in an estimated 95% of those who are infected with the Omicron variant and about 80% of those infected with the original SARS Co-V-2 strain of the virus.

Depressive symptoms with or without cognitive impairment are quite prevalent, note researchers who conducted the new study, appearing in *JAMA Psychiatry*. They are estimated to have occurred in about 15% of those infected with the original strain of COVID, who of course had not been vaccinated. But these symptoms also occur in about 5% of triple-vaccinated people who are exposed to the Delta and Omicron variants

of the virus. Many of the symptoms, which include anhedonia (inability to feel pleasure), slowing of motor skills, low motivation and low energy, and short-term memory impairment, can persist for years and are thus, say the researchers, "a major public health problem," especially considering the vast number of people who are infected by COVID every year.

The team, led by **Jeffrey H. Meyer, M.D., Ph.D.**, of the University of Toronto, Canada, a 2015 BBRF Distinguished Investigator and 2000 and 1998 BBRF Young Investigator, tested the hypothesis that COVID-DC (COVID with lingering depression and/ or cognitive symptoms) may involve a condition called gliosis, marked by a proliferation of glial cells in the brain. Gliosis has been linked with depression and other neuropsychiatric conditions, as well as neurodegenerative disorders such as Alzheimer's disease. Glial cells, which include microglia and astroglia, are often described as "helper" cells. Abnormal proliferation of glial cells is known to occur after damage to the central nervous system or brain. The researchers explored the idea that gliosis might also be among the possible consequences of COVID infection, whose effects are known from postmortem and other studies to directly affect brain cells.

The researchers recruited 40 people for their study, conducted in 2021 and 2022. Twenty subjects had mild to moderate cases of acute



Jeffrey H. Meyer, M.D., Ph.D.

University of Toronto

2015 BBRF Distinguished Investigator;

2000 and 1998 Young Investigator

COVID infection from which they had recovered, but with continuing depression and/or cognitive symptoms (COVID-DC). The other 20 were healthy controls. Participants were about 60% female and on average in their early thirties. Of the 20 who had COVID-DC, 60% developed depressive or cognitive symptoms 0 to 6 months after being diagnosed with COVID; in 40%, the interval was 7 to 24 months.

The 40 participants were given PET scans, an imaging technology that is able to measure the prevalence ("distribution volume") of a molecule called translocator protein. This measure, called TSPO VT, is used as an index to detect the presence and severity of gliosis in the brain. Measurements in the healthy controls provided a reliable contrast with COVID-DC patients since they had been recruited and scanned for other research prior to the beginning of the pandemic.

The PET scans measured TSPO VT in five brain regions, all of which, when injured, can exhibit gliosis, and all of which are implicated in the depressive and cognitive symptoms associated with COVID-DC. The five regions are: the dorsal putamen, ventral striatum, prefrontal cortex, anterior cingulate cortex, and hippocampus.

At the time of their PET scans, in addition to depression, 30% of those with COVID-DC reported headaches and 15% confusion; many also reported continuing physical symptoms such as nasal congestion or runny nose, fatigue, and aches and pains. 45% had suffered

a major depressive episode prior to being infected with the COVID virus.

Compared with the controls, participants who had COVID-DC were found to have elevated TSPO VT levels, particularly in two brain regions: the ventral striatum and dorsal putamen. Higher readings in the latter area also were found to correlate with motor slowing in those participants.

What do these findings mean? Higher TSPO VT levels are understood to signal "greater density of activated microglia, and to a lesser extent, astroglia." This is evidence that gliosis is occurring, and the researchers said that one explanation was that such an inflammatory response may be a direct response to injury, with the greatest injury, based on this evidence, occurring in the two cited regions. There is good evidence in other brain illnesses that higher TSPO VT also occurs, for example, in the hippocampus in Alzheimer's and in other regions in obsessive-compulsive disorder, as well as following brain injury or stroke.

"Injury to the ventral striatum and dorsal putamen is a plausible explanation for evidence of gliosis and is consistent with many symptoms observed in COVID-DC," the team said. Aberrant ventral striatal function may lead to anhedonia, and dorsal putamen injury is associated with motor slowing and low motivation or energy, they added.

The possible injuries to these affected regions could be the result, they said, of direct virally induced injury to the striatum and projections from other brain areas that lead to it, combined with "additional brain-wide effects of virally induced injury and elevated bodily inflammatory signaling-initiated brain gliosis." Interestingly, gliosis may have both damaging and curative dimensions, but chronic gliosis is generally associated with neuropsychiatric disease.

For this reason, the researchers said, "clinical trials of novel interventions for COVID-DC may consider suppressing adverse consequences of gliosis or suppressing gliosis entirely." They suggested clinical tests of two investigational drugs now in phase 2 and 3 tests: one that targets translocator protein (TSPO) and the other, called a P2X7 inhibitor, thought to reduce proliferation of microglia. It remains possible, the researchers said, that gliosis, while present, may play a subordinate role to other pathology, such as injury to neurons, in causing or promoting the depressive and cognitive symptoms of COVID-DC.

The team also included **Romina Mizrahi, M.D., Ph.D.**, a 2014 BBRF Independent Investigator and 2010 BBRF Young Investigator; **Nathan Kolla, M.D., Ph.D.**, a 2013 BBRF Young Investigator; and **M. Ishrat Husain, MBBS, M.D.**, a 2019 BBRF Young Investigator.

In PTSD, Evidence That a Single Ketamine Infusion May Enhance Extinction of Recalled Traumatic Memories

Next-Generation Therapies: PTSD

Story Highlights:

A pilot study generated evidence that a single ketamine infusion may have enhanced extinction of traumatic memories, following their initial retrieval, in PTSD patients. Ketamine combined with talk therapy is a potential novel future intervention for PTSD and anxiety disorders, the researchers concluded.

Journal: *Neuropsychopharmacology*
June 3, 2023

Intrusive memories of trauma are among the classic symptoms of PTSD that can haunt and impair sufferers. The traumatized individual re-experiences the traumatic event via a memory which carries the event's original emotional intensity and vividness. These intrusions can be spurred by stimuli or cues in the environment which are, in themselves, innocuous. An example often given is the sound of a car backfiring that triggers memories of combat in a PTSD-affected war veteran.

But what if traumatic memories could be rewritten—overwritten and replaced with other memories, or in some other way modified so that the memory-induced fear response to the original trauma is, in the language of neuroscience, “extinguished”?

A first-line treatment for PTSD aims at fear extinction (or reduction) via exposure therapy. It works very well in some patients, although its benefits are often not enduring. In exposure therapy, the therapist provides a safe environment in which to expose the traumatized individual, often gradually, to the original traumatic memory and things they fear or avoid. This process helps the patient to master their response to fear triggers or cues and to better tolerate the original traumatic memory. When successful, the involuntary connection between environmental triggers and memory of the original trauma is at least for the time being interrupted.

This way of taming fear is thought to take advantage of a phenomenon called memory reconsolidation. In reconsolidation, recall of a memory opens a window in time—in humans it lasts from about 10 minutes following recall to as long as 10 hours—during which the memory exists in what researchers call a “labile” state. It can potentially be modified, before it is once again “reconsolidated” in the brain areas in which memory traces are stored (the hippocampus and amygdala).

Researchers at Yale University led by 2015 BBRF Independent Investigator **Ilan Harpaz-Rotem, Ph.D.**, have recently reported on their efforts to study what happens in the brain when the drug ketamine is administered to PTSD patients, followed by an intensive exposure treatment. They and others have been exploring whether ketamine treatments, alone or in combination with psychotherapy (including exposure therapy), might improve outcomes in PTSD, perhaps by enhancing mechanisms involved in fear extinction.

There have been indications of ketamine's potential utility in PTSD and anxiety in various studies, but it is still not understood how changes in the brain induced by the drug might enhance the therapeutic process in fear and anxiety patients. Developed as an anesthetic, ketamine has been shown to rapidly reduce symptoms of severe, treatment-resistant



Ilan Harpaz-Rotem, Ph.D.

Yale University

2015 BBRF Independent Investigator

depression when delivered at doses far below those used in anesthesia. A co-author of the new study, **John H. Krystal, M.D.**, was among the first to test and demonstrate the benefits of ketamine in severely depressed patients. Dr. Krystal is a member of the BBRF Scientific Council, winner of the BBRF Colvin Prize for his work on ketamine, and a 2006 and 2000 BBRF Distinguished Investigator and 1997 Independent Investigator. **Ben Kelmendi, M.D.**, also of Yale and a 2016 BBRF Young Investigator, was also a co-author on the new paper, which appeared in *Neuropsychopharmacology*. Co-first authors were Drs. Or Duek and Nachshon Korem of Yale.

Team leader Dr. Harpaz-Rotem devoted his 2015 BBRF grant to testing whether a single dose of ketamine, followed by intensive prolonged exposure therapy, enhances therapy's effectiveness. Exploring in the newly published study why this might be so, the team hypothesized that ketamine temporarily increases the brain's capacity to rewire its connections, possibly affording a window of opportunity to enhance the effects of trauma memory extinction during therapy. Past research indicates ketamine promotes neurogenesis (birth of new neurons), cell proliferation, and the creation of new synapses, which all occur in parts of the brain involved in memory reconsolidation.

Twenty-seven patients with moderate to severe PTSD persisting for a year or longer, and in most cases based on trauma that occurred over 10 years prior, were randomly assigned to receive either a single sub-anesthetic dose of ketamine or the anti-anxiety

drug midazolam following retrieval of each participant's traumatic memory. Then, 24 hours following the infusion of either drug, all participants received 4-day trauma-focused psychotherapy. Symptoms were clinically assessed prior to treatment, at the end of treatment, and 30 days post-treatment. Brain activity and structural features were also assessed at these points, using MRI brain scanning. A large fraction of participants had current episodes of major depressive disorder in addition to PTSD, and most had histories of substance dependency. Most were between 35 and 45 years old and over one-third were female. Sources of trauma varied and had combat, sexual, and violent precipitants, among others.

PTSD symptoms improved about equally in the two groups. But there was evidence that the single ketamine infusion might have enhanced extinction of traumatic memories, following their initial retrieval. The evidence for ketamine's utility was in differences noted between participants in the ketamine and midazolam groups. In patients who received ketamine, the team noted diminished reactivity of the amygdala to the recall of traumatic events and a weakening of connectivity between the amygdala and hippocampus. There was evidence, too, that ketamine promoted therapeutic changes to key bundles of white matter (composed mainly of axons that connect brain regions) in a part of the brain called the uncinate fasciculus (which connects limbic areas with the amygdala and other areas involved in memory retrieval).

The researchers said it was possible that the interaction of psychotherapy and ketamine yields an advantage in the therapeutic reorganization of synapses, but the current pilot study was not able to prove this. "Further exploration of dosage, frequency, and timing of ketamine when combined with psychotherapy is needed" in future studies, they said. Their results were consistent, they said, with a recent study in participants with harmful drinking patterns. "While ketamine [alone] was ineffective in reducing drinking, when administered in conjunction with alcohol cues, ketamine interfered with the reconsolidation of alcohol-reward memories and significantly reduced drinking."

In the current study, however, all participants received psychotherapy and thus it was impossible to determine the specific effect of ketamine alone and the effect of the combined therapy. Nevertheless, the researchers concluded, since "the enhancement of post-retrieval [fear] extinction presented in our study was demonstrated using real-life traumatic events, the [clinical] applicability of this procedure is high and it might serve as a potential novel future intervention for PTSD and anxiety disorders." Based on the results of this BBRF-funded study, Dr. Harpaz-Rotem has received funding from the NIMH to further investigate the potential of ketamine to enhance the effect of exposure therapy in a larger sample of patients.

Rapid-Acting Pill to Treat Postpartum Depression is Approved

Next-Generation Therapies: **Depression**

Story Highlights:

The FDA has approved the first short-course, rapid-acting oral treatment for patients with postpartum depression (PPD). Called zuranolone (marketed as Zurzuva), it is given in pill form for 14 days and begins to reduce severe PPD symptoms in as little as 3 days.

Journal: *American Journal of Psychiatry*
July 28, 2023

On August 4th, the U.S. Food and Drug Administration (FDA) approved zuranolone, the first oral medication designed to treat postpartum depression (PPD) in adults. The drug, which is rapid-acting, was developed by Sage Therapeutics and Biogen, and will be marketed under the name Zurzuva.

"Postpartum depression is a serious and potentially life-threatening condition in which women experience sadness, guilt, worthlessness—even, in severe cases, thoughts of harming themselves or their child," said Tiffany R. Farchione, M.D., director of the Division of Psychiatry in the FDA's Center for Drug Evaluation and Research. "Because postpartum depression can disrupt the maternal-infant bond, it can also have consequences for the child's physical and emotional development. Having access to an oral medication will be a beneficial option for many women coping with extreme, and sometimes life-threatening, feelings."

Because of its accessibility, zuranolone is an important advance. The first-ever rapid-acting medicine for postpartum depression, brexanolone, has been on the market since 2019. Brexanolone is administered via continuous infusion in a medical facility over a period of about 60 hours. While zuranolone, like brexanolone, can reduce symptoms of severe depression within 3 days of its administration, it is taken in pill form. The FDA recommended a dosage of

50mg for zuranolone, taken once daily for 14 days.

The efficacy of zuranolone for the treatment of PPD in adults was demonstrated in two randomized, double-blind, placebo-controlled, multicenter studies. The trial participants were women with PPD who met the Diagnostic and Statistical Manual of Mental Disorders ("DSM") criteria for a major depressive episode and whose symptoms began in the third trimester or within four weeks of delivery.

The results of one of those pivotal trials have just been published in the *American Journal of Psychiatry*. The research team was led by Kristina M. Deligiannidis, M.D., of Zucker Hillside Hospital/Northwell Health in New York. **Marlene Freeman, M.D.**, a 2000 and 1998 BBRF Young Investigator, of Massachusetts General Hospital and Harvard Medical School, was a member of the team.

The researchers note in their paper that about 17% of women globally develop PPD either during pregnancy or following childbirth, and that the condition is generally underdiagnosed and often untreated, exposing mothers and their newborns to considerable health risks that in severe cases of PPD includes risk of suicide in affected mothers. Death from suicide accounts for about 20% of all postpartum maternal deaths. The risk of PPD is about twice as great in women with



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*Massachusetts General Hospital
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 2000, 1998 BBRF Young Investigator



Cynthia Neill Epperson, M.D.
University of Colorado School of Medicine
 2005 Independent Investigator
 1997, 1995 Young Investigator

a family history of psychiatric illness, according to the researchers.

Knowledge about what causes PPD has grown markedly, thanks to basic research conducted over the last 25 years. **Cynthia Neill Epperson, M.D.**, who received three BBRF grants from 1995 to 2005, and others, revealed the possible role of the inhibitory neurotransmitter GABA in the illness. It is thought by many that depression occurring during the perinatal period is distinct in causation from depression at other times of life. Pronounced fluctuations in reproductive hormone concentrations—and the way in which some women respond to these—is thought to play a central role in onset. Notably, levels of the hormone allopregnanolone, which rise during pregnancy, peak in the 3rd trimester, then plummet following childbirth, appear to alter functional connectivity in the brain and may affect GABA-A receptors. Both brexanolone and zuranolone modulate the activity of these receptors.

Dr. Deligiannidis and colleagues enrolled 196 patients with severe PPD (accompanied in many cases by moderate to severe anxiety) in their randomized, double-blinded clinical trial. Half received 50 mg/day of zuranolone over 14 days, and half a placebo. 170 completed the trial. The participants were about 30 years old, on average; 25% identified as Black or African American, 33% as Hispanic or Latina, and 69% as White. PPD onset was in the 3rd trimester for one-third of the women, while onset came within 4 weeks after childbirth for two-thirds. 82% never had PPD previously. About

15% continued to use standard antidepressant medicines during the trial, in addition to either zuranolone or placebo. The women were followed for 45 days from the beginning of the trial, although they were also assessed at days 3, 15, and 28.

“Women with PPD receiving zuranolone demonstrated statistically significant and clinically meaningful improvements in depressive symptoms at day 15 compared with the placebo group,” the team reported. “The effects were rapid (by day 3), were sustained at all measured time points through day 45, and were observed across all measured [indices], reflecting a broad overall improvement in depressive symptoms. These benefits were mirrored in patients’ self-reported assessments,” the team noted. Clinically important symptoms of anxiety and insomnia also responded more to zuranolone than to placebo. The duration of the antidepressant impact of zuranolone beyond 45 days remains to be determined.

The team reported that zuranolone was “generally well tolerated.” All adverse events related to the treatment were mild or moderate, and mostly involved sleepiness, dizziness or a sedative effect. Some patients in the zuranolone group reporting such symptoms had their dosage reduced to 40mg/day, and 14 of 16 such participants did complete the trial.

The trial studied the drug only in women with severe PPD; the impact in women with less severe PPD was not studied. Participants were not permitted to breastfeed during the trial, since there is as yet no conclusive data on potential impacts from zuranolone (this may be studied in future research). Also, there was a strong placebo effect in the trial, which was attributed by the team to the amount of attention given to each participant—8 visits from the clinical team over the 45 days of the trial. Such attention has been linked with the placebo effect in past trials of antidepressants.

Based on this trial and another Phase 3 trial which tested zuranolone at about 40mg/day for 14 days, the FDA approved the medicine—the first short-course, rapid-acting oral treatment for patients with PPD.

The trial was funded by Sage Therapeutics and Biogen. Nine of the 14 authors of the study paper are employees and may hold stock in the companies. Other team members reported research and/or advisory or consulting relationships with the companies.

A Connectivity Signature Predicting Response to Antipsychotic Therapy is Identified in First-Episode Psychosis Patients

Diagnostic Tools / Early Intervention: **Psychosis, Schizophrenia**

Story Highlights:

Researchers report encouraging news in the search for robust biomarkers to predict treatment response to antipsychotics in individuals with first-episode psychosis. The predictors they found using functional brain imaging involved connectivity between the cerebellum and the cerebral cortex, where lower connectivity at baseline predicted better response to antipsychotics.

Journal: American Journal of Psychiatry
August 30, 2023

In people who experience a first psychotic episode—often the prelude to schizophrenia and related disorders—the individual's response to antipsychotic medicines can be crucial, and typically, varies considerably from patient to patient.

It is widely considered that how well a first-episode patient responds to antipsychotic medications often affects how the patient fares over the long-haul—both in terms of psychosis symptoms and how well they can function in society. “Identification of predictors of response at an early stage of illness would help physicians make optimal individualized treatment plans and benefit long-term quality of life for patients,” note a team of researchers in a newly published paper in the *American Journal of Psychiatry*.

The team reports encouraging news in its search for robust biomarkers that might predict treatment response to antipsychotics. They were led by **Anil K. Malhotra, M.D.**, of the Feinstein Institutes for Medical Research and Zucker Hillside Hospital. Dr. Malhotra is a member of BBRF's Scientific Council, a 2006 and 2001 BBRF Independent Investigator and a 1999 Young Investigator. The new paper's first author is Hengyi Cao, Ph.D., a 2018 BBRF Young Investigator whose grant was devoted to using functional imaging to understand behaviors in psychotic disorders. Four other BBRF grantees were among the co-authors.

MRI-based functional brain imaging has been a key tool in attempts to understand how connectivity in the brain changes in people with psychosis. The knowledge gained to date has not, however, yielded biomarkers reliable enough across the full spectrum of patients to be able to predict treatment response or long-term symptom trajectory. Drs. Malhotra, Cao and colleagues developed and tested a method aimed at combining several distinct modalities in which fMRI is used to observe connectivity in the brain. It's possible, for example, to look at network connections in the brain when the brain is in a “resting state”; as well as in various active states that can be induced in test subjects by asking them, during the scan, to perform various kinds of tasks. Different tasks make demands upon different brain regions, or different networks spanning brain regions.

The team combined multiple fMRI paradigms with the hope of identifying neural traits most predictive of response to antipsychotic treatment—across the entire brain, not just in one specific region of interest. Provided such connectivity traits were identified, the team hoped to be able to predict, using modeling based on machine learning, the degree to which an individual patient's symptoms would be reduced when they were put for the first time on a regimen of antipsychotic medicine.



Anil K. Malhotra, M.D.

*Feinstein Institutes for Medical Research/Zucker
Hillside Hospital*
BBRF Scientific Council,
2006 and 2001 BBRF Independent Investigator;
1999 Young Investigator

Two groups of patients were recruited. All were in the early stages of psychotic illness; each had cumulatively taken antipsychotics for less than 2 weeks since their initial psychotic episode. One group comprised 49 patients with first-episode psychosis (30 were male, average age about 24). A second group of 24 similar patients (20 males, average age 22) was used as a “validation sample,” to test whether any connectivity biomarkers identified in the main sample could be replicated in their predictive accuracy. Patients in both groups were carefully assessed and imaged using different fMRI modalities at “baseline,” after which each was randomly assigned to begin treatment on either risperidone or aripiprazole for 12 weeks. The severity of psychosis symptoms was assessed multiple times during the 12 weeks. Computer-based modeling was used to “train” a model that might enable identification of a connectivity-based biomarker based on the fMRI scans made before treatments began that would predict how well each patient responded to the 12 weeks of antipsychotic treatment.

The researchers succeeded in identifying “a functional connectome-based neural signature for the prediction of individualized treatment outcome in patients with first-episode psychosis.” There were both “positive” and “negative” predictors of treatment response. Positive predictors were mainly connections between the cerebellum and the cerebral cortex, where lower connectivity at baseline predicted better response to antipsychotics. The researchers noted that this finding was consistent with their past findings that increased

connectivity between cerebellum and cortex was consistently present, and abnormal, in people with psychotic disorders; also, that higher connectivity between cerebellum and cortex tended to predict worse clinical outcome after 2 years of continuous antipsychotic treatment.

“These lines of evidence converge,” the team said, “to show that cerebellar-cortical hyper-connectivity is a highly robust pathological finding in psychosis,” and “has the potential to be clinically used as a predictor of illness development and prognosis.” The stronger connectivity may result from the dysregulation of dopamine in cortical cognitive systems, the team said. Dopamine receptors are the target of antipsychotic medicines, but they are located in abundance throughout the brain. The new evidence helps identify where at least some of the pathology underlying psychosis resides.

The study also identified connectivity patterns that negatively predicted response to antipsychotic medicines—mainly connections within cortical cognitive systems. Taken together, the positive and negative predictors validated a connectome-based functional signature as a promising early predictor for individualized response to antipsychotic treatment in first-episode psychosis.

The predictions generated by the modeling in this study were based on data from individual patients and predicted results in each with considerable accuracy. The difference, on average, between the psychotic symptom score predicted by the

model and the actual score in each patient after 12 weeks of treatment was about 1.6 (the actual scores, on average, were about 18-20 at baseline and about 8-9 following 12 weeks of therapy). This relatively small variance between prediction and actual post-treatment score suggested to the team the potential of the connectivity signal to “assist clinical judgment for individual patients.” The results did not vary between the two medicines that patients were randomly assigned to take (although the sample size was small).

In addition to calling for replication of their results in larger and more diverse groups of patients, the team suggested that their method might be used to investigate possible signatures of outcomes and responses to treatment in different kinds of symptoms, for instance negative symptoms (affecting cognition and social functioning) in schizophrenia.

The team also included: **Todd Lencz, Ph.D.**, 2013 BBRF Independent Investigator and 2001 Young Investigator; **Juan A. Gallego, M.D.**, 2013 BBRF Young Investigator; **Anita D. Barber, Ph.D.**, 2009 BBRF Young Investigator; and **Delbert G. Robinson, M.D.**, 2005 BBRF Independent Investigator.

Anti-Inflammatory Medicine Reduced Positive Symptom Severity in Chronic Schizophrenia Patients with Elevated Inflammatory Markers

Next-Generation Therapies: **Schizophrenia**

Story Highlights:

A small clinical trial testing the anti-inflammatory drug canakinumab in a group of chronic schizophrenia patients with elevated blood markers of inflammation found that marker levels were reduced at 8 weeks, accompanied by a measurable reduction in severity of positive symptoms.

Journal: *Brain, Behavior and Immunity*
October 15, 2023

In recent years, a steadily growing body of evidence has indicated an association between elevated levels of inflammation and psychiatric illness.

The word “association” is very important in this context: it means that, in schizophrenia, for example, some fraction of patients have significantly elevated markers of inflammation. But as to the key question of cause and effect, the jury is out. Does inflammation contribute to causation? Or does having the illness in some way cause inflammation levels to rise? Or are the two phenomena merely coincident?

A research team led by two BBRF grantees, **Thomas W. Weickert, Ph.D.**, and his wife, **Cynthia Shannon Weickert, Ph.D.**, has just reported in the journal *Brain, Behavior and Immunity* on the exploration of this specific question: in patients with schizophrenia with elevated levels of inflammation, would administering a drug to reduce the inflammation have any impact on reducing schizophrenia symptoms?

Dr. Thomas Weickert’s 2016 BBRF Independent Investigator award was devoted to testing a new anti-inflammatory treatment in schizophrenia. Dr. Cynthia Weickert, a 2004 BBRF Independent Investigator and 2001 and 1999 BBRF Young Investigator, conducted work in schizophrenia patients that suggested elevated immune system activity. The Weickerts are currently at SUNY Upstate Medical University and Neuroscience

Research Australia; Dr. Cynthia Weickert has an appointment at the University of New South Wales, Australia. The team also included 2003 BBRF Young Investigator **Roshel Lenroot, M.D.**, and 2008 BBRF Young Investigator **Julia Lappin, MBChBN, MRCPsych**.

Drs. Weickert and colleagues point out that the question has been tested before: will anti-inflammatory medicines help reduce symptoms in schizophrenia? Results, they note, have been inconsistent and inconclusive. There are many possible reasons, but one important reason one is that schizophrenia, like other psychiatric illnesses, is highly “heterogeneous”; symptoms differ in kind and severity across the full spectrum of patients. It is almost certain that causal mechanisms differ, as well. One implication is that what helps one patient or group of patients might not help another.

In their trial, Drs. Weickert and team exclusively recruited chronically ill schizophrenia patients with elevated markers of inflammation in their peripheral blood. It’s possible that prior tests of anti-inflammatories didn’t register significant positive results because many of the participants did not have elevated inflammation levels to begin with. The team wanted to test a specific anti-inflammatory medicine on patients they knew to have elevated inflammation levels.

Twenty-seven such patients were recruited for the study, which was



Thomas W. Weickert, Ph.D.
2016 BBRF Independent Investigator



Cynthia S. Weickert, Ph.D.
2004 BBRF Independent Investigator
2001, 1999 Young Investigator

SUNY Upstate Medical University Neuroscience Research Australia, University of New South Wales

conducted in Australia. To be included, a participant had to have at least two elevated markers of peripheral (bodily) inflammation, out of three such general markers tested. The markers indicated levels of: two cytokines (small proteins that help regulate immune system cells in the body), specifically, Interleukin 1-Beta (IL-1 β) or IL-6; high-sensitivity C-reactive protein (hsCRP), a protein whose level correlates with immune activation; and a marker called NLR that measures the ratio in the blood of neutrophils to lymphocytes (two types of white blood cells).

The cohort was composed of 12 females and 15 males with a diagnosis of schizophrenia (18) or schizoaffective disorder (9). The average age was late-thirties; the average duration of illness was about 12 years; the typical participant had been hospitalized three times over the course of their illness, and was moderately overweight (BMI ~ 32).

Fourteen participants were assigned to receive a single injection under the skin of an approved medicine called canakinumab, a monoclonal antibody that blocks the activity of IL-1 β . Thirteen participants received a placebo injection. All 27 continued to take the antipsychotic medicines they had been taking before the start of the trial.

Why the focus on blocking the activity of IL-1 β ? Levels of IL-1 β are known to be elevated in a “substantial subgroup” of chronically ill schizophrenia patients, as evidenced in blood, cerebrospinal fluid, and brain tissue. Past studies have shown that elevated peripheral IL-1 β levels correlate with impairment in attention, working memory, language,

and episodic memory in schizophrenia patients. The Weickerts have previously found, moreover, elevated IL-1 β expression in white blood cells in 40% of patients with chronic schizophrenia, as well as higher levels of IL-1 β expression in cells of the prefrontal cortex in regions where new neurons are generated, and in the midbrain, in about an equal fraction of patients. The C-reactive protein marker was chosen because its level is elevated in 60% of patients admitted to hospital for a psychotic episode and 40% of chronically ill schizophrenia patients. Elevation in CRP has been linked with thinning of the cortex and problems with attention. Past clinical trials have demonstrated that injection of canakinumab quickly lowers peripheral CRP levels in peripheral blood.

Results of the trial, based on comparisons between the two groups of inflammatory marker levels in peripheral blood and symptom severity at baseline and at 4 and 8 weeks post-injection, showed that a single injection of the drug (150mg) “was effective in reducing a peripheral marker of inflammation [CRP].” Levels of CRP declined continuously for the first 4 weeks post-injection and were significantly reduced at all times through 8 weeks relative to baseline levels.

Markers of inflammation were lower; but did this correlate with a reduction in the severity of symptoms? Negative symptoms—various cognitive impairments experienced by all schizophrenia patients—were not impacted by canakinumab or by the placebo. But the drug did have an impact described as “statistically

significant” on schizophrenia’s positive symptoms—hallucinations, delusions, and odd or intrusive thoughts.

Those in the canakinumab group “had a significant reduction in positive symptom severity score 8 weeks following the injection,” the team reported. While “the magnitude of the reduction would not generally be considered clinically robust,” they added, “it is important to note that most novel treatments do not reduce” these symptoms, particularly if the patients, as in this trial, continue to take their regular antipsychotic medicine throughout the trial. The team found that the in the canakinumab group, reduction in CRP levels at week 4 predicted the degree to which a patient’s positive symptoms would be reduced in severity at week 8. Reductions in CRP levels are also considered positive for general health, as elevated levels are strongly linked with heart disease.

Future trials to confirm or extend the team’s results will need to include many more patients with elevated inflammation markers, including those at earlier stages of schizophrenia and psychotic disorders. To be truly meaningful, any benefit in reducing symptoms would need to be sustained for much longer than the 8 weeks tested in this trial. To that end, the team hopes to test canakinumab in inflammation-affected patients with higher dosages of the medicine, and with longer treatment administration, including “top-up” or additional injections over time. Also, they noted, “treating people closer to the onset of the illness when inflammation has not been present for a long time may have the potential to show larger effects.”

Precisely Targeting and Predicting the Impact of TMS Brain Stimulation for Depression with Combined TMS/fMRI

Next-Generation Therapies: **Depression**

Story Highlights:

Researchers identified a predictor of rTMS brain stimulation therapy in depression: the more a preliminary TMS pulse showed activity in a deep-brain structure was reduced, the greater the response to 3 days of rTMS therapy. Potential implications: predicting patient response before therapy begins; and precisely targeting treatments to relieve specific psychiatric symptoms.

Journal: *Nature Mental Health*
November 27, 2023

As non-invasive brain stimulation—variations of TMS (transcranial magnetic stimulation)—continues to evolve and is administered to a growing number of patients with depression (and other illnesses, including OCD), researchers seek to understand how and why it is able to help reduce symptoms in patients who respond positively.

A newly published study appearing in *Nature Mental Health*, led by 2016 BBRF Young Investigator **Desmond J. Oathes Ph.D.**, of the University of Pennsylvania, adds to what is known about how TMS works, and suggests it may be possible to target specific symptoms of psychiatric illnesses using individual brain targets to maximize outcomes that might be predicted before treatment begins. **Yvette I. Sheline, M.D.**, a 2005 and 2002 BBRF Independent Investigator and 1998 Young Investigator, was a co-author of the paper.

Approximately one-third to one-half of patients with depression, including those who have not responded to other depression therapies, are found to respond to standard rTMS, the form of the therapy in which magnetic pulses are applied repetitively to an area just above the scalp. BBRF Scientific Council member **Mark S. George, M.D.**, used BBRF Young Investigator grants in the 1990s to explore and develop TMS technology, a process that contributed in 2008 to FDA approval of TMS for major

depression. In recent years, variations on rTMS, notably iTBS (intermittent theta-burst stimulation) and SAINT, which deliver more stimulation (in the case of SAINT, over a period of just 5 days), rather than lesser levels of stimulation over 4-6 weeks in standard rTMS, have been used in the clinic and have led to patient remission rates of up to 80% in treatment-resistant individuals.

In developing the SAINT protocol, two-time BBRF Young Investigator Nolan R. Williams, M.D., and colleagues, had the idea of improving the targeting of the spot above the scalp where magnetic pulses are focused. Imaging scans were used to arrive at an optimal location for each patient. Implicit in this calculation was the observation that the area directly beneath the scalp, in the brain's dorsolateral prefrontal cortex (DLPFC), was part of a neural network that connected with a structure deeper in the brain called the subgenual anterior cingulate cortex (sgACC). The latter could not be reached directly by rTMS magnetic pulses, but it could be modulated because it was connected to the DLPFC through functional connections in the brain. SAINT focuses the treatment on the precise spot in the DLPFC most likely to affect neural activity in the sgACC.



Desmond J. Oathes, Ph.D.

University of Pennsylvania

2016 BBRF Young Investigator

This and other research showed that the sgACC is “hyperconnected” to other brain regions in people who have depression. Stimulation of the DLPFC appeared to have the effect of reducing that hyperconnectivity.

The new research by Dr. Oathes and colleagues carefully probes the relationship between non-invasive brain stimulation, activity in the sgACC, and, in turn, its relation to the degree to which a given patient’s depression symptoms are reduced.

Thirty-six unmedicated patients with clinical depression were analyzed in the study. Each was assessed and given brain imaging scans prior to receiving a full course of iTBS therapy (a form of rTMS therapy) over 3 days. They were assessed again after the intervention was administered. The assessments involved interpretation of brain imaging data as well as data about symptom severity.

The rTMS therapy in each patient was individually targeted, based on pre-therapy resting fMRI and structural MRI data. These data were used to target an unusual combined TMS and fMRI session consisting of a short burst of pulses given while the brain was being scanned in real time by fMRI. The purpose of this brief, preliminary administration of TMS was to assess what researchers call the “evoked response” in the sgACC, the deep-brain region whose activity full treatment is designed to modulate.

Based on this preliminary scanning and testing, Dr. Oathes’ team discovered that the degree to which the preliminary, brief TMS exposure generated a “negative BOLD signal” in the sgACC, the greater was the patient’s response to a full course of rTMS. A negative BOLD signal means that neural activity in the region being examined is reduced. In this case it means: the more a preliminary TMS pulse showed that activity in the sgACC was reduced, the greater the response would prove to be when the 3-day rTMS intervention was subsequently given.

After the participants received therapy, the same assessment tools were used to again test the “evoked response” of the sgACC to a brief TMS pulse. Those participants whose “evoked response” was most reduced, compared with their response prior to therapy, were those whose depression symptoms had responded the most to the therapy.

When non-invasive brain stimulation is applied, not only is the targeted area affected, but also, inevitably, other, “off-target” areas. This is a function of how interconnected brain circuits tend to be. One interesting finding in the new study was that the impact of rTMS on the sgACC, specifically, appeared to be responsible for reductions in depression symptoms. Even though the same treatment course also resulted in decreases in anxiety symptoms experienced by most patients, the team found that these did not appear to be related to the impact of the therapy on the sgACC, but rather on “off-target” areas.

The study’s results suggest the possibility that the more precisely the relationship is known between the targets of brain stimulation and their specific impacts on symptoms, the more possible it may become to target specific symptoms of psychiatric disorders. Another implication, if the current results are replicated in larger clinical trials, is that it may be feasible to predict the likely impact of stimulation of specific targets in the brain without having to put the patient through a trial-and-error process of targeting one area and then waiting to see the impact after a full course of therapy.

BBRF's Historic Contribution to Research is Featured in Major Science Journal

The article reproduced in the following pages details BBRF's contribution to the field of neuropsychiatric research.

It was first published in the journal *Neuropsychopharmacology* (online on May 8, 2023 and in print in January 2024).

The article is co-authored by Jeffrey Borenstein, M.D., President and CEO of BBRF, Geoffrey Simon, BBRF Board Chairman, and Peter J. Tarr, Ph.D., Editorial Director, Science and Chief Science Writer for BBRF.



PERSPECTIVE



The Brain & Behavior Research Foundation's philanthropic support for neuropsychiatric research

Jeffrey Borenstein¹, Geoffrey A. Simon¹ and Peter J. Tarr¹✉

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In the 36 years since its founding, the Brain and Behavior Research Foundation (BBRF) has become one of the world's largest non-government funders of grants for neuropsychiatric research. A number of lessons can be drawn from the BBRF experience. One is that scientific competence in the organization, and full control over selection of grantees, has always resided in a Scientific Council composed of leaders in the field. Fund-raising has been conducted separately, and all public dollars contributed have been used to fund grants. The Council has sought to support the best research, no matter who is doing it or where it is being done. Over 80% of 6300 grants awarded have jump-started the careers of young investigators judged to demonstrate unusual promise. These early-career grants have been the equivalent of seed funding, enabling the best and brightest entrants to the field to perform research that, if successful, can provide a basis for much larger, career-sustaining grants. Much of the funded research has been basic research, although many contributions leading to clinical advances have also resulted from BBRF grants. BBRF has learned that it pays to have a diversified research portfolio, with thousands of grantees attacking the problem of mental illness from many different angles. The Foundation's experience also demonstrates the power of patient-inspired philanthropic support. Donors repeatedly express satisfaction that some aspect of mental illness that they care deeply about is being addressed, and find comfort and support from the sense of joining with others in the mission.

Neuropsychopharmacology; <https://doi.org/10.1038/s41386-023-01595-3>

In the 36 years since its founding, the Brain and Behavior Research Foundation (BBRF) has become one of the world's largest non-government funders of grants for neuropsychiatric research. Through 2022, the organization awarded over 6300 research grant awards to over 5300 investigators, most of them in the early stages of their career. These grants, totaling \$440 million, have helped to launch many productive careers and in the words of Nobel laureate Dr. Eric Kandel, they have had an important role "in helping to structure the field" of neuropsychiatry [1].

The founders of BBRF believed the field of psychiatric research was underfunded relative to other medical fields [2], and knew that 85% of funds then available were provided by government [3]. There was a desire to develop new funding sources, but there were some well-known obstacles to overcome. Perhaps the most serious were cultural.

One of BBRF's founders, Dr. Herbert Pardes, observed in the 1980s that many people with mental illnesses were not in a position to advocate, either due to functional incapacity or for fear of being stigmatized. At the same time, he noted, "most people who don't suffer from a psychiatric illness figure they will *never* suffer from one—an attitude very few reasonable people have about, for instance, cancer or heart disease." [4] Stigma, indeed, was at the root of the funding problem, also seriously limiting the willingness of private foundations and charities to get involved. Cancer had long been stigmatized, but by the 1980s, citizen-advocacy groups like the American Cancer Society (ACS) had worked wonders, helping to lower stigma by raising public

awareness. There was no comparable organization for mental illness. The Mental Health Association and several other reputable U.S. organizations were doing good work, but none placed their primary focus on raising funds for research.

BBRF, which was initially called the National Alliance for Research on Schizophrenia and Depression (NARSAD), emerged in 1987. Eight years earlier, Dr. Pardes, then director of the National Institute of Mental Health, had encouraged a group of parents whose children were diagnosed with schizophrenia to form an advocacy organization, which they named the National Alliance for the Mentally Ill (NAMI). Some years later, they asked Dr. Pardes: "Shouldn't an organization be started to complement NAMI's work, that would support research specifically?" He arranged to meet with leaders of NAMI and the Kentucky-based Schizophrenia Foundation. BBRF was born of this meeting [5]. The Foundation is thus rooted in the desire for new and more effective treatments on the part of individuals whose loved ones are experiencing mental illness—an important manifestation in our field of patient advocacy.

Dr. Pardes and colleagues formed a Scientific Council that would serve as the Foundation's core. Perhaps the most important lesson to be drawn from the BBRF experience is that scientific competence in the organization has always resided in the Scientific Council. There was from the outset, in the words of Dr. Pardes, "a wall as inviolate as that between church and state" separating the Foundation's grant-making and fund-raising. The latter was performed by a professional staff supported from earliest days

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by separate external grants. Every dollar donated for research by the public to BBRF has been devoted to research grants.

As BBRF's grant-making capacity rapidly grew, the decision to assign full control of selecting grantees to special committees of the Scientific Council, composed then as now of volunteers who are leaders in their respective fields and subfields, was definitive. The professional organization responsible for fund-raising did its job well, serving as the constant connection between the Foundation and patients and their families who had brought the organization into existence. The Scientific Council, for its part, has always independently assessed annual grant applications. It has sought from the outset to fund the best research—no matter who is doing it and no matter where it is being done. BBRF's Scientific Council now has 187 members, among whom are 50 members of the National Academy of Medicine, 42 chairs of academic psychiatry and neuroscience departments, 16 NIH chiefs & directors, 7 members of the National Academy of Sciences, and 3 recipients of the National Medal of Science.

A second key lesson we've learned from our 36 years of history concerns the multiplier effect of effective grant-making. By far the largest number of BBRF's grants awarded to date—over 80%—have had the purpose of jump-starting the careers of young investigators judged by the Scientific Council to demonstrate unusual promise [6]. These early-career grants have been the equivalent of seed funding, enabling the best and brightest entrants to the field to perform research that, if successful, can provide a basis for much larger, career-sustaining grants from federal sources including the NIH and NIMH. The Foundation's grant-making procedures have resulted in advancement of the careers of individuals of diverse backgrounds. Each grant applicant has the opportunity to share with Scientific Council reviewers their personal experiences, including any adverse conditions that they may have had to overcome to launch their careers.

Over the 36 years of our institutional history, the Foundation's grantees have made vital contributions to basic science and clinical research, and have been instrumental in enabling the development of new treatments, in this way specifically fulfilling the intentions of the families of patients who launched the Foundation. Just a few of their achievements include contributions to the development of the first rapid-acting antidepressants; development of transcranial magnetic stimulation (TMS) to treat patients with refractory depression; development of optogenetics technology to study neuronal and circuit function; application of induced pluripotent stem cell technology to study the neurodevelopmental origins of psychiatric illnesses; seminal studies of the neurobiology of addiction, and of the impact of stress upon brain structures implicated in anxiety and depression; pathbreaking studies of genetic risk in schizophrenia and bipolar disorder, of risk factors that are potentially predictive of suicidal behavior, and of depression risk as observed over multiple generations; and progress toward novel preventive measures, particularly maternal choline supplementation during pregnancy.

The impact of our early-career grants may be the most important mark of the Foundation's success. "Having a NARSAD" would become a mark of a young researcher's legitimacy, their work's potential having been recognized by authorities in the field. This early-career impact is especially gratifying given the widely noted difficulty in recent years of securing funding to establish new careers in the field.

NIMH Director and BBRF Scientific Council member Dr. Joshua Gordon received BBRF Young Investigator grants in 2001 and 2003. "It was a really challenging time," he has recalled. "My whole scientific career depended on a \$60,000 piece of equipment and then all of sudden I was cut off. It was only because I received the Young Investigator award at this same time that I was able to do the project I needed to do. That got me my next award and then a K Award from the NIMH." [7].

Myrna Weissman, Ph.D., is one of many researchers in the field whose cumulative lifetime achievement was supported at each step by Foundation grants. In 1991, when she received her first BBRF grant, she was already committed to the idea of studying major depressive disorder longitudinally. By 2005, when she received her third grant, Dr. Weissman could build on 20 years of data derived from a carefully recruited cohort of individuals at high and low risk for the disorder. These investigations—still ongoing—would help to reveal, among other things, gender differences in rates of depression, with women at disproportionate risk; the impact of parental depression upon succeeding generations; and the great opportunity of reducing offspring depression risk by treating depressed parents. "At the start of my career," Dr. Weissman has noted, "I received funding from BBRF to get pilot data. You have to have a funding agency that trusts you and is flexible. The kind of flexibility and trust BBRF provided was extraordinary. To do one study, we had to get DNA samples from our families, and that takes time. We were awarded the grant in 2005, but they let us extend the study. The Foundation has stepped to the plate whenever we've wanted to do something we thought was innovative and exciting and didn't think anyone else would fund." [8].

A testimonial from another member of our Scientific Council calls attention to the question of risk in early-career grant-making. Dr. Karl Deisseroth of Stanford University was a Lasker Basic Medical Research award co-winner in 2021 for his part in the development of optogenetics. He stresses that "my first steps that led to these advances were supported by BBRF [Young Investigator grants, in 2003 and 2005] and were not part of a traditional disease-related research program." Dr. Deisseroth stresses the immense importance of communicating to the public that supports research—whether via tax dollars or philanthropic giving—"that any specific goal of a research portfolio is best served with a major *basic research* component, which BBRF has pioneered and exemplified, where *direct links between research and goal are not known, or even knowable*" when a grant is awarded [9].

Another pathbreaking researcher, Dr. Mark George of the Medical University of South Carolina, received two early-career BBRF Young Investigator grants at a time when the work for which he would become known was out of favor. "In 2000," Dr. George has noted, "depression was thought of as a chemical imbalance. It is now discussed as a brain disorder involving disrupted circuits in the brain. My own work proposing noninvasive brain stimulation—transcranial magnetic stimulation (TMS)—as a treatment arose out of brain imaging studies of depression and sadness, and began to define these dysfunctional circuits. Because I was operating out of a different paradigm, my ideas were not well received. Luckily, early BBRF grants helped me continue my science." This leads Dr. George to propose what we take to be another of the key lessons from our history: "Organizations like BBRF are successful if they can support innovative research that disrupts current modes of thinking. This research then fosters innovations that lead to new understanding and hopefully new treatments." [10].

No discussion of our Foundation would be complete without acknowledging the immense influence of the family that did more to insure our financial and institutional success than any other. Constance and Stephen Lieber became involved in the Foundation's first year, having asked Dr. Pades at a scientific symposium "whether there is anything we can do to help." Their daughter had been diagnosed with schizophrenia, and they sought ways to accelerate research just at the time when the fledgling Foundation sought philanthropic donors committed to the cause. Connie Lieber would go on to lead the Foundation as its president for 18 years; Steve Lieber would serve as board chairman for 12 years, holding the position until his passing in 2020.

The Liebers' passion to support research was accompanied by an interest in finding ways to recognize individual researchers. In the Foundation's first year, they created the Lieber Prize for outstanding achievement in schizophrenia research. Steve Lieber once explained the idea of prizes for psychiatry as a kind of "Nobel equivalent, in a field that is underrecognized." The Maltz Prize, named for Tamar and Milton Maltz, extraordinary leaders and supporters of BBRF, enables the annual Lieber Prize winner to reward a promising schizophrenia researcher. BBRF also awards prizes for outstanding achievement in mood disorders (Colvin Prize); childhood and adolescent research (Ruane Prize); and cognitive neuroscience (Goldman-Rakic Prize). Other annual prizes awarded by BBRF include the Klerman and Freedman Prizes, which recognize outstanding young investigators for exceptional clinical and basic research in mental illness; and the Pardes Humanitarian Prize in Mental Health, established to recognize a physician, scientist, public citizen, or organization making a major humanitarian contribution to the field.

After Steve Lieber's passing in 2020, Helen Mayberg, M.D., a three-time BBRF grantee, prize winner and Scientific Council member, reflected on precisely what it was about Connie and Steve Lieber that made such an impact. Early-career grants from the Foundation had helped Dr. Mayberg conduct research leading to an alternative, distinctly neurological view of depression and mood disorders, stressing circuits and networks in the brain that interact with one another in ways that change from moment to moment and which can fall out of synch. One grant supported her pioneering studies of deep-brain stimulation in treatment-resistant depression.

"Thinking broadly about the impact of the Liebers," Dr. Mayberg wrote, "I think it was the wisdom of their effort to enable a community—the community of researchers—to solve the very difficult problem of mental illness. It's the pragmatic realization that there isn't one solution. There's the appreciation that it pays to have a diversified portfolio. Steve and Connie created an environment that has enabled thousands of researchers to attack the problem from many different angles at once." [11].

A final, poignant lesson from BBRF's history concerns the power of what our founders called "patient-inspired philanthropic support." We have shown that thousands of people—over 70,000 people have made donations to BBRF—are pleased to give, in many cases repeatedly and in increasing amounts. A commitment to research, they understand, is about paving the path to a better future. Our donors repeatedly express satisfaction to us that some aspect of mental illness that they care very much about is being addressed, and we find that they gain comfort and support from the sense of joining with others in that mission. That insight, from Connie Lieber and Herb Pardes, was published in the pages of the *American Journal of Psychiatry* 17 years ago [12]. It is every bit as true and as important today as it was then.

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J. John Mann, M.D.

John S. March, M.D., M.P.H.

Stephen Maren, Ph.D.

Keri Martinowich, Ph.D.

Daniel H. Mathalon, Ph.D., M.D.

Helen S. Mayberg, M.D.

Carla A. Mazefsky, Ph.D.

Colleen A. McClung, Ph.D.

Ronald McKay, Ph.D.

James H. Meador-Woodruff, M.D.

Herbert Y. Meltzer, M.D.

Kathleen R. Merikangas, Ph.D.

Richard J. Miller, Ph.D.

Guo-Li Ming, M.D., Ph.D.

Karoly Mirnics, M.D., Ph.D.

Bitu Moghaddam, Ph.D.

Lisa M. Monteggia, Ph.D.

David A. Morilak, Ph.D.

Charles B. Nemeroff, M.D., Ph.D.

Eric J. Nestler, M.D., Ph.D.

Andrew A. Nierenberg, M.D.

Patricio O'Donnell, M.D., Ph.D.

Dost Ongur, M.D., Ph.D.

Maria A. Oquendo, M.D., Ph.D.

Martin P. Paulus, M.D.

Godfrey D. Pearlson, M.D., Ph.D.

Peter Penzes, Ph.D.

Mary L. Phillips, M.D., M.D. (CANTAB)

Marina R. Picciotto, Ph.D.

Daniel S. Pine, M.D.

Robert M. Post, M.D.

James B. Potash, M.D., M.P.H.

Susan Powell, Ph.D.

Pasko Rakic, M.D., Ph.D.

Judith L. Rapoport, M.D.

Perry F. Renshaw, M.D., Ph.D., M.B.A.

Kerry J. Ressler, M.D., Ph.D.

Victoria B. Risbrough, Ph.D.

Carolyn Rodriguez, M.D., Ph.D.

Bryan L. Roth, M.D., Ph.D.

Laura M. Rowland, Ph.D.

Scott J. Russo, Ph.D.

Gerard Sanacora, M.D., Ph.D.

Akira Sawa, M.D., Ph.D.

Alan F. Schatzberg, M.D.

Nina R. Schooler, Ph.D.

Robert Schwarcz, Ph.D.

Yvette I. Sheline, M.D.

David A. Silbersweig, M.D.

Vikaas S. Sohal, M.D., Ph.D.

Matthew W. State, M.D., Ph.D.

Murray B. Stein, M.D., M.P.H., F.R.C.F.C.

Joanna C. Steinglass, M.D.

Stephen M. Strakowski, M.D.

Carol A. Tamminga, M.D.

Laurence H. Tecott, M.D., Ph.D.

Flora M. Vaccarino, M.D., Ph.D.

Rita J. Valentino, Ph.D.

Jeremy M. Veenstra-VanderWeele, M.D.

Susan M. Voglmaier, M.D., Ph.D.

Aristotle N. Voineskos, M.D., Ph.D.

Nora D. Volkow, M.D.

Karen Dineen Wagner, M.D., Ph.D.

Daniel R. Weinberger, M.D.

Myrna M. Weissman, Ph.D.

Jared W. Young, Ph.D.

L. Trevor Young, M.D., Ph.D.,

F.R.C.P.C., F.C.A.H.S.

Carlos A. Zarate, Jr., M.D.

Jon-Kar Zubieta, M.D., Ph.D.

2023 New BBRF Scientific Council Members

The high quality of the research we fund is made possible by the BBRF Scientific Council.

This group of 192 prominent mental health researchers reviews each grant application and selects the most promising ideas with the greatest potential to lead to breakthroughs.

The Scientific Council guides the Foundation to fund creative and impactful basic, translational, and clinical research relevant to the whole spectrum of mental health.



Olusola Ajilore, M.D., Ph.D.
University of Illinois-Chicago



Julie A. Blendy, Ph.D.
*Perelman School of Medicine,
University of Pennsylvania*
1998 Young Investigator
Grantee



Ege T. Kavalali, Ph.D.
*Vanderbilt University's School
of Medicine*
2012 Distinguished
Investigator Grantee



Keri Martinowich, Ph.D.
*Lieber Institute for Brain
Development, Johns Hopkins
Medical Campus*
Johns Hopkins School of
Medicine
2013, 2008 Young Investigator
Grantee



Carmen Andreescu, M.D.
University of Pittsburgh
2009 Young Investigator
Grantee



Andrea B. Goldschmidt, Ph.D.
University of Pittsburgh



Gregory A. Light, Ph.D.
University of California, San Diego
2014 BBRF Baer Prizewinner
2013 Independent
Investigator Grantee
2006, 2003 Young
Investigator Grantee



Martin P. Paulus, M.D.
*Laureate Institute for Brain
Research*
2000 Young Investigator
Grantee



Tracy L. Bale, Ph.D.
University of Colorado



Laura M. Huckins, Ph.D.
Yale School of Medicine
2017 Young Investigator
Grantee



Beatriz Luna, Ph.D.
University of Pittsburgh
1997 Young Investigator
Grantee



Joanna C. Steinglass, M.D.
*New York State Psychiatric
Institute / Columbia University
Irving Medical Center*
2007, 2004 Young
Investigator Grantee



Debra A. Bangasser, Ph.D.
Georgia State University



Thomas L. Kash, Ph.D.
*University of North Carolina at
Chapel Hill School of Medicine*
2014 Independent
Investigator Grantee
2010 Young Investigator
Grantee

BBRF Grants Drive Scientific Breakthroughs Leading Toward Improved Treatments, Cures, and Methods of Prevention

BY THE NUMBERS SINCE 1987

As of November 2023

AWARDED TO SCIENTISTS



\$450+ MILLION

GRANTS

6,500



GRANTEES

5,400



UNIVERSITIES & MEDICAL CENTERS

599

COUNTRIES, INCLUDING THE U.S.

41

The path to being awarded a BBRF Grant starts with an application. Grant applicants describe why they think their project could help lead to new insights and advances in the field of mental illness. Applicants represent the best and the brightest talent from world-class institutions. BBRF's Scientific Council, led by Dr. Herbert Pardes, volunteers their time to review and evaluate applications. BBRF Grants support a broad range of the best ideas in brain research. Funding is focused on four priority areas to better understand and treat mental illness, aiming toward prevention and cures.

Research Categories

Basic Research

To understand what happens in the brain to cause mental illness

Diagnostic Tools/Early Intervention

To recognize early signs of mental illness and treat as early as possible

New Technologies

To advance or create new ways of studying and understanding the brain

Next-Generation Therapies

To reduce symptoms of mental illness and ultimately cure and prevent brain and behavior disorders

BBRF Young Investigator Grants in 2023



BBRF Young Investigator Grants give early-career scientists the initial funding they need to begin to test their ideas and solidify their academic research careers. YI Grants provide scientists with \$35,000/year for two years totaling \$70,000. This seed money enables them to generate the preliminary data, or “proof of concept” that they need to compete for larger grants from traditional funding sources like the National Institutes of Health.

All BBRF grant recipients are chosen by our world-renowned Scientific Council which is comprised of 192 scientists who rigorously evaluate every grant application, identifying the most promising, innovative science, with the greatest potential for significant breakthroughs.

Co-Chairs of the Young Investigator Grant Selection Committee



Judy M. Ford, Ph.D.

Professor, Department of
Psychiatry
*University of California,
San Francisco*

BBRF Scientific Council Member
2003 BBRF Independent
Investigator



Suzanne N. Haber, Ph.D.

Professor, Department of
Pharmacology and Physiology
*University of Rochester School of
Medicine and Dentistry*

BBRF Scientific Council Member
2011 BBRF Distinguished
Investigator

“The YI program is the only program of its kind. We fund young investigators to address questions critical to mental health. BBRF helps launch the careers of 150 scientists each year and a YI grant is often the bridge needed to launch an independent lab. Members of the Scientific Council focus on funding grants that will take the field to the next level of treatment, prevention, and hopefully cures for mental illness.”

— Dr. Judy Ford

2023 BBRF Young Investigator Grants by Illness

Some grantees are listed under multiple categories as their grant projects are relevant to more than one illness.

ADDICTION / SUBSTANCE-USE DISORDERS

Lillian J. Brady, Ph.D.

 Basic Research

Sarah J. Brislin, Ph.D.

 Basic Research

Julia M. Cox, Ph.D.

 Basic Research

Prashant C. Donthamsetti, Ph.D.

 Basic Research

Gabor Egervari, M.D., Ph.D.

 Basic Research

Yasmin Escobedo Lozoya, Ph.D.

 Basic Research

Giulia R. Fois, Ph.D.

 Basic Research

Megan E. Fox, Ph.D.

 Basic Research

Constanza Garcia Keller, Ph.D.

 Basic Research

Ming-Fen Ho, Ph.D.

 Basic Research

 Next-Generation Therapies

Elizabeth N. Holly, Ph.D.

 Basic Research

Barbara Juarez, Ph.D.

 Basic Research

Robert Kagabo, Ph.D.

 Next-Generation Therapies

Hao Li, M.D., Ph.D.

 Basic Research

Debora Masini, Ph.D.

 Basic Research

 Next-Generation Therapies

Brittany D. Needham, Ph.D.

 Next-Generation Therapies

Ashley C. Parr, Ph.D.

 Basic Research

William E. Pelham III, Ph.D.

 Basic Research

 Diagnostic Tools/Early Intervention

David Saunders, M.D., Ph.D.

 Next-Generation Therapies

Damiano Terenzi, Ph.D.

 Next-Generation Therapies

Christopher W. Tschumi, Ph.D.

 Basic Research

Andrew M. Wikenheiser, Ph.D.

 Basic Research

Andrea S. Young, Ph.D.

 Basic Research

Rui Zhang, Ph.D.

 Next-Generation Therapies

Qingyu Zhao, Ph.D.

 Diagnostic Tools/Early Intervention

ATTENTION-DEFICIT HYPERACTIVITY DISORDER (ADHD)

Davide Aprile, Ph.D.

 Basic Research

Gerard J. Broussard, Ph.D.

 Basic Research

Giulia R. Fois, Ph.D.

 Basic Research

J. Wren Kim, Ph.D.

 Basic Research

Nataliia Kozhemiako, Ph.D.

 Basic Research

Yang Liu, Ph.D.

 Basic Research

 Diagnostic Tools/Early Intervention

Debora Masini, Ph.D.

 Basic Research

 Next-Generation Therapies

Marianne Oldehinkel, Ph.D.

 Next-Generation Therapies

 Basic Research

Alessandro Piccin, Ph.D.

 Basic Research

Eszter Szekely, Ph.D.

 Basic Research

ANXIETY DISORDERS

Vineet Augustine, Ph.D.

 Basic Research

Erica B. Baller, M.D.

 Basic Research

Laurie Bayet, Ph.D.

 Basic Research

Andrea Boscutti, M.D.

 Next-Generation Therapies

 New Technologies

Jessica I. Buthmann, Ph.D.

 Diagnostic Tools/Early Intervention

Cristiana Cruceanu, Ph.D.

 Basic Research

Nicholas Alonzo Frost, M.D., Ph.D.

 Basic Research

Janos Fuzik, Ph.D.

 Basic Research

Marta Garcia-Forn, Ph.D.

 Basic Research

Erin E. Hisey, Ph.D.

 Basic Research

Elizabeth N. Holly, Ph.D.

 Basic Research

Alexandra S. Klein, Ph.D.

 Basic Research

Clare A. McCormack, Ph.D.

 *Basic Research*

Nicky J. Mehtani, M.D.

 *Next-Generation Therapies*

Heidi Catherine Meyer, Ph.D.

 *Basic Research*

Brittany D. Needham, Ph.D.

 *Next-Generation Therapies*

Takuya Osakada, Ph.D.

 *Basic Research*

Bruno Oriol Porras-Garcia, Ph.D.

 *Next-Generation Therapies*

Christopher T. Sege, Ph.D.

 *Next-Generation Therapies*

 *New Technologies*

Tarjinder Singh, Ph.D.

 *Basic Research*

Sydney Trask, Ph.D.

 *Next-Generation Therapies*

Brad Verhulst, Ph.D.

 *Basic Research*

Jessica J. Walsh, Ph.D.

 *Next-Generation Therapies*

Tao Xie, Ph.D.

 *Basic Research*

Wen Xin, Ph.D.

 *Basic Research*

AUTISM SPECTRUM DISORDER (ASD)

Sarah D. Ackerman, Ph.D.

 *Basic Research*

Davide Aprile, Ph.D.

 *Basic Research*

Laurie Bayet, Ph.D.

 *Basic Research*

Michelle C.D. Bridi, Ph.D.

 *Basic Research*

Gerard J. Broussard, Ph.D.

 *Basic Research*

Seungwon (Sebastian) Choi, Ph.D.

 *Basic Research*

Marta Garcia-Forn, Ph.D.

 *Basic Research*

Taeyoung Hwang, Ph.D.

 *Basic Research*

 *Next-Generation Therapies*

Valentina Ignatova, Ph.D.

 *Basic Research*

J. Wren Kim, Ph.D.

 *Basic Research*

Nataliia Kozhemiako, Ph.D.

 *Basic Research*

Marianne Oldehinkel, Ph.D.

 *Next-Generation Therapies*

 *Basic Research*

Christopher W. Tschumi, Ph.D.

 *Basic Research*

Jessica J. Walsh, Ph.D.

 *Next-Generation Therapies*

Xiaoting Wu, Ph.D.

 *Basic Research*

BIOLOGY OF THE BRAIN

*These projects focus on
how the brain works*

CHILDHOOD COGNITION & MOTIVATION

Youngsun Theresa Cho, M.D., Ph.D.

 *Diagnostic Tools/Early Intervention*

EARLY-LIFE IMMUNE CHALLENGES

Emilia Favuzzi, Ph.D.

 *Basic Research*

 *Next-Generation Therapies*

IMPACT OF PSYCHEDELICS

Srividya Ganapathy, Ph.D.

 *Next-Generation Therapies*

IMPULSIVE AGGRESSION

Debora Masini, Ph.D.

 *Basic Research*

 *Next-Generation Therapies*

IMPACT OF GENETIC VARIANTS

Mariana Moyses-Oliveira, Ph.D.

 *Basic Research*

EMOTION REGULATION

Erik C. Nook, Ph.D.

 *Basic Research*

EARLY RESPONSES TO

ENVIRONMENT

Albert D. Piersson, Ph.D.

 *Basic Research*

SLEEP-RELATED CIRCUITRY

Mubarak H. Syed, Ph.D.

 *Basic Research*

FUNCTIONAL CONSEQUENCES OF GENETIC VARIATIONS

Xuran Wang, Ph.D.

 *Basic Research*

BIPOLAR DISORDER

Chinnakkaruppan Adaikkan, Ph.D.

 *Basic Research*

 *Next-Generation Therapies*

Debora Masini, Ph.D.

 *Basic Research*

 *Next-Generation Therapies*

Ana P. Silva, Ph.D.

 *Basic Research*

Emma K. Stapp, Ph.D.

 *Next-Generation Therapies*

BORDERLINE PERSONALITY DISORDER

Erin A. Kaufman, Ph.D.

 *Next-Generation Therapies*

Fabian Streit, Ph.D.

 *Basic Research*

DEPRESSION

Chinnakkaruppan Adaikkan, Ph.D.

 *Basic Research*

 *Next-Generation Therapies*

Andrea Boscutti, M.D.

 *Next-Generation Therapies*

 *New Technologies*

Joshua C. Brown, M.D., Ph.D.

 *Next-Generation Therapies*

Jessica I. Buthmann, Ph.D.

 *Diagnostic Tools/Early Intervention*

Jennifer C. Chan, Ph.D.

 *Basic Research*

Joshua L. Cohen, M.D., Ph.D.

 *Next-Generation Therapies*

Elise C. Cope, Ph.D.

 *Next-Generation Therapies*

 *Basic Research*

Cristiana Cruceanu, Ph.D.

 *Basic Research*

Jinye Dai, Ph.D.

 *Basic Research*

Neir Eshel, M.D., Ph.D.

 *Basic Research*

Tanner C. Francis, Ph.D.

 *Basic Research*

Polymnia Georgiou, Ph.D.

 *Basic Research*

Livea D. Godoy, Ph.D.,

 *Next-Generation Therapies*

Simon B. Goldberg, Ph.D.,

 *Next-Generation Therapies*

 *Basic Research*

Eric Goldwaser, M.D., Ph.D.

 *Next-Generation Therapies*

 *Basic Research*

Serena B. Gumusoglu, Ph.D.

 *Basic Research*

Elizabeth N. Holly, Ph.D.

 *Basic Research*

Artemis Iatrou, Ph.D.

 *Basic Research*

Brett Jones, M.D.

 *Next-Generation Therapies*

 *Basic Research*

Nikolaos Karalis, Ph.D.

 *Basic Research*

 *Next-Generation Therapies*

Aiste Lengvenyte, M.D.

 *Diagnostic Tools/Early Intervention*

Victor M. Luna, Ph.D.

 *Basic Research*

 *Next-Generation Therapies*

Chiara Maffei, Ph.D.

 *Diagnostic Tools/Early Intervention*

 *New Technologies*

Jordan Marrocco, Ph.D.

 *Basic Research*

 *Next-Generation Therapies*

Clare A. McCormack, Ph.D.

 *Basic Research*

Nicky J. Mehtani, M.D.

 *Next-Generation Therapies*

Luis Mercado, Ph.D.

 *Basic Research*

 *Next-Generation Therapies*

Heidi Catherine Meyer, Ph.D.

 *Basic Research*

Brittany D. Needham, Ph.D.

 *Next-Generation Therapies*

Anders M. Nelson, Ph.D.

 *Basic Research*

 *Next-Generation Therapies*

Lena K.L. Oestreich, Ph.D.

 *Basic Research*

Ashley C. Parr, Ph.D.

 *Basic Research*

Bruno Oriol Porras-Garcia, Ph.D.

 *Next-Generation Therapies*

María Sancho Alonso, Ph.D.

 *Basic Research*

Tarjinder Singh, Ph.D.

 *Basic Research*

Nili Solomonov, Ph.D.

 *Next-Generation Therapies*

Brad Verhulst, Ph.D.

 *Basic Research*

Isabella Wagner, Ph.D.

 *Basic Research*

Jessica J. Walsh, Ph.D.

 *Next-Generation Therapies*

Marc J. Weintraub, Ph.D.

 *Next-Generation Therapies*

Christian M. Wood, Ph.D.

 *Basic Research*

Mani Yavi, M.D.

 *Next-Generation Therapies*

EATING DISORDERS

Silvia G. Galfrè, Ph.D.

 *Basic Research*

Sasha C. Gorrell, Ph.D.

 *Next-Generation Therapies*

Roberta Haddad-Tóvolli, Ph.D.

 *Basic Research*

Marito Hayashi, Ph.D.

 *Basic Research*

Daniela Herrera Moro Chao, Ph.D.

 *Basic Research*

Kristin N. Javaras, Ph.D.

 *Basic Research*

Carolina Makowski, Ph.D.

 *Diagnostic Tools/Early Intervention*

Rose E. Presby, Ph.D.

 *Basic Research*

Laura E. Rupprecht, Ph.D.,

 *Basic Research*

Ames K. Sutton Hickey, Ph.D.

 *Basic Research*

Margaret L. Westwater, Ph.D.

 *Basic Research*

Andrew M. Wikenheiser, Ph.D.,

 *Basic Research*

OBSESSIVE-COMPULSIVE DISORDER (OCD)

Davide Aprile, Ph.D.

 *Basic Research*

Gerard J. Broussard, Ph.D.

 *Basic Research*

Gonçalo Cotovio, M.D., Ph.D.

 *Next-Generation Therapies*

John Falligant, Ph.D.

 *Basic Research*

Adam C. Frank, M.D., Ph.D.

 *Diagnostic Tools/Early Intervention*

 *New Technologies*

Silvia G. Galfrè, Ph.D.

 *Basic Research*

Allison E. Girasole, Ph.D.

 *Basic Research*

Christopher T. Sege, Ph.D.

 *Next-Generation Therapies*

 *New Technologies*

OTHER DISORDERS

ALZHEIMER'S DISEASE

Giulia R. Fois, Ph.D.

 *Basic Research*

Isabella Wagner, Ph.D.

 *Basic Research*

CATATONIA

Aaron D. Besterman, M.D.

 *Diagnostic Tools/Early Intervention*

EPILEPSY

Sarah D. Ackerman, Ph.D.

 *Basic Research*

Yiyao Zhang, Ph.D.

 *Basic Research*

 *Next-Generation Therapies*

FRAGILE X

J. Wren Kim, Ph.D.

 *Basic Research*

FRONTOTEMPORAL DEMENTIA

John Falligant, Ph.D.

 *Basic Research*

IMPULSIVE AGGRESSION

Debora Masini, Ph.D.

 *Basic Research*

 *Next-Generation Therapies*

INTELLECTUAL DISABILITY

Marta Garcia-Forn, Ph.D.

 *Basic Research*

INTELLECTUAL DISABILITY, CEREBRAL PALSY

Nataliia Kozhemiako, Ph.D.

 *Basic Research*

MULTIPLE SCLEROSIS

Erica B. Baller, M.D.

 *Basic Research*

Janos Fuzik, Ph.D.

 *Basic Research*

PARKINSON'S DISEASE

María Sancho Alonso, Ph.D.

 *Basic Research*

PEDIATRIC ACUTE-ONSET NEUROPSYCHIATRIC SYNDROME (PANS)

Silvia G. Galfrè, Ph.D.

 *Basic Research*

PROLONGED GRIEF DISORDER

Saren H. Seeley, Ph.D.

 *Basic Research*

SPINAL CORD INJURY

Anders M. Nelson, Ph.D.

 *Basic Research*

 *Next-Generation Therapies*

STROKE

Lena K.L. Oestreich, Ph.D.

 *Basic Research*

TRAUMATIC BRAIN INJURY

Chiara Maffei, Ph.D.

 *Diagnostic Tools/Early Intervention*

 *New Technologies*

POST-TRAUMATIC STRESS DISORDER (PTSD)

Seungwon (Sebastian) Choi, Ph.D.

 *Basic Research*

Constanza Garcia Keller, Ph.D.

 *Basic Research*

Matthew James Girgneti, Ph.D.

 *Basic Research*

Erin E. Hisey, Ph.D.

 *Basic Research*

Artemis Iatrou, Ph.D.

 *Basic Research*

Taylor J. Keding, Ph.D.

 *Basic Research*

 *Next-Generation Therapies*

Roger Marek, Ph.D.

 *Basic Research*

Heidi Catherine Meyer, Ph.D.

 *Basic Research*

Takuya Osakada, Ph.D.

 *Basic Research*

Joongkyu Park, Ph.D.

 *Basic Research*



Rebecca K. Reh, Ph.D.

 *Diagnostic Tools/Early Intervention*

Christopher T. Sege, Ph.D.

 *Next-Generation Therapies*

 *New Technologies*

Sydney Trask, Ph.D.

 *Next-Generation Therapies*

Brad Verhulst, Ph.D.

 *Basic Research*

Tao Xie, Ph.D.

 *Basic Research*

Wen Xin, Ph.D.

 *Basic Research*

PSYCHOSIS

Shokouh Arjmand, Pharm.D.

 *Next-Generation Therapies*

 *Basic Research*

David Benrimoh, M.D.

 *Diagnostic Tools/Early Intervention*

Michelle C.D. Bridi, Ph.D.

 *Basic Research*

Harriet R. Feldman, M.D., Ph.D.

 *Basic Research*

Kaushik J. Lakshminarasimhan, Ph.D.

 *Basic Research*

 *Next-Generation Therapies*

Stuart Oldham, Ph.D.

 *Basic Research*

Maria Belen Pardi, Ph.D.

 *Basic Research*

Adam J. Rossano, M.D., Ph.D.

 *Basic Research*

SCHIZOPHRENIA

Sarah D. Ackerman, Ph.D.

 *Basic Research*

Chinnakkaruppan Adaikkan, Ph.D.

 *Next-Generation Therapies*

 *Basic Research*

Shokouh Arjmand, Pharm.D.

 *Next-Generation Therapies*

 *Basic Research*

André M. Bastos, Ph.D.

 *Basic Research*

Laurie Bayet, Ph.D.

 *Basic Research*

David Benrimoh, M.D.

 *Diagnostic Tools/Early Intervention*

Michelle C.D. Bridi, Ph.D.

 *Basic Research*

Gerard J. Broussard, Ph.D.

 *Basic Research*

Christopher M. Davenport, Ph.D.

 *Basic Research*

John Falligant, Ph.D.

 *Basic Research*

Harriet R. Feldman, M.D., Ph.D.

 *Basic Research*

Giulia R. Fois, Ph.D.

 *Basic Research*

Wei-Kai Huang, Ph.D.

 *Basic Research*

Taeyoung Hwang, Ph.D.

 *Basic Research*

 *Next-Generation Therapies*

Valentina Ignatova, Ph.D.

 *Basic Research*

Srdan M. Joksimovic, Ph.D.

 *Basic Research*

Madhuvanathi Kannan, Ph.D.

 *Basic Research*

Kaushik J. Lakshminarasimhan, Ph.D.

 *Basic Research*

 *Next-Generation Therapies*

Eastman M. Lewis, Ph.D.

 *Basic Research*

Alana I. Mendelsohn, M.D., Ph.D.

 *Basic Research*

Sayan Nandi, Ph.D.

 *Basic Research*

Stuart Oldham, Ph.D.

 *Basic Research*

Maria Belen Pardi, Ph.D.

 *Basic Research*

Ashley C. Parr, Ph.D.

 *Basic Research*

Toby Pillinger, Ph.D.

 *Next-Generation Therapies*

Tyler Prestwood, M.D., Ph.D.

 *Basic Research*

Wei Qi, M.D.

 *Next-Generation Therapies*

 *New Technologies*

Giulia Quattrocolo, Ph.D.

 *Basic Research*

Adam J. Rossano, M.D., Ph.D.

 *Basic Research*

Kirsten E. Schoonover, Ph.D.

 *Basic Research*

Mototaka Suzuki, Ph.D.

 *Basic Research*

 *New Technologies*

Geoffrey Terral, Ph.D.

 *Basic Research*

Christopher W. Tschumi, Ph.D.

 *Basic Research*

Jessica J. Walsh, Ph.D.

 *Next-Generation Therapies*

Xiaoting Wu, Ph.D.

 *Basic Research*

SUICIDE PREVENTION

Steven Lamontagne, Ph.D.

 *Diagnostic Tools/Early Intervention*

Aiste Lengvenyte, M.D.

 *Diagnostic Tools/Early Intervention*

2023 Young Investigators Institutional Affiliations at the Time of the Grant

(in alphabetical order)

Aarhus University, Denmark (2)
Albert Einstein College of Medicine
American University
Boston University
Brigham and Women's Hospital
Central Institute of Mental Health, Mannheim, Germany
Centre for Addiction and Mental Health, University of Toronto, Canada (3)
Children's Hospital of Philadelphia
Children's Hospital of Philadelphia Research Institute
Cohen Veterans Bioscience
Columbia University (4)
Donders Institute for Brain, Cognition and Behaviour, The Netherlands
Duke University
Fundação Champalimaud, Portugal
George Washington University
Harvard University (5)
Harvard Medical School (3)
Howard University
Hugo W. Moser Research Institute at Kennedy Krieger, Inc.
Icahn School of Medicine at Mount Sinai (6)
Indian Institute of Science, India
Indiana University
INSERM, France

Institut de Bioenginyeria de Catalunya, Spain
Institut de Neurosciences de la Timone, France
Institut d'Investigacions Biomèdiques August Pi I Sunyer (IDIBAPS), Spain
Institut d'Investigacions Biomediques de Barcelona, Spain
Institut du Cerveau/Paris Brain Institute, France
Institute of Psychiatry/King's College London, U.K.
Interdisciplinary Institute for Neuroscience – CNRS, France
Johns Hopkins University (3)
Karolinska Institute, Sweden (2)
Lieber Institute for Brain Development
Massachusetts General Hospital (2)
Mayo Clinic College of Medicine
McGill University, Canada
McLean Hospital (4)
Medical College of Wisconsin
Medical University of South Carolina,
Murdoch Childrens Research Institute / University of Melbourne, Australia
National Institute of Child Health and Human Development (NICHD/NIH)
National Institute of Mental Health (NIMH/NIH) (2)
National Institute on Alcohol Abuse & Alcoholism/NIH
New York University (3)
New York University School of Medicine

2023 Young Investigators Institutional Affiliation at the Time of the Grant (continued)

Northwestern University (2)	University of Maryland, Baltimore
Norwegian University of Science and Technology, Norway	University of Milan, Italy
NYU Langone Health	University of Minnesota (2)
Pennsylvania State University	University of Montpellier, France
Princeton University (2)	University of New Mexico
Purdue University	University of North Carolina at Chapel Hill
Queensland University of Technology, Australia	University of Pennsylvania (2)
Rutgers University (2)	University of Pennsylvania School of Medicine
St. Jude Children's Research Hospital	University of Pisa, Italy,
Scintillon Institute	University of Pittsburgh (2)
Sleep Institute, Associação Fundo de Incentivo à Pesquisa, Brazil	University of Queensland, Australia
Stanford University (5)	University of South Carolina
Stockholm University, Sweden	University of Southern California
Temple University (2)	University of Texas Southwestern Medical Center at Dallas
Texas A&M University	University of Utah (3)
The Francis Crick Institute, U.K.	University of Vienna, Austria
Touro University	University of Virginia,
University of Alberta, Canada	University of Washington
University of Amsterdam, The Netherlands	University of Wisconsin
University of Arkansas for Medical Sciences	University of Wisconsin-Milwaukee
University of Bordeaux, France (2)	Vanderbilt University (3)
University of California, Berkeley	Washington University, St. Louis (2)
University of California, Los Angeles (2)	Washington University School of Medicine
University of California, San Diego (5)	Wayne State University
University of California, San Francisco (5)	Weill Cornell Medical College (2)
University of Cambridge. U.K.	West Virginia University
University of Cape Coast, Ghana	Yale University (2)
University of Houston	Yale University School of Medicine (3)
University of Iowa	

Scientific Council Dinner

Presenting the Klerman & Freedman Awards

July 28, 2023



ANNUAL KLERMAN PRIZE FOR EXCEPTIONAL CLINICAL RESEARCH

Danella M. Hafeman, M.D., Ph.D.

*University of Pittsburgh School
of Medicine*

HONORABLE MENTION

Linden Parkes, Ph.D.

*Rutgers University &
University of Pennsylvania*

ANNUAL FREEDMAN PRIZE FOR EXCEPTIONAL BASIC RESEARCH

Ritchie Chen, Ph.D.

*University of California,
San Francisco*

HONORABLE MENTIONS

Madeline Andrews, Ph.D.

Arizona State University

Neir Eshel, M.D., Ph.D.

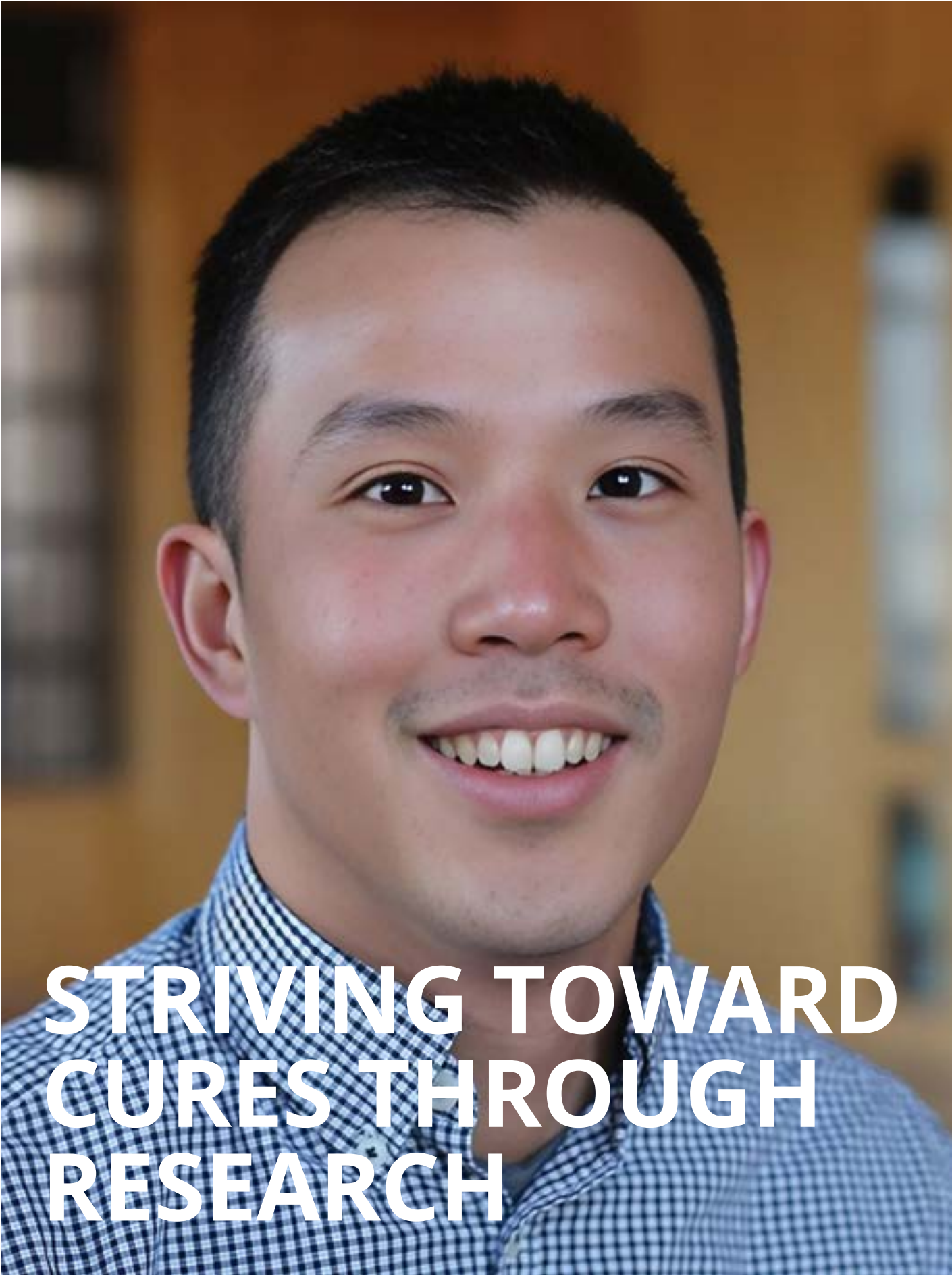
Stanford University

At BBRF's Scientific Council Dinner, Dr. Jeffrey Borenstein awarded the annual Klerman & Freedman Prizes to five outstanding BBRF Young Investigator Grantees for their exceptional clinical and basic research. The prizewinners are selected by committees of the Foundation's Scientific Council.

The researchers were recognized for significant findings related to schizophrenia, psychosis, depression, neurodevelopmental disorders, and the biology of the brain. Their important work is furthering the quest to identify the biological roots of mental illness to enable the development of new diagnostic tools, more effective and targeted treatments, and to pave the way toward prevention.



The evening concluded with a conversation between Dr. Jeffrey Borenstein and Dr. Holly Lisanby, a BBRF Scientific Council Member who is the Director of the Division of Translational Research at the National Institute of Mental Health. They discussed neuromodulation and a paper by Dr. Lisanby reviewing efforts to improve the safety and effectiveness of electroconvulsive therapy (ECT).

A close-up portrait of a young man with short dark hair, smiling warmly at the camera. He is wearing a blue and white checkered button-down shirt. The background is a soft-focus indoor setting with warm tones.

STRIVING TOWARD CURES THROUGH RESEARCH

Scientist Spotlight with: Ritchie Chen, Ph.D.

2023 BBRF Freedman Prize for Exceptional Basic Research

2020 BBRF Young Investigator Award

Visceral sensations, such as heart palpitations, hunger pangs, and pain, profoundly shape our mental state and behavior. However, physiological theories of emotion, proposed over a century ago, have been impossible to causally investigate in a controlled and temporally precise manner. Dr. Chen is inventing technologies for modulating affective and social behaviors, opening up new possibilities for treating mental health disorders. He has developed a cutting-edge technology that can non-invasively control cells throughout the mammalian body. He has identified the ultrasensitive and red-shifted light-sensitive protein called Channelrhodopsin ChRmine for non-invasive optogenetic control of electroactive cells throughout the brain and body. This technology enables transcranial optogenetic control of neuromodulator centers in the mouse brain without the need for intracranial surgery. Using this technology, he was able, non-invasively, to target serotonin neurons in the brain's raphe nucleus to enhance pro-social behavior in mice. He has since expanded this work to the heart, highlighting the key causal roles that bodily feedback can play in influencing emotional behavioral states.

WHAT IS THE CURRENT STATE OF YOUR BBRF FUNDED RESEARCH?

The research funded by my BBRF Young Investigator grant award has enabled our lab to pursue a range of different projects over the last year, about which we are very excited.

HOW DO YOUR INTERACTIONS WITH PEOPLE LIVING WITH MENTAL ILLNESS AFFECT YOUR RESEARCH AND VICE-VERSA?

Many individuals are faced with inadequate therapeutic options for treatment-resistant mental health conditions. As an engineer, I am interested in developing solutions to better diagnose and treat psychiatric and neurological disorders.

"I am grateful for the Freedman Prize, an unexpected and important milestone in my career. This award will remind me of the power of engineering technologies in driving new approaches for basic neuroscience research."

WHAT HAS BBRF SUPPORT MEANT TO YOUR CAREER?

Support from the BBRF strongly encouraged me to continue with my research to study the physiological basis of emotions.

IN THE BEST POSSIBLE SCENARIO, HOW WOULD YOUR WORK IMPACT THE PEOPLE LIVING WITH MENTAL ILLNESS AND THEIR FAMILIES?

Recently, a patient, who had been misdiagnosed with anxiety for a significant portion of their life, learned that they were suffering from dysautonomia, a disorder that disrupts the autonomic nervous system. The patient's symptoms, which included a rapid heartbeat, shortness of breath, chest discomfort, and an overwhelming sense of impending doom, exemplify the idea that "anxiety is a rational response when one feels as though their body is on the brink of collapse." While conditions such as anxiety are typically categorized under psychiatric disorders, the influence of physical feedback on these emotional states cannot be overstated. The patient I have mentioned expressed a hope that our work, which explores the connection between tachyarrhythmias and anxiety-like behavior, will inspire further research and stimulate discussions about the importance of recognizing physical symptoms in the diagnosis, understanding, and treatment of mental health disorders.

Scientist Spotlight with: **Danella M. Hafeman, M.D., Ph.D.**

2023 BBRF Klerman Prize for Exceptional Clinical Research

2019 BBRF Young Investigator Award

Dr. Hafeman's research focuses on youth diagnosed with or who are at risk for bipolar disorder. She is interested in understanding clinical and neural mechanisms of risk and resilience in these youth, with the goal of preventing progression of mood disorders in this vulnerable population. Much of her recent work has focused on predictors of bipolar disorder in youth at familial risk. In two papers, she has described dimensional predictors (e.g., anxiety/depression, mood lability, and subthreshold manic symptomatology) of a disorder in school-age and preschool youth. These prospective analyses have important clinical and research implications, describing a group of youth at ultra-high risk for the development of bipolar disorder. Dr. Hafeman hopes to use these data to construct a risk calculator for the development of bipolar disorder in at-risk youth, facilitating clinically relevant prediction at the individual level. She also aims to assess relationships between polygenic risk score and onset of bipolar disorder in these at-risk youth.

WHAT IS THE CURRENT STATE OF YOUR BBRF FUNDED RESEARCH?

I am very excited to be soon submitting an application for an R01 (a federally funded grant that is potentially career-sustaining) to follow up on the promising findings from my 2019 BBRF award. I propose to assess Within-Person Network Instability (WiPNI) as a potential biomarker for bipolar disorder on a larger scale. I have presented this work at the World Youth Bipolar Day, and the resulting publication is currently in preparation.

"Winning the Klerman Prize is a tremendous honor, and I am humbled to receive this prestigious award amongst an amazingly talented pool. I am also deeply grateful to BBRF for the support to take a risk on an innovative idea, as well as the opportunity for this recognition—it is incredibly meaningful at this point in my career."

HOW DO YOUR INTERACTIONS WITH PEOPLE LIVING WITH MENTAL ILLNESS AFFECT YOUR RESEARCH AND VICE-VERSA?

I am a physician-scientist, and no matter how well-funded my research is, I will never stop assessing and treating youth and young adults with bipolar disorder. I love my job as a psychiatrist in an outpatient setting, where I have the privilege of interacting with young people and their families, and really watching people improve, grow, and flourish. It is these interactions that inspire me to do my research: how can we do this better, and improve lives more quickly?

WHAT HAS BBRF SUPPORT MEANT TO YOUR CAREER?

The BBRF support has provided me with additional resources and substantial opportunity during the transition to my first R01 and facilitated the collection of compelling pilot data for a second R01 submission. I really feel that this support has provided a springboard for me to go from an "early career" to "mid-career" researcher.

IN THE BEST POSSIBLE SCENARIO, HOW WOULD YOUR WORK IMPACT THE PEOPLE LIVING WITH MENTAL ILLNESS AND THEIR FAMILIES?

I strive to do research that is person-centered and can have implications for the individual patient. Ideally, the impact of my research will lead to (1) the incorporation of biomarkers (e.g., imaging, polygenic risk score) to improve assessment and treatment choices for people living with bipolar disorder; and (2) improved strategies for early intervention (e.g., mindfulness interventions) that can change mood trajectories. Fundamental to these goals is decreasing stigma toward bipolar disorder and other forms of mental illness, through a recognition of biological underpinnings and hope for better outcomes.



2023 INTERNATIONAL MENTAL HEALTH RESEARCH SYMPOSIUM

BBRF's annual symposium was held both in-person and virtually in 2023. Five 2023 Outstanding Achievement Prizewinners gave presentations on topics that included schizophrenia, obesity and bipolar disorder, pediatric mood and anxiety disorders, and cognitive neuroscience. The symposium also featured a presentation from a speaker representing Special Olympics International, the winner of the 2023 Pardes Humanitarian Prize in Mental Health.

This year's program was reflective of the greatly accelerating pace of discovery in mental health research as well as the expanded range of studies the Brain & Behavior Research Foundation has grown to support.

An overview of the entire Symposium was provided by Dr. Carol Tamminga, a BBRF Scientific Council member who served as the Symposium moderator.

The entire symposium is available to watch on the BBRF website:

<https://bbrfoundation.org/blog/2023-international-mental-health-research-symposium-presentations>



Geoffrey Simon, Dr. Roger McIntyre, Dr. Katie McLaughlin, Dr. Elizabeth Phelps, Dr. Amy Pinkham, Dr. Philip Harvey, Dr. Jeffrey Borenstein, and Dr. Carol Tamminga.

Presentations

Self-knowledge in Schizophrenia: Importance, Characteristics, and Treatment

Philip D. Harvey, Ph.D.

*Leonard M. Miller School of Medicine, University of Miami
VA Medical Center, Miami*

BBRF LIEBER PRIZEWINNER FOR OUTSTANDING ACHIEVEMENT IN
SCHIZOPHRENIA RESEARCH

Social Cognition and Social Difficulties in Schizophrenia

Amy E. Pinkham, Ph.D.

The University of Texas at Dallas

BBRF MALTZ PRIZEWINNER FOR INNOVATIVE AND PROMISING
SCHIZOPHRENIA RESEARCH

Does Obesity Metastasize to the Brain: Implications for Clinical Care and Identifying the Causes and Cures for Persons Living with Bipolar Disorder

Roger S. McIntyre, M.D., FRCPC

University of Toronto, Canada

BBRF COLVIN PRIZEWINNER FOR OUTSTANDING ACHIEVEMENT IN
MOOD DISORDERS RESEARCH

The Long Shadow of Childhood Adversity: Implications for Children's Brain and Behavioral Development

Katie McLaughlin, Ph.D.

University of Oregon

BBRF RUANE PRIZEWINNER FOR OUTSTANDING ACHIEVEMENT IN
CHILD & ADOLESCENT PSYCHIATRIC RESEARCH

The Human Amygdala, Threat, and Anxiety: Translational Progress and Challenges

Elizabeth A. Phelps, Ph.D.

Harvard University

BBRF GOLDMAN-RAKIC PRIZEWINNER FOR OUTSTANDING
ACHIEVEMENT IN COGNITIVE NEUROSCIENCE RESEARCH

Minds Matter: Mental Health and Intellectual Disabilities

Károly Mirnics, M.D., Ph.D.

*Speaking on behalf of Special Olympics International, the
Pardes Humanitarian Award Prizewinner*

We would also like to thank our 2023 Sponsors

BRONZE SPONSOR



VIP SPONSOR





The Pardes Humanitarian Prize in Mental Health

Bestowed annually since 2014, the Pardes Prize is named in honor of Dr. Herbert Pardes, the first recipient of the award. The Prize recognizes a person(s) or organization whose humanitarian work is transformative and of great magnitude, changing lives and bringing the joy of living to those facing challenges to mental health. It was established to honor those who comprehensively care, teach, investigate, work, and passionately advocate for improving the mental health of society and have had a powerful impact on reducing the pain inflicted by psychiatric illness.



Dr. Károly Mirnics accepting the Pardes Humanitarian Prize in Mental Health Award on behalf of Special Olympics International from Dr. Jeffrey Borenstein.

The recipient of the Pardes Humanitarian Prize in Mental Health is chosen by a distinguished international Selection Committee from nominations solicited worldwide. The Prize focuses public attention on the burden of mental illness on individuals and on society, and the urgent need to expand and enhance mental health services in the United States and globally.

The 2023 Pardes Humanitarian Prize in Mental Health was awarded on October 27th and honored an extraordinary organization. Special Olympics International is a leading advocate for the inclusion of people with disabilities and a

powerful force in the efforts to reduce stigma and raise awareness about the mental health needs of individuals with intellectual disabilities.

The 2023 Honorary Pardes Humanitarian Prize in Mental Health was awarded to Dr. Henry Jarecki for his profound humanitarian impact on the world through his unique and lasting contribution to preserving academic and scientific freedom.

PREVIOUS PRIZE WINNERS

2022

Altha J. Stewart, M.D.,
Robert van Voren, FRCPsych (HON)

Honorary Tribute:
Clubhouse International, Sean Mayberry

2021

Kay Redfield Jamison, Ph.D., Elyn R. Saks,
J.D., Ph.D., & Charlene Sunkel

Honorary Tribute:
*John M. Davis, M.D.,
Michael R. Phillips, M.D., MPH, &
Norman Sartorius, M.D., Ph.D., FRCPsych*

2020

Myrna M. Weissman, Ph.D.
& Sir Michael Rutter, FRS

Honorary Tribute:
E. Fuller Torrey, M.D.

2019

William T. Carpenter, Jr., M.D.

Honorary Tribute:
*Cynthia Germanotta &
Born This Way Foundation*

2018

Judge Steven Leifman

Honorary Tribute:
Suzanne and Bob Wright

2017

Doctors Without Borders/
Médecins Sans Frontières

Honorary Tribute:
Constance E. Lieber

2016

Vikram Patel, Ph.D., F.Med.Sci. &
Charles F. Reynolds, III, M.D.

Honorary Tribute:
Senator Edward M. Kennedy

2015

Beatrix (Betty) A. Hamburg, M.D.
and David A. Hamburg, M.D.

Honorary Tribute:
Rosalynn Carter

2014

Herbert Pardes, M.D.

INTERNATIONAL AWARDS DINNER

OCTOBER 27, 2023

The BBRF International Awards Dinner honored the winners of the Pardes Humanitarian Prize in Mental Health as well as the five Outstanding Achievement Prizewinners who are advancing the science that is changing what it means to live with a mental illness. Winners are selected by special committees of the BBRF Scientific Council.

LIEBER PRIZE

Outstanding Achievement in Schizophrenia Research



Philip D. Harvey, Ph.D.

Leonard M. Miller Professor of
Psychiatry and Behavioral Sciences
& Senior Health Research Scientist

Leonard M. Miller School of Medicine,
University of Miami

Senior Health Research Scientist,
VA Medical Center, Miami

• BBRF Scientific Council

RUANE PRIZE

Outstanding Achievement in Child & Adolescent Psychiatric Research



Katie McLaughlin, Ph.D.

Executive Director, Ballmer Institute
Knight Chair and Professor of
Psychology

University of Oregon

• 2016 Klerman Prizewinner for
Exceptional Clinical Research

• 2013 Young Investigator

MALTZ PRIZE

Innovative & Promising Schizophrenia Research



Amy E. Pinkham, Ph.D.

Professor of Psychology

The University of Texas at Dallas

GOLDMAN-RAKIC PRIZE

Outstanding Achievement in Cognitive Neuroscience



Elizabeth A. Phelps, Ph.D.

Pershing Square Professor of Human
Neuroscience, Department of
Psychology

Harvard University

COLVIN PRIZE

Outstanding Achievement in Mood Disorders Research



Roger S. McIntyre, M.D., FRCPC

Professor of Psychiatry and
Pharmacology

University of Toronto, Canada

Chairman & Executive Director,
*Brain and Cognition Discovery
Foundation, Toronto*

Professor,
Guangzhou Medical University, China

• 2007 BBRF Independent Investigator



Healthy Minds, hosted by BBRF's President & CEO Dr. Jeffrey Borenstein, aims to remove the stigma of mental illness and demonstrate that with help, there is hope. The series features inspiring personal stories from people who have experienced mental health issues, as well as the latest information from experts on new approaches to the diagnosis, treatment, and prevention of mental illness.

Healthy Minds is available on PBS stations around the U.S. and online at PBS.org.

Season 9 is now available to watch at: <https://www.pbs.org/show/healthy-minds-with-dr-jeffrey-borenstein/>

Previous seasons can be found online at: www.bbrfoundation.org/healthyminds-tv

Season 8 Episodes:

- 801 – *Chemical Dependency: A Holistic Approach to Treatment*
- 802 – *Help for Veterans and Military Families: The Headstrong Project*
- 803 – *Schizophrenia: Understanding Diagnosis and Treatment*
- 804 – *Suicide Prevention, Part One: What You and Your Family Need to Know*
- 805 – *Suicide Prevention, Part Two: What You and Your Family Need to Know*
- 806 – *Wisdom and Healthy Aging*
- 807 – *Childhood Anxiety and Depression: What Every Parent Needs to Know*
- 808 – *Eating Disorders: Diagnosis and Treatment*
- 809 – *Borderline Personality Disorder*
- 810 – *Bipolar Disorder, Part One: A Conversation with Kay Redfield Jamison, Ph.D.*
- 811 – *Bipolar Disorder, Part Two: A Conversation with Kay Redfield Jamison, Ph.D.*
- 812 – *NAMI: National Alliance on Mental Illness*
- 813 – *Creating Community and Giving Hope: Clubhouse International*

Dr. Borenstein & *Healthy Minds* Win 2023 ACNP Media Award

In December 2023, the American College of Neuropsychopharmacology (ACNP) named Jeffrey Borenstein, M.D. as the recipient of its 2023 Media Award. The award was presented at the 62nd Annual Meeting of the ACNP in recognition of major contributions to the education of the public about mental illness and substance abuse research, and the positive impact of research on treatment. The award is intended to be an expression of appreciation from the College toward outstanding public education leaders who provide complete, accurate, and unbiased information to society about brain diseases.

As noted in the ACNP press release:

Dr. Borenstein's communications work is exemplary and embodies the nature of the Media Award by providing complete, accurate, and unbiased information to our society about brain diseases.

Over the years "Healthy Minds" has won many awards, including 17 "Telly" awards and has been nominated three times for an "Emmy" award. This program performs a service that no other popular communications vehicle in any medium performs.



By regularly reaching millions of households, it delivers urgently needed public information messages about mental health and the challenges of living with mental illnesses. It also humanizes the face of both neuropsychiatric research and psychiatry practice because of the intimate and always approachable way Dr. Borenstein interviews his guests. The show is devoid of jargon and makes the subject approachable. Importantly, the program and its host are consistently hopeful and each week Dr. Borenstein opens each episode by saying "with help there is hope"; reminding people that if they can seek help, they stand to benefit.

The Emmy-nominated series is produced by the Brain & Behavior Research Foundation, presented by Connecticut Public Television (CPTV) and distributed by the National Educational Telecommunications Association (NETA). Funding for season 8 was provided by The American Psychiatric Association Foundation, The Bank of America Charitable Gift Fund, and The John & Polly Sparks Foundation.

Meet the Scientist Webinar Series

Dr. Jeffrey Borenstein hosts the free monthly “Meet the Scientist” webinar series where leading brain and behavior researchers discuss their current work on the latest in new technologies, early intervention strategies, and next-generation therapies for mental illness. Each hour-long webinar includes time for researchers to answer questions posed by the audience participants. This popular series offers the public access to some of the world’s top scientists who discuss their cutting-edge research.

All webinars are available for viewing on the BBRF website.

The following Webinars were offered in 2023:



Sex Differences in Mental Health Disorders

Tuesday, January 10, 2023

Erin S. Calipari, Ph.D.

Vanderbilt University School of Medicine



What Genetics is Telling Us About Substance Use Disorders

Tuesday, July 11, 2023

Sandra Sanchez-Roige, Ph.D.

University of California, San Diego



Therapeutic Targeting of the Microbiome for Neurodevelopmental Disorders

Tuesday, February 14, 2023

Shelly A. Buffington, Ph.D.

University of Texas Medical Branch



Morning Light Therapy for Tourette’s Disorder

Tuesday, August 8, 2023

Emily Ricketts, Ph.D.

University of California, Los Angeles



Neuroscience of Stress and Metabolism

Tuesday, March 14, 2023

Rachel Amy Ross, M.D., Ph.D.

Albert Einstein College of Medicine/ Psychiatric Research Institute of Montefiore and Einstein



A Precision-Health Approach to Bipolar Disorder

Tuesday, September 12, 2023

Sarah H. Sperry, Ph.D.

University of Michigan



Changes in Infant Emotion Regulation Following Maternal Treatment for Postpartum Depression

Tuesday, April 11, 2023

Ryan J. Van Lieshout, M.D., Ph.D., FRCPC

McMaster University, Canada



Self-Injurious Thoughts & Behaviors in Youth

Tuesday, October 10, 2023

Mindy Westlund Schreiner, Ph.D.

University of Utah



How the Brain’s Dopamine Circuitry Helps Regulate Cognitive Flexibility and Reward-Seeking

Tuesday, May 9, 2023

Nikhil Urs, Ph.D.

University of Florida



Pediatric PTSD: Neurobiology and Treatment

Tuesday, November 14, 2023

Ryan J. Herringa, M.D., Ph.D.

University of Wisconsin-Madison



Developing New Treatments for Childhood Anxiety and OCD: Can Cognitive Control Help Kids Grow Out of Illness?

Tuesday, June 13, 2023

Kate D. Fitzgerald, M.D.

Columbia University



Understanding Resilience to Schizophrenia through Genetics

Tuesday, December 12, 2023

Jonathan Hess, Ph.D.

SUNY Upstate Medical University

Helping Children & Adolescents with Emotional Problems

A Q&A with Daniel S. Pine, M.D.



Daniel S. Pine, M.D.

Chief, Emotion and Development Branch

Chief, Child and Adolescent Research in the Mood and Anxiety Disorders Program

National Institute of Mental Health (NIMH)

This special webinar was broadcast via Zoom on November 30, 2023.

Teachers, school counselors and other educational professionals are on the front line of dealing with kids with mental health issues and can often be among some of the first people to see that a child is struggling. Enhancing the potential for early intervention is important and because educational professionals have relationships with students and their families, they are often the people who guide students and their families to resources. As educational professionals learn more about mental health issues, their ability to make appropriate referrals for evaluation will improve for students and their families.

This conversation shared with parents and educators the key symptoms and attributes associated with pediatric mood and anxiety disorders. BBRF President & CEO Dr. Jeffrey Borenstein and Dr. Daniel Pine discussed novel insights for improving treatment and offered tools to help families and educators address how best to help children and teens with emotional issues. The webinar also highlighted particularly pressing questions in research on pediatric mood and anxiety disorders and discussed future research.



The webinar is available on the BBRF website:

<https://bbrfoundation.org/event/helping-children-adolescents-emotional-problems>



In 2019, the **Moritz Hilder Innovative Brain Research Fund** was established by the Trustee of the Jane Hilder Harris Trust with an endowment gift of \$3.5 million. It was created to preserve and honor the memory of Moritz Hilder, the father of the late Jane Hilder Harris.

This generous endowment will be held in perpetuity to advance medical research with the objective of gaining a basic understanding of post-traumatic stress disorder (PTSD) and its prevention, treatment and cure. Primary emphasis will be given to research involving innovative concepts where, although there may be a high risk of failure, the rewards of success would be substantial, and to researchers who typically would not be in a position to secure funding from more traditional funding sources.

The Moritz Hilder Innovative Brain Research Fund is held in a professionally managed, separate endowment fund. On an annual basis, 5% of the endowment fund will be expended to support PTSD research. A special committee of the BBRF Scientific Council selects which BBRF Grants will be funded by this Endowment. In 2023, the Endowment provided \$197,008 for the funding of six PTSD research projects.

We are deeply honored to have received this generous and impactful gift.

THANK YOU TO OUR DONORS

Your support this year enabled us to fund \$10.3 million to scientists looking for better treatments, cures, and methods of prevention for mental illness.

Since 1987, in partnership with you, our donors, BBRF has awarded more than \$450 million to fund more than 6,500 grants to more than 5,400 leading scientists around the world.

With your help BBRF has been able to foster new research pathways that led to transformative breakthroughs. We deeply appreciate your support and commitment to advance psychiatric research.



BENEFITS OF BECOMING A RESEARCH PARTNER

Barbara Toll

BBRF Board Member & Research Partner

Many BBRF donors have a very personal interest in brain and behavior research. They know from often difficult, first-hand experience the devastation mental illness can bring upon family and friends, and they know that research will ultimately bring about better understanding and treatments.

Our **Research Partners Program** offers donors the opportunity to personally select and support scientists based on various criteria, including, but not limited to, illness specialty area or specific institutions, or a combination of these. Researchers are selected by the donor (our Research Partner) after members of our all-volunteer Scientific Council have conducted an independent peer review of the submitted applications and have made their recommendations for grant awards.

The **Research Partners Program** enables donors to choose among the best and brightest scientists and the most promising, cutting-edge proposals in mental illness research.

The results from these studies often provide the pilot data needed to apply for much larger federally funded grants (from the National Institute of Mental Health and the N.I.H., for example).

To date the BBRF **Research Partners Program** has funded more than 1,500 research grants.

To learn more about the Research Partners Program, please contact us at **646-681-4889** or **researchpartner@bbrfoundation.org**.

Visit us at bbrfoundation.org/researchpartners.

Research Partners Program

JAN ABRAMS

Jan Abrams Investigators

Young Investigator

Aparna Bhaduri, Ph.D.

University of California, Los Angeles

Young Investigator

Rachel D. Reetzke, Ph.D.

*Hugo W. Moser Research Institute at
Kennedy Krieger, Inc — Johns Hopkins
University*

LYNN & JOEL ALTSCHUL

Jon Altschul Investigators

Young Investigator

Anouar Khayachi, Ph.D.

McGill University

Young Investigator

Heidi Catherine Meyer, Ph.D.

Boston University

ARAMONT CHARITABLE FOUNDATION

Aramont Charitable Foundation
Investigator

Young Investigator

Xingjian Zhang, Ph.D.

University of California, Los Angeles

ANONYMOUS

Young Investigator

Lindsay D. Oliver, Ph.D.

*Centre for Addiction & Mental Health
University of Toronto*

ANONYMOUS

Young Investigator

Andrea Boscutti, M.D.

University of Texas Health — Houston

SIDNEY R. BAER, JR. FOUNDATION

Sidney R. Baer, Jr. Foundation
Investigator

Young Investigator

David Benrimoh, M.D.

Douglas Mental Health University Institute

BARRETTA FAMILY FOUNDATION

Gina & Russell Barretta Investigator

Young Investigator

Neal D. Amin, M.D., Ph.D.

Stanford University

BARBARA & MICHAEL BASS

Barbara and Michael Bass Investigator

Young Investigator

Guusje Collin, M.D., Ph.D.

Radboud University

JAN & BARRY BRANDT

Brandt Family Investigator

Young Investigator

Tyler Prestwood, M.D., Ph.D.

Stanford University

FREDERICK & ALICE COLES AND THOMAS & NANCY COLES

Frederick & Alice Coles and Thomas &
Nancy Coles Investigators

Young Investigator

Sarah H. Sperry, Ph.D.

University of Michigan

Young Investigator

Joongkyu Park, Ph.D.

Wayne State University

ANONYMOUS

The Quincy Ells Fund Investigator

Young Investigator

Megan A. Boudewyn, Ph.D.

University of California, Santa Cruz

ANONYMOUS

Nathan And Phyllis Goodman
Investigators

Young Investigator

Nicole M. Benson, M.D.

McLean Hospital - Harvard University

Young Investigator

Garrett A. Kaas, Ph.D.

Vanderbilt University Medical Center

ANONYMOUS

Dana W. Investigators

Young Investigator

Stephanie L. Hickey, Ph.D.

Michigan State University

Young Investigator

Toby Pillinger, Ph.D.

*Institute of Psychiatry/King's College
London*

BOB & RITA ELMEN FAMILY FOUNDATION

Bob & Rita Elmen Family Foundation
Investigator

Young Investigator

Ana P. Silva, Ph.D.

*Centre for Addiction and Mental Health —
University of Toronto*

FAMILIES FOR BORDERLINE PERSONALITY DISORDER RESEARCH

Families For Borderline Personality
Disorder Research Investigators

Young Investigator

Lois W. Choi-Kain, M.D.

McLean Hospital - Harvard University

Young Investigator

Sarah Kathryn Fineberg, M.D., Ph.D.

Yale University

Young Investigator

Fabian Streit, Ph.D.

*Central Institute of Mental Health,
Mannheim — University of Heidelberg*

Young Investigator

Jenna M. Traynor, Ph.D.

McLean Hospital — Harvard University

GRACE JONES RICHARDSON FAMILY FOUNDATION

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Christopher F. Miehlisch
Delaphine Miller
Lori Miller-Levine
Duncan N. Mitchell
Neil Molberger
Gregory A. Monk
Juan (Joji) Maniquis Montelibano
Frank Morgan
Matthew P. Morgan
Beth Morse's beloved son
Mark I. Moskowitz
Virginia (Ginny) L. Mott
Isabelle Moua
Roland E. Mueller
Patrick Rory Muldoon
Ronald P. Mullan
Ronald (Ronnie) Mullan
John (Jack) M. Mulvoy
Bradley Munroe
Andrew (Andy) L. Munson
John Naiman
Cassandra Narburgh
Adam Neely
Barry J. Nelson
Karen S. Nelson
Priscilla Nelson
Christopher J. Neubecker
Kirk D. Newell
Patrick Nolan
Nicholas Nordin
Kyrallynn R. Nycum
Greta L. Oclair's beloved loved one
June E. O'Connor
Donna M. O'Dell
Emily Odle
Donald M. O'Hara, Jr.
Jesse E. Ohlin
Rachel L. Okon
Alyssa G. O'Neill
Matthew Ornstein
Cameron R. Orr
Sandra (Sandy) Otero
Pradeep Padukone
Barbara W. Page
Sri Hari Palacio
Romano Paliska
James D. Palmer III

Albert L. Pan
Robert Parenteau
Arthur Peck, M.D.
Samantha (Sam) Saint Aubyn
Peluso-Swartzlander
Mary Person-Huebner
William Brodie Phillips
Sybil Pierce
Jessica A. Piesman
Michael P. Piet
Asuncion Pla Esteve
Kali-Luna Polhill-Koenig
Jennifer E. Porath Gordon Novelich
Mollie Porvancher-Adinolfi
Nicholas M. Pouh
George C. Powell, Sr.
DeLana Powers
Ankita Pradhan
Donna M. Pritchett
Peppino Puleo
Joel M. Purrington
Vu Huynh Quang
Dylan Quast
Bert R. Queen
Fiza Quraishi
Ramez E. Qureshi
Jerome (Jerry) Rabkin
Luca Rader
Arthur J. Radin, C.P.A.
Matthew R. Radula
Hotan Rafiee-Tary
Jane (Janie) E. Ragel
Lisa M. Raimondo-Nowell
Sidharth Ramakrishnan
Cassandra (Cassie) N. Ray
Anthony M. Razziano
Evan G. Rea
William (Bill) Reader
John Reason
Mason J. Reed
Brian E. Reese
Steve Reinlieb
Marcia A. Relph
Samuel D. Rendall
Michael Reschke
Eamonn P. Reynolds
Scott T. Richards
James Richardson
William Richardson
Priscilla Riddick-Trotter
Mary Frances Ritsko
William (Bill) H. Ritsko
Jonathon J. Robbins

Sarah M. Robbins
Will Robinson
David A. Rock
Carolee Rogers
David Rogers
Elizabeth Rolland
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Thomas Rorick
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Christina (Chrissy) Rossi
Matthew S. Rothman
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Sylvia Shick
Kimberlee A. Shuman
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Benjamin L. Silver
Beatrice Silverman
Sheldon Silverman
Duncan N. Simic
Benjamin Simmons-Courtney
Kevin Simms
Marcia Simon-Kaplan

Memorial Tributes (continued)

John T. Sinnott	Joseph Tursone	Robert T. Woods
Carol R. Smith	Colby Twinam	Surekha Yadawad
Edmund Smith	Alexa N. Uroskie-Smith	Edward A. Yakamavage
Edward J. Smith	Brett A. Van Vort	Hizeko Yoshimura
George J. Smith	Glen P. Vandehey	Genevieve (Gene) Young
Marnie Smith	Charles Varkoly	Neal Zafran
William H. Smith II	Robert (Bob) L. Veenstra	Timothy F. Zalent
Donald E. Snyder	Harriet Vicente	Marilyn Zalokar
Drew Sobotka	Joaquin B. Villarreal	Anna Zarski
Patrick Sorrell	Allen E. Vincent	Peter W. Zartman
Nathan A. Soukup	Gregory Von Burg	Edward N. Ziegler
Eleanor (Ellie) Southworth	Paul Von Burg	Gladys P. Ziegler
Louis A. Spadaccini	Alexandra von Wussow's beloved father	Barry L. Zimmerman
Gena Spaulding	Ken Vorisek	
Anneke Speller	Riley P. Wachhorst	
Morton Spool	Wm. F. Wagner, Jr.	
Susan Spool	Jonathan Waldman	
Jeffrey T. Sramek	K.T. Walker	
Doretha St. Clair	Capt. Anders (Andy) G. Wallin II	
Ian J. Stancato	Tordis Wallin	
Brian A. Stapleton	Charlie M. Walls-Rowland	
John A. Stapleton	Thomas Walsh	
Gregory L. Starling	Sharon Wang	
Tyler R. Starling	Kenneth R. Warning	
Edward G. Steinmetz, Jr.	Charlotte S. Weiss	
Marlene H. Steiskal	Susan L. Wessman	
Finley S. Still	Robert M. Wetzel	
Barbara Stoller	Allan Wexler	
Mary J. Strub-Caulkins	Timothy L. Whitmer	
Hilda R. Studebaker	Dan Wicker, Jr.	
Daniel Sullivan	Michael G. Wieman	
Kristin M. Sullivan	Jon K. Wilbur	
Anne Sykes	Gertrude Wildgruber	
Martin P. Szuba, M.D.	Ronald C. Wilkson	
H. Vonn Taylor	Amy L. Williams	
John W. Telford	John C. Williams	
Charles S. Testa	Mark Williams	
Beth Thompson	Andrea Williams-Barnes	
John Thornley	Lynn Williams-Figg	
Nicholas Tindall	Martin S. Willick, M.D.	
Brian Tittle	Peter B. Wilson	
Jake M. Topley	Richard K. Wilson	
Troy Torres, Jr.	Timothy (Timo) Wilson	
Victoria (Tori) Trang Cook	Barbara A. Winkler-Monsanto, M.D.	
Richard Trommer	Mike Winn	
Donald Trybula	Douglas Wistner	
Alexandros Tsaoussis-Maddock	Cheryl E. Wolfe	
Liz Tucker	June Wolfe	
Robert T. Tucker	Farrell J. Wolfson	
Doris H. Tufte	Kenneth A. Wood	
Adele C. Tursone	Alan H. Woodard	
	Ryan M. Woodland	

Financial Summary*

Consolidated Statement of Financial Position

	DECEMBER 31, 2023	DECEMBER 31, 2022
ASSETS		
Current Assets		
Cash and cash equivalents	\$26,905,317	\$19,059,866
Investments, at fair value, current portion	14,629,688	11,104,800
Contributions receivable	415,480	2,202,142
Pledges receivable, current portion	7,470	190,277
Prepaid expenses and other assets	230,236	225,037
Total Current Assets	42,188,191	32,782,122
Pledges receivable, net, less current portion	9,738	9,738
Assets held in charitable remainder trust	1,885,836	1,701,437
Fixed assets, net	9,684	6,884
Investments, at fair value, less current portion	8,947,449	8,947,449
Right-of-Use Asset	125,457	424,126
Total Assets	\$53,166,355	\$43,871,756
LIABILITIES AND NET ASSETS		
Current Liabilities		
Accounts payable and accrued expenses	\$75,069	\$95,271
Grants payable	14,887,604	15,522,195
Operating lease liability, current portion	148,179	353,580
Accrued compensation	48,077	40,776
Annuities payable	826,641	778,644
Charitable gift annuities payable	13,599	142,918
Total Current Liabilities	15,999,169	16,933,384
Operating lease liability, net, less current portion	—	144,891
Total Liabilities	15,999,169	17,078,275
Net Assets		
Without donor restrictions	26,492,489	17,792,107
With donor restrictions	10,674,697	9,001,374
Total Net Assets	37,167,186	26,793,481
Total Liabilities and Net Assets	\$53,166,355	\$43,871,756

* The Foundation's complete audited financial statements are available on our website.

Consolidated Statement of Activities

	YEAR ENDED DECEMBER 31, 2023	YEAR ENDED DECEMBER 31, 2022
SUPPORT AND REVENUE		
Contributions	\$14,613,749	\$12,828,748
Special events, net	89,157	46,716
Contribution of services	2,048,285	2,116,704
Bequests	5,022,129	8,618,365
Net realized and unrealized gains (losses) on investments	3,929,558	(4,857,163)
Net appreciation (depreciation) of assets held in charitable remainder trusts	184,399	(426,076)
Dividend and interest income	908,154	268,882
Total Support and Revenue	26,795,431	18,596,176
EXPENSES		
Program Services		
Research grants and awards	9,232,426	8,868,584
Scientific advancement	2,309,627	2,414,690
Program support	2,352,532	2,258,132
Total Program Services	13,894,585	13,541,406
Supporting Services		
Fundraising**	897,389	850,240
Administration**	1,629,752	1,565,890
Total Supporting Services	2,527,141	2,416,130
Total Expenses	16,421,726	15,957,536
Change in Net Assets	10,373,705	2,638,640
Net Assets, beginning of year	26,793,481	24,154,841
Net Assets, end of year	\$37,167,186	\$26,793,481

** All fundraising and administration expenses are funded by specially designated grants.

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