BRAIN & BEHAVIOR RESEARCH FOUNDATION

2024 ANNUAL REPORT

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

BRAIN & BEHAVIOR
RESEARCH FOUNDATION
Awarding **NARSAD** Grants

ADDICTION, ADHD, ANXIETY, AUTISM, BIPOLAR DISORDER, BORDERLINE PERSONALITY DISORDER, DEPRESSION, EATING DISORDERS, OCD, PSYCHOSIS, PTSD, SCHIZOPHRENIA, SUICIDE PREVENTION.

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.

100% OF EVERY DOLLAR DONATED FOR RESEARCH GOES TO RESEARCH

Jeffrey Borenstein, M.D.
President & CEO

Judith M. Ford, Ph.D.
President, Scientific Council

Geoffrey Simon
Chair, Board of Directors

Dear Friends,

Philanthropic support for research is more important now than ever. Significant changes and uncertainty in government funding have made scientific research funded by Brain & Behavior Research Foundation (BBRF) grants absolutely crucial in supporting research leading to innovative new therapies that help people living with psychiatric illness.

The Brain & Behavior Research Foundation (BBRF) is the world's largest private funder of mental health research grants. In 2024 BBRF funded over $11 million in research grants across a broad spectrum of brain illnesses. Since 1987 BBRF has awarded more than $462 million in research grants to more than 5,600 scientists around the world who are working to find better treatments, cures, and methods of preventing mental illness.

Many important BBRF-funded research breakthroughs were published in major psychiatric and medical journals during 2024. This annual report highlights the *Leading Research Achievements by BBRF Grantees, Prizewinners & Scientific Council Members*. These important scientific advances and research discoveries underscore the vital role BBRF plays in helping people and families living with mental illness.

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. This past year the Scientific Council evaluated more than 900 grant proposals and awarded 150 Young Investigator Grants, which fund two years of research for $35,000 per year, and 10 Distinguished Investigator Grants, which fund one year of research for $100,000 per year.

In July, BBRF hosted its annual *Scientific Council Dinner* and celebrated the exceptional life and accomplishments of the late Dr. Herbert Pardes, who led the BBRF Scientific Council from its inception nearly 40 years ago. BBRF also awarded the highly esteemed Klerman & Freedman Prizes at the event.

In October, at BBRF's *International Mental Health Research Symposium*, six award-winning scientists presented their outstanding 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 2024 Symposium are available to watch online at: www.bbrfoundation.org/event/international-mental-healthresearch-symposium.

At the *Annual Awards Dinner* following the Symposium, we presented the BBRF Outstanding Achievement Prizes, as well as the Pardes Humanitarian Prize in Mental Health.

BBRF's periodical, ***Brain & Behavior Magazine***, highlights many of the latest mental health breakthroughs of our grant recipients. In order to provide the most timely mental health news, BBRF publishes a weekly email newsletter, ***"eNews,"*** that is available free of charge to donors, the scientific community, and the public. It features research breakthroughs of BBRF grantees, award-winners, and Scientific Council members.

Meet the Scientist is our free monthly webinar series that offers viewers the latest findings on psychiatric illnesses from international experts in the field of brain and behavior research.

BBRF also produces the award-winning television series ***Healthy Minds***, which is broadcast on public television stations throughout the U.S. It is also available online at: pbs.org. The series provides vital information about psychiatric conditions and treatments, as well as cutting-edge research advancements. Season 9 is available to watch online at: https://bbrfoundation.org/healthy-minds-tv and Season 10 will be available on PBS this fall.

BBRF is very enthusiastic about three new initiatives to raise mental health awareness among news reporters and the public: The Carter Center's Mental Health Journalism Fellowship Program and Mental Health Parity Newsroom Collaborative, The Poynter Institute for Media Studies' Mental Health Reporting Project, and MindSite News.

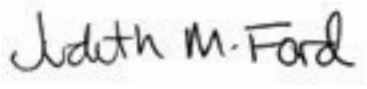
We are inspired by the magnitude and impact of the discoveries being made by the thousands of scientists BBRF has been able to fund thanks to your generous ongoing support.

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. While steady 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**.

With all the uncertainty now facing mental health research, your solid commitment to help BBRF guarantee funding for research grants that develop improved treatments, cures, and methods of prevention for people living with mental illness is more important now than ever before. Together, we will continue to fund innovative and impactful research that will lead to new mental health breakthroughs.

Sincerely,

Jeffrey Borenstein, M.D.
President & CEO



Judith M. Ford, Ph.D.
President, Scientific Council

Geoffrey Simon
Chair, Board of Directors

2024 **LEADING RESEARCH ACHIEVEMENTS** BY FOUNDATION GRANTEES, PRIZE WINNERS, & SCIENTIFIC COUNCIL MEMBERS

Suicide Risk Fluctuates Across the Menstrual Cycle, Affecting Different Women Differently

Basic Research; Diagnostic Tools/Early Intervention: Suicide Prevention

Story Highlights:

A new study reveals how suicide risk fluctuates not only in correspondence with the menstrual cycle and shifting hormone levels, but also how symptoms fluctuate daily within individuals and how impacts vary from person to person. Increased depressive symptoms accounted for perimenstrual worsening of suicidal ideation and planning, suggesting a focus for future intervention efforts.

Journal:
American Journal of Psychiatry
January 1, 2024

Women and men are known to have different vulnerabilities to psychiatric and other illnesses. There are a variety of reasons, including biological differences between the sexes and a vast number of socio-demographic and cultural factors.

Thanks in significant part to research by BBRF Scientific Council member, three-time BBRF Distinguished Investigator and Selo Prize winner Myrna M. Weissman, Ph.D., it has been known for several decades that women have a much greater lifetime risk than men of developing depression—as much as a 70 percent greater risk.

One remarkable and related fact that is rarely discussed is that women are three times more likely than men to report suicidal ideation, suicidal planning, or a suicide attempt over the lifespan.

Both of these disparities in risk have some relation to cyclical ovarian hormone fluctuations; the details in each case have been the subject of intense research over many years. Recently, a team of investigators led by 2018 BBRF Young Investigator **Tory Anne Eisenlohr-Moul, Ph.D.,** of the University of Illinois Chicago, published findings of what may be the most detailed study to date seeking to understand how daily changes in a range of psychiatric symptoms influence acute changes in suicidal ideation and planning, and how brain-based sensitivities to normal cycling hormones can shape these changes. Dr. Jacyln Ross and Jordan Barone were co-first authors of the team's paper, which appeared in the *American Journal of Psychiatry*.

Given the greater female vulnerability to suicidal behavior, the team notes, "understanding suicide risk factors that are unique to females is critical to addressing the public health crisis" that suicide represents. There were nearly 50,000 deaths by suicide reported in the U.S. in the most recent year for which statistics are available.

A litany of psychiatric symptoms, noted at single time points, have been strongly correlated with suicide risk. These symptoms include depression, hopelessness, and anhedonia (the inability to experience pleasure), as well as the perception of being a burden to others, heightened sensitivity to rejection, agitation, anxiety, and anger. Although research identifying these correlates has begun to clarify who is most vulnerable to suicidal ideation and suicide attempts across the lifespan, "questions of when and how suicide risk increases remain largely unanswered," the team notes in its paper. A knowledge gap of particular importance concerns short-term risk factors that can help clinicians predict when their patients may transition from thinking about suicide to making an actual plan for suicide, the team stresses. This helped

Tory A. Eisenlohr-Moul, Ph.D.

University of Illinois, Chicago
2018 BBRF Young Investigator

to guide the design of their study.

Risk factors that vary over time, "beyond the usual suspects of psychopathology and sociodemographic variables," were their focus, and specifically, the monthly menstrual cycle. "Accumulating evidence shows that ovarian hormone fluctuations modulate imminent suicide risk for many females, resulting in increased likelihood of suicide attempts and deaths during the premenstrual and menstrual weeks." But studies to date have tended to involve small numbers of patients, have not distinguished chronic suicidal ideation from acute suicidal planning, and haven't taken into account that people differ widely in their response to the monthly cycle. Patients with the cycle-linked mood disorder premenstrual dysphoric disorder (PMDD), who often report symptoms such as irritability and rage, have been found to have very high rates of suicidal behavior. According to another paper published by the team, about 72% of women with PMDD report suicidal thinking and about one-third report a history of suicide attempt.

The team recruited 119 psychiatric outpatients who experienced regular periods and reported past-month suicidal ideation. These participants provided a series of daily ratings of psychiatric symptoms across at least one menstrual cycle.

Nearly all of the psychiatric symptoms varied in correspondence with fluctuations (up or down) in daily suicidal ideation. A limited set of symptoms—depression, hopelessness, rejection sensitivity, and the perception of being a burden—were found to specifically predict an individual's increases in suicidal planning. Of note, individuals also differed from one another with respect to which psychiatric symptoms were the best predictors of daily suicide risk, indicating a need for personalized approaches in the future, the team said.

It was observed that many patients demonstrated worsening of psychiatric symptoms, suicidal ideation, and suicidal planning over the "perimenstrual" period, i.e., around the time leading up to and during menstruation. Depressive symptoms (depression, hopelessness, perception of being a burden, and anhedonia) "were the most robust statistical factors predicting perimenstrual exacerbation of suicidality," the team reported.

"While most past suicide research has highlighted broad psychopathological and sociodemographic factors as predictors of suicide risk, our study reveals an acute perimenstrual risk period for suicidal ideation and planning," the team wrote. This, they said, "presents an opportunity for biological and psychosocial intervention" among those particularly at risk. By measuring fluctuations in suicidality over time, the research may help caregivers formulate predictions about when specific individuals are most at risk.

"We urge researchers to model individual symptom trajectories," the team wrote, to better identify subtypes of neurobehavioral hormone sensitivity. The study's finding that depressive symptoms are robust predictors of perimenstrual worsening of suicidal ideation and planning suggests to the team that "intervention efforts may benefit from strategies that specifically target depressive symptoms as they fluctuate across the cycle, given their powerful role in the cycle-suicidality relationship."

Preliminary Trial of Psychoactive Drug Ibogaine Yields 'Initial Evidence' for Powerful Therapeutic Potential

Next-Generation Therapies: PTSD, Depression, Anxiety

Story Highlights:

In a preliminary clinical test in military veterans, researchers obtained "initial evidence" suggesting the psychoactive compound ibogaine, when co-administered with magnesium, "could be a powerful therapeutic" to safely treat a variety of psychiatric symptoms, including PTSD, depression, anxiety, and suicidality, which can emerge following traumatic brain injury.

Journal:
Nature Medicine
January 5, 2024

In an exploratory and preliminary clinical test, a team of researchers at Stanford University has obtained "initial evidence" suggesting that a psychoactive compound called ibogaine, when co-administered with magnesium, "could be a powerful therapeutic" to safely treat a variety of psychiatric symptoms, including PTSD, major depression and anxiety, and suicidality, all of which may emerge following traumatic brain injury (TBI).

Ibogaine, derived from the root bark of a shrub, has been used for traditional religious and healing purposes in Africa for centuries. Sometimes called an atypical psychedelic, the Stanford researchers prefer to classify it as an "oneirogen," based on a Greek word that describes its main psychotropic effect: therapeutic dosing leads to dreamlike states of consciousness that persist for several hours and sometimes even longer. Proponents of the compound say it facilitates self-reflection and self-evaluation. These are qualities that in recent years have been attributed to psychedelic compounds such as MDMA, psilocybin, and LSD. Like those agents, ibogaine since 1970 has been listed by the U.S. Drug Enforcement Administration as a Schedule I compound, with no officially recognized medical use and with a "high potential for misuse."

Subsequent experience suggests that ibogaine is not addictive, although it does have powerful and potentially harmful effects on users. Until now little academic research has been conducted into its possible therapeutic value. The paucity of research is directly due to its illegality in the U.S., although the drug can be used legally in Mexico and Canada.

Ibogaine is a drug of interest to some researchers for several reasons, the chief of which is that those who suffer from traumatic injuries to the brain are often not helped, or helped only partly, by existing FDA-approved therapies. TBI is considered the "signature injury of U.S. military veterans from recent military conflicts, most often caused by blast exposure," note the authors of the paper reporting results of a small trial with ibogaine just published in the journal *Nature Medicine*. Lead author of the study was **Nolan R. Williams, M.D.**, of Stanford, winner of BBRF's Colvin Prize in 2024 and its Klerman Prize in 2019, and a BBRF Young Investigator in 2018 and 2016. **Jennifer Keller, Ph.D.**, a 2009 BBRF Young Investigator, was among the co-authors.

First-line therapies for conditions often arising following TBI—cognitive rehabilitation, psychotherapy, and medications that target specific symptoms—tend to be less effective for veterans compared with other populations, the Stanford team says, with remission rates ranging from 20% to 40% following treatment. "Most concerningly, veterans make up 20% of U.S. suicides," they note, although they account for only 6% of the population.

Nolan R. Williams, M.D.

Stanford University
2024 BBRF Colvin Prize
2019 BBRF Klerman Prize
2018, 2016 BBRF Young Investigator

The Stanford researchers entered into a collaboration with a company called Ambio Life Sciences, which had received a grant from a nonprofit called Veterans Exploring Treatment Solutions (VETS), Inc. to test ibogaine in a group of 30 male volunteers who, independently of the university, had enrolled themselves in what is called an open-label trial to be conducted at a clinic in Tijuana, Mexico.

All of the participants were males who had served in U.S. special forces and had suffered mild to moderate traumatic brain injury which impaired their functioning.

Prior to treatment with ibogaine the 30 participants were found to have an average rating of 30 on a WHO disability scale, which translates into "mild to moderate" disability. "These men were incredibly intelligent, high-performing individuals who experienced life-altering functional disability from TBI during their time in combat," Dr. Williams said. "They were all willing to try most anything that they thought might help them get their lives back."

After being administered ibogaine along with concurrent injections of magnesium to reduce the potential impact on heart arrhythmia and other known potential cardiac side effects, "participants showed a remarkable reduction in symptoms" of disability, PTSD, depression and anxiety," the team reported. The benefits were sustained at the 1-month follow-up. In fact, disability measures "continued to improve and psychiatric symptom remission and response rates remained high" after 1 month, the team noted. "Neuropsychological testing revealed areas of improvement after treatment particularly in processing speed and executive function, without any detrimental changes observed. With regard to safety, no serious or unexpected adverse events occurred and management of adverse effects was uncomplicated." Such adverse effects included nausea, headache, and anxiety. Motor effects were uncommon and resolved within 24 hours.

The disability score of a typical participant declined from 30 to 19.9 in the assessment immediately following treatment; this further plunged to 5.1 after one month ("no disability"). Mean percentage reduction in PTSD, depression and anxiety symptoms was 81%; 93% were "responders" and the remission rate was 83%. Suicidal ideation, present in 47% pre-treatment, fell to 0% just after treatment and 7% after one month. The tests of neurocognitive functioning included a finding that reaction time slowed significantly post-treatment, which may translate into a reduction in impulsivity among those in whom this was a problem. Impulsivity is associated with relapse after substance-use treatment and also with suicidality.

An important factor in considering the strong results is the potential impact of "complementary therapies" which all participants received as part of the trial in Tijuana. Each participant was paired with a licensed therapist experienced in coaching patients undergoing ibogaine treatment. Each was coached prior to the administration of the treatment, during the treatment while the dream-like effects of ibogaine were being experienced, and following treatment—the next day, when coaches and patients discussed the processing of their emotions and how to interpret and "integrate" insights they may have gained from the treatment into their everyday lives.

Further study of the data from the brain scans made before and after the treatments will lead to additional papers by the team. Dr. Williams hopes that results of subsequent studies with more diverse patient populations including those with more severe TBI, will reveal "a host of different brain areas" that may be involved in any therapeutic impact of ibogaine—data that might "help us to treat other forms of PTSD, anxiety, and depression that aren't necessarily linked to TBI."

In Childhood Anxiety, CBT Helps By Normalizing Hyperactive Brain Circuits, Study Finds

Basic Research: Anxiety

Story Highlights:

A study of brain network changes in young people with anxiety disorders treated with CBT suggests how the therapy helps normalize some but not all circuit irregularities. The latter might be separately targeted to further reduce symptoms.

Journal:
American Journal of Psychiatry
January 24, 2024

A new study of brain network and circuit responses in young people with anxiety disorders who were treated with a course of cognitive behavioral therapy (CBT) suggests how the therapy helps to normalize some but not all irregularities that are likely involved in generating anxiety symptoms. This in turn suggests how CBT and possible adjuncts to it might be modified to improve outcomes.

CBT is a first-line treatment for pediatric anxiety disorders, which are highly prevalent and can be impairing if left untreated. The therapy usually involves graded exposures of the patient to stimuli that evoke fear. The premise behind the approach is extinction of fear through restructuring of cognitive responses to fear stimuli. The impact of such therapy, when effective, is an interruption of processes that lead the patient to avoidant behavior and thinking which makes it difficult to carry on normal activities, from schooling to play and socializing.

A research team led by Melissa A. Brotman, Ph.D., Chief of the Neuroscience and Novel Therapeutics Unit at the National Institute of Mental Health (NIMH), sought to discover treatment-related changes in regional activation patterns throughout the brain in young people with anxiety disorders after they were treated for 12 weeks with CBT plus an adjunctive treatment using computerized cognitive training.

First author of the paper reporting the team's results was **Simone P. Haller, D. Phil.**, a 2020 BBRF Young Investigator. Three other BBRF-affiliated researchers were members of the team, including **Ned H. Kalin, M.D.**, of the University of Wisconsin, a BBRF Scientific Council member and editor-in-chief of the *American Journal of Psychiatry*, in which the team's paper appeared.

As the team points out, neurobiological models posit that pathological anxiety "arises from dysregulated cognitive processes and defensive responses." Alterations in functional brain networks are presumed to account for this, including circuitry involved in attention, salience (determining the relative importance of stimuli coming in from the senses), and threat perception.

While CBT is well proven to be effective in many pediatric anxiety patients, it is by no means perfect. "Response rates are variable, leaving a large portion of treated youths with significant symptoms following treatment," the team notes. "The moderate success rate may be due, in part, to limited understanding of the mechanisms" in the brain that are therapeutically modified by CBT.

To address the paucity of research on CBT-related brain mechanisms in young people, the team studied responses in 69 young people, two-thirds female, diagnosed with primary anxiety disorders (generalized, social,

Simone P. Haller, D. Phil

National Institute of Mental Health
2020 BBRF Young Investigator

separation, phobia) who received CBT plus computer-guided cognitive therapy for 12 weeks. These children, who on average were about 12–13 years old and were unmedicated, received functional MRI (fMRI) brain scans prior to beginning treatment, and then again following the full course of treatment. The scans were made while the participants were asked to perform a simple task while emotionally neutral or angry faces were displayed on a computer screen. The results in these children were compared with scans made under the same conditions for a group of 62 demographically matched healthy children. Most of the participants in the anxiety group and many in the comparison group came from families with annual incomes of $90,000 or more.

The team reported several major findings. Among these was that the unmedicated children with anxiety disorders who received CBT differed from heathy children at baseline, when the first fMRI scan was made. Prior to treatment, youths with anxiety disorders showed widespread hyperactivation of neural circuits, consistent with previous research. These treated children differed at baseline in reaction time to stimuli (slower) and in levels of activation (hyperactivated) brain-wide. Second, in children with anxiety, reaction time as well as hyperactivation specifically in the fronto-parietal region reverted to normal levels with CBT treatment. Third, a number of regions in the brain's cortex and in subcortical regions "remained hyperactive in patients after CBT treatment."

Subcortical regions include the brain's limbic circuitry which handles lower-order emotional processing of inputs from sensory systems and includes the amygdala.

Perhaps the most important aspect of the study was the finding that in young patients, some patterns of hyperactivation, including several frontal regions of the brain as well as the right amygdala, did not change with treatment. The team cannot be certain why this is the case, but theorized that while cognitive aspects of CBT "may first reduce subjective feelings of fear or anxiety, defensive systems may show more persistent dysfunction." They speculate that CBT may more effectively target cortical circuits, while subcortical (including limbic) dysfunction "may lag in responsivity and/or might require more direct interventions" to alter exaggerated defensive reactions.

The team suggested that longer-term follow-up of youths who complete CBT treatment "may reveal normalization of amygdala function," adding to the benefit the therapy may have on cortical circuitry. It is possible, they say, that youths who continue to demonstrate aberrant cortical and subcortical functioning may be especially prone to relapse as time passes. Overall, it makes sense, they suggest, to follow treated youths over time, and also to assess the effectiveness of complementary adjunctive treatments given alongside CBT, particularly those that have a direct impact on subcortical structures of the brain such as the amygdala.

The research team also included: **Rany Abend, Ph.D.**, 2019 BBRF Young Investigator; **Katharina Kircanski, Ph.D.**, 2013 BBRF Young Investigator.

How Immune Activation May Alter the Brain and Cause Depression-Related Behavior During Chronic Social Stress

Basic Research: Depression

Story Highlights:

A protein called MMP8, released during chronic social stress by circulating immune cells, can invade the brain and alter the shape of the space between neurons, research shows. This can adversely affect the nucleus accumbens and possibly other brain areas. In mouse experiments, such changes were causally linked with adverse changes in behavior such as social avoidance.

Journal:
Nature
February 7, 2024

Extensive research over several decades, some of it led by investigators supported by BBRF, has demonstrated that stress, including psychosocial stress (stress that arises from social interactions) is one of the most important risk factors for depression.

Great effort has therefore been devoted to research seeking to reveal precisely how stress perturbs the biology of the brain and other bodily systems to produce the diverse range of depression symptoms.

A new study appearing in the journal *Nature* reports on experiments that add an important new dimension to what is known about how, when someone is experiencing social stress, the functioning of the body's immune system may be involved in helping to cause depression or increase its risk.

The new study was led by 2020 BBRF Young Investigator **Flurin Cathomas, M.D.**, of the Icahn School of Medicine at Mount Sinai; the senior author of the team's paper was **Scott J. Russo, Ph.D.**, a member of BBRF's Scientific Council, a two-time BBRF Young Investigator grantee, and Director of the Brain-Body Research Center at Mount Sinai. Seven other BBRF grantees were involved in this study, including **Eric J. Nestler, M.D., Ph.D.**, a member of BBRF's Scientific Council, a three-time BBRF prize winner, and 1996 Distinguished Investigator.

As authors of the new study note, immune system interactions with the central nervous system (the CNS: the brain and spinal cord) and organs of the body are tightly regulated, biologically. Psychosocial stress can affect the two-way communications between these two elements—the CNS and the rest of the body. In people experiencing chronic stress, past research has shown that the innate immune system is activated, resulting in the mobilization of immune cells including white blood cells in peripheral organs and blood, as well as the production of tiny proteins called cytokines, which can trigger inflammation.

One intriguing discovery in psychiatry research has been that a subset of people with stress-related psychiatric disorders, including major depression, "display a state of chronic low-grade inflammation." Two phenomena associated with such inflammation are an increase in the affected individual's pro-inflammatory cytokines in circulation throughout the body, as well as an increase in white blood cell numbers. In experiments in mice, it has been shown that stress also can induce changes to the thin but vital membrane separating the brain and the rest of the body, the blood-brain barrier (BBB), which normally plays a protective role, keeping toxins and microbes out of the brain. When modified by stress-induced inflammation, the BBB in mice allows the entry of circulating proteins into

Scott J. Russo, Ph.D.

Icahn School of Medicine at Mount Sinai
BBRF Scientific Council
2008, 2006 BBRF Young Investigator

Flurin Cathomas, M.D.

Mount Sinai / Psychiatric University Hospital Zurich
2020 BBRF Young Investigator

the brain that normally can not pass through the barrier. One region in the brain affected by such invasion, in mice, is the nucleus accumbens (NAc), which is central in the processing of rewards and also in the response to aversive stimuli, and implicated in depression.

All of these processes are potentially involved in causing depression symptoms in people, although much remains to be learned about the mechanisms that enable these changes to occur. The new study by Drs. Cathomas, Russo and colleagues reveals new detail about mechanisms through which stress-induced immune changes can affect the function of the brain's neurons and ultimately, behavior.

In the presence of stress, immune factors at work in the body's periphery, such as a rise in the circulation of pro-inflammatory cytokines, likely have a direct impact on neurons—for example, by binding directly to receptors expressed in neurons. The new study demonstrates a distinct way in which stress promotes interactions of immune cells in the periphery with the brain—an indirect way that appears to result in adverse changes in social behavior.

The new research focuses on the role in this process of enzymes called MMPs (matrix metalloproteinases) and in particular MMP8. This enzyme, like others in the MMP family, has roles in shaping and regulating the space between neurons, called extracellular space (ECS), as well as the extracellular matrix (ECM), which is a dense web-like material that individual neurons extend out into ECS.

Experiments by the team in mice and humans leads them to conclude that MMP8, which is released during chronic social stress by immune cells circulating in the body's periphery, can invade the brain perhaps due to damage to the BBB, and alter the shape of ECS and ECM in the brain's nucleus accumbens and possibly other brain areas. In mouse experiments, such changes were causally linked by the team with changes in behavior—changes analogous to those observed when a person experiences chronic social stress (social avoidance, for example).

In short, therefore, the new research provides evidence for a specific mechanism by which the peripheral immune system can affect, via alteration of the ECS, the functioning of neurons in the brain and alter behavior. One potential implication is that it may be possible to develop treatments that target not the brain directly (which is always difficult), but rather molecules such as MMP8 in the peripheral immune system—a totally novel approach to potentially treat psychiatric illnesses including depression.

The researchers note the need for further studies to "more specifically manipulate different components of the ECS in various brain regions," to link them with specific changes in brain physiology and behavioral changes, and also to identify additional factors that might alter the ECS under conditions of chronic stress. A mouse experiment in which MMP8 was depleted resulted in stressed mice that were not socially avoidant; this approach had no impact on non-social behaviors. "Further research is needed to untangle neuroimmune mechanisms of stress-induced vs. non-social behavior alterations," they say. Still, the current results suggest MMP8 or ECS changes may serve as biomarkers for psychiatric illness as well as novel targets for future treatments.

The research team also included: **James W. Murrough, M.D.**, 2009 BBRF Young Investigator; **Eric M. Parise, Ph.D.**, 2021 Young Investigator; **Romain Durand-de Cuttoli, Ph.D.**, 2022 BBRF Young Investigator; **Lyonna F. Parise, Ph.D.**, 2022 BBRF Young Investigator; **Long Li, Ph.D.**, 2021 BBRF Young Investigator; **Kenny L. Chan, Ph.D.**, 2022 BBRF Young Investigator.

Food-Seeking Circuit in the Brain That Can Override Hunger or 'Fullness' Signals May Shed Light on Eating Disorders

Basic Research: Eating Disorders

Story Highlights:

Researchers discovered that a specific group of neurons in a region of the brainstem previously associated with fear, when activated, induces mice to forage and eat, even when they are not hungry—especially rewarding highly caloric foods. The research could provide important insights into eating disorders such as binge eating disorder and anorexia nervosa.

Journal:
Nature Communications
March 7, 2024

Research that was initially focused on fear, anxiety, and defensive behaviors has resulted in a series of unexpected discoveries that have shed new light on eating behaviors, and, possibly, on eating disorders involving both compulsive eating when already "full" as well as aversion to food even when hungry.

In a paper newly appearing in *Nature Communications*, a team led by 2014 BBRF Young Investigator **Avishek Adhikari, Ph.D.**, of UCLA, reports that it has identified a brain circuit in mice whose activation causes the animals to search for food even when they are not hungry. By manipulating the circuit, the researchers demonstrated they could increase or decrease food-seeking in mice, a discovery that might have translational potential in people with eating disorders since the circuit, or one very similar to it, likely also exists in the human brain.

The paper's first author, who designed and performed many of the experiments just reported, was **Fernando M. C. V. Reis, Ph.D.**, also of UCLA. His 2018 BBRF Young Investigator award helped support research that was originally focused on fear memory. A second Young Investigator grant in 2022 funded work that enabled Dr. Reis and colleagues to pursue unexpected discoveries pertaining not just to fear and defensive behaviors but to the brain's food-seeking circuitry as well.

The investigators were studying an area in the brain called the periaqueductal grey, or PAG. This region was known to have an important role in fear, but not in the pursuit of food. The team was initially investigating how cells in the PAG that release the neurotransmitter GABA (called VGAT-expressing neurons) affect fear. In the course of their experiments, the team was surprised to discover that these VGAT-expressing neurons can dramatically alter feeding—their activation led the animals to forage for food and to eat on a full stomach.

The PAG is located in the brainstem, notes Dr. Adhikari, "which is very old in evolutionary history, and because of that, it's functionally similar between humans and mice." While acknowledging the team's findings were a surprise, he said it did "make sense that foraging is rooted in such an ancient part of the brain, since foraging is something all animals must do."

Dr. Adhikari received his graduate and postdoctoral training in the labs of two past BBRF grantees, both of whom are members of BBRF's Scientific Council: Joshua Gordon, M.D., Ph.D., who is director of the National Institute of Mental Health, and Karl Deisseroth, M.D., Ph.D., who in 2021 received the Lasker Basic Medical Research Award for his role in developing optogenetics technology.

Fernando M.C.V. Reis, Ph.D.

University of California, Los Angeles
BBRF Scientific Council
2022, 2018 BBRF Young Investigator

Avishek Adhikari, Ph.D.

University of California, Los Angeles
2014 BBRF Young Investigator

Dr. Adhikari's studies initially focused on how fear and anxiety help animals assess risks and minimize exposure to threats. The PAG is particularly associated with the panic response, in rodents and people. "When we used optogenetics to selectively simulate only this specific group of VGAT-expressing GABA neurons in the PAG, we found it did not affect the animals' fear responses; rather, it caused them to forage and eat," he notes.

The team further observed that when these cells were stimulated, mice that were not hungry started to specifically crave fatty food—so much that they were willing to endure mild electrical shocks to their feet in order to obtain this food. (The harmless but unpleasant shocks are like those one gets when experiencing static electricity.) Stimulation of the cells was not, in other words, inducing feeding by creating hunger, which is an unpleasant feeling; rather, activation was associated with pleasure and reward.

Conversely, when the researchers experimentally suppressed the activity of the same cells in the PAG, mice that were very hungry ate significantly less. This shows, says Dr. Reis, that "this circuit can bypass normal hunger pressures regarding how, what, and when to eat."

This suggests the potential relevance of the results to eating disorders in people. It is almost certain that humans also have VGAT cells in the PAG, as these neurons have been confirmed in a wide range of animals, including rodents, cats, and monkeys.

If additional research confirms that humans also have VGAT-expressing cells in the PAG, researchers can then try to determine if overactivity in the circuit is correlated with the feeling of reward and with craving high-calorie food even when an individual is not hungry. It is also conceivable that if underactive, the same circuit might also help explain reduced pleasure associated with eating, perhaps leading in some people to the avoidance of food. Compulsive eating when not hungry is a behavior seen in binge eating disorder. Avoiding nutrition even when deprived of calories is seen in anorexia nervosa.

In the near-term, there is much more basic research to do. In their paper, the team suggests the need to investigate, for instance, how connections between the VGAT-expressing cells in the PAG and cells in a brain area called the zona incerta may regulate important aspects of the motivation to forage and eat. Circuits controlling appetite and eating behaviors, they say, can perhaps "be viewed as a broad complex loop network" whose distinct functional and computational components "require further characterization."

The research team included **Jonathan C. Kao, Ph.D.**, 2020 BBRF Young Investigator; **Alcino J. Silva, Ph.D.**, a 1999 BBRF Independent Investigator.

A Stem Cell-Based Therapeutic 'Rescue Strategy' is Developed for Timothy Syndrome, an Autism Spectrum Disorder

Next-Generation Therapies: Autism Spectrum Disorders

Story Highlights:

A BBRF grantee has published results of experiments demonstrating a potential therapeutic "rescue strategy" using stem cell technology for a devastating neurodevelopmental disorder called Timothy Syndrome (TS), one of the autism spectrum disorders. By modifying messenger RNA activity for a gene involved in disease pathology, they prevented the emergence of disease pathology in human neurons implanted in living mice.

Journal:
Nature
April 24, 2024

A team of researchers led by two-time BBRF grantee **Sergiu Pasca, M.D.**, of Stanford University, has published results of experiments demonstrating a potential "rescue strategy" using stem cell-based models for a devastating neurodevelopmental disorder called Timothy Syndrome (TS).

TS is widely considered to be among the autism spectrum disorders, with patients often having severely impaired communication and socialization skills, as well as delayed development of speech and language. TS also can have other serious impacts on health including epilepsy and a cardiac disorder called long QT syndrome that affects heart rhythm. The newly reported experiments, while specifically targeting pathology in severe TS, could have future applications in other illness involving the brain including schizophrenia, bipolar disorder, and intellectual disability.

The new research that Dr. Pasca and colleagues report in a cover story in the journal *Nature* has its origins over 15 years ago in the Pasca lab. Dr. Pasca received his first grant from BBRF in 2012 (Young Investigator) and in 2017 he received a BBRF Independent Investigator grant. He is among the pioneering researchers who have harnessed stem-cell technology to grow human brain cells in the laboratory. His lab developed guided neural "organoids" from stem cells and has pioneered the first "assembloids" that model circuit formation in three dimensions in the lab setting. In a more recent innovation, they have transplanted organoids into living animal brains, where they make connections and take part in functional circuits. This has made possible unprecedented experiments to reveal pathologies in human brain illnesses, particularly those like schizophrenia and autism which in some forms likely have origins in the first months of life, during development of the fetal brain and other organ systems. The lab's technologies, deployed in living rodents, are especially valued because we cannot access the human fetus experimentally to observe emerging pathology. The same approach has also provided a unique test-bed for assessing new therapeutics, as the newly reported research demonstrates.

The organoids used by Dr. Pasca and others are based on cells harmlessly sampled from patients; skin cells, for example, can be reprogrammed in the lab to redevelop as cells of the brain, heart, or any organ. Importantly, every reprogrammed cell bears the genome of the patient-donor. If the donor has genetic mutations linked with an illness like Timothy Syndrome, then a novel kind of experiment becomes possible. One can watch these cells from their earliest days as they develop and begin to manifest pathologies caused (at least in part) by their illness-related variant genes.

As reported last year in BBRF's magazine *Brain & Behavior*, Dr. Pasca's

Sergiu P. Pasca, M.D.

Stanford University
2017 BBRF Independent Investigator
2012 BBRF Young Investigator

team engrafted cortical organoids derived from cells donated by patients with a severe kind of Timothy Syndrome called type 1 (TS1). These organoids developed in the lab, and after transplantation into a living rodent, integrated with the host brain in ways that clearly revealed pathologies consistent with the illness. This provided key insights that led to the dramatic experiments just reported.

It began to be clear even from earlier experiments in test tubes performed more than a decade ago by Dr. Pasca that neurons grown from cells donated by TS patients displayed certain characteristic pathologies. For example, these cells had problems regulating the flow of calcium into and out of neurons, a flaw that is associated with abnormally high levels of neural excitation. There were also pathologies affecting the way neurons migrate in the brain.

These defects are caused by a mutation in a gene called *CACNA1C*, known to be mutated in TS (and several other psychiatric illnesses) and, much more specifically, the way in which the *CACNA1C* gene is processed in cells to ultimately give rise to *CACNA1C* proteins.

Every gene in our bodies, when activated, generates a "message" in the form of RNA ("messenger RNA") that tells a cell to manufacture a specific protein. Under normal conditions, the *CACNA1C* gene produces several variants, or "alternate" messenger RNAs (mRNAs). These alternate messages are the result of a process called "alternate mRNA splicing" that occurs just before the message is sent to protein production factories in cells called ribosomes. These messages contain instructions for making the *CACNA1C* protein. The variant of *CACNA1C* carrying the TS mutation is present early in the developing brain and patient-derived neurons seem to make even more of it than those from healthy controls.

As development progresses, the *CACNA1C* RNA "message" normally transitions to a slightly different, more mature form. Dr Pasca reasoned that interfering with RNA splicing to yield the more mature non-mutated form might prevent defects associated with Timothy Syndrome type 1.

To achieve this, his lab developed a therapeutic strategy that has been used successfully to treat several other serious illnesses including spinal muscular atrophy and Duchenne muscular dystrophy. Both also have pathologies rooted in splicing variations in RNA messages. As was done in those illnesses, the researchers created chemically modified pieces of RNA called antisense oligonucleotides (ASOs). The ASOs act like missiles within cells, homing in precisely on specific spots in pre-spliced RNA messages of a gene, causing the splicing of the message to be slightly modified. This modification can do a variety of things depending on the ASO's design. In the case of Dr. Pasca's team, the ASO was designed to interfere with the splicing of *CACNA1C* favoring the variation not carrying the TS1 mutation.

In rats that had received cortical organoid transplants grown from the cells of TS patients, the team injected the tiny ASO molecules into the fluid that bathes the spinal cord and brain. These did indeed alter the splicing of RNA messages in the human cells growing inside the rodent brains, resulting in "robust" reversal, or "rescue" of pathologies in the neurons caused by the mutation—those involving calcium flow as well as migration.

The proof-of-concept experiment was successful, but much more work needs to follow before ASOs can be considered for treating people with Timothy Syndrome. It is not yet clear what impact, if any, the therapeutic strategy will have on cardiac symptoms in TS; tests with cardiac organoids grown from patients' cells are a near-term priority. It is not clear what impact the treatments will have on pathology that happens to predate the treatment. Also, long-term tests must be performed in experimental animals to evaluate toxicities potentially related to the treatment, as well as to determine if the design of the ASOs can be optimized to generate better therapeutic results.

Yet the strategy is an important and promising one, the team believes, and illustrates how this platform involving stem cells and organoids could be used to study other neuropsychiatric diseases and to evaluate the therapeutic efficiency and safety of ASOs and other approaches including small molecule candidate drugs. The approach will be particularly relevant, the team says, when animal models are not available or do not fully recapitulate human pathophysiology for a given illness.

New First-in-Class Schizophrenia Medicine Reduced Positive and Negative Symptoms in Decisive Phase 3 Trials

Next-Generation Therapies: Schizophrenia

Story Highlights:

KarXT, a new medicine for treating schizophrenia—one that appears to help reduce both positive and negative symptoms of the illness—has passed a first hurdle in phase 3 clinical testing. If ultimately approved, it would be the first antipsychotic treatment that does not target the D2 dopamine receptor.

Journal:
JAMA Psychiatry,
May 1, 2024

The Lancet,
January 13, 2024

*****KarXt, marketed under the trade name Cobenfy, was approved by the FDA in September, 2024.

A new medicine for treating schizophrenia—one that appears to help reduce both positive and negative symptoms of the illness—has passed a first hurdle in phase 3 clinical testing. Phase 3 is often pivotal in deciding whether a medicine is effective and safe enough to obtain FDA approval.*****

The medicine, xanomeline-trospium, is called KarXT by Karuna Therapeutics, the company that is developing it and which paid for the initial phase 3 trial. The drug has a novel mechanism of action that distinguishes it from all previously approved antipsychotic medicines.

In December 2023, the pharmaceutical giant Bristol-Myers Squibb Co. entered into a deal valued at over $14 billion to purchase Boston-based Karuna. The announcement came just weeks after positive results of the first of two positive KarXT phase 3 trials were published in the journal *Lancet*. Senior author of the paper reporting the results was **Steven M. Paul, M.D.**, a BBRF Scientific Council emeritus member who is currently Chief Scientific Officer and President of R&D at Karuna. One of the paper's co-authors was 2007 BBRF Young Investigator **Christoph U. Correll, M.D.** of Northwell/Zucker Hillside Hospital.

252 patients with acute psychosis requiring hospitalization were enrolled in the randomized, double-blinded, placebo-controlled trial. Half received KarXT for 5 weeks and half received placebo. KarXT was observed to significantly reduce both "positive" and "negative" symptoms of schizophrenia compared with placebo. In addition to reductions in both kinds of symptoms, patients receiving the new medicine in most cases were able to tolerate it well, reporting only moderate side effects. Larger and longer phase 3 clinical trials are now underway.

KarXT is the culmination of research begun decades ago to find a new way of treating symptoms of schizophrenia. Since the first antipsychotic medicine approved in the 1950s, every antipsychotic approved to date targets a cellular receptor for the neurotransmitter dopamine called the D2 receptor. Some "atypical" or second-generation antipsychotic medicines, including clozapine, also have important therapeutic effects related to their impact on receptors for serotonin. Both first- and second-generation antipsychotics are often very effective in reducing delusions and hallucinations that are the chief positive symptoms of the illness. But they have essentially no impact on negative symptoms such as blunted affect, anhedonia, lack of motivation and asociality, and no appreciable impact on cognitive symptoms.

The idea that led to KarXT began with the aim of developing a drug with a novel mechanism of action—one that would not block D2 dopamine receptors but rather would stimulate cellular receptors called the M1 and

Steven M. Paul, M.D.
(Lancet and JAMA Psychiatry papers)

BBRF Scientific Council Emeritus

Carol A. Tamminga, M.D.
(JAMA Psychiatry paper)

BBRF Scientific Council
2011 BBRF Lieber Prize;
2010, 1998 BBRF Distinguished Investigator

M4 muscarinic receptors. These receptors are part of the cholinergic (acetylcholine) neurotransmitter system. The theory was that agents targeting the muscarinic acetylcholine receptors might indirectly impact the balance in the brain between the dopamine and acetylcholine systems, including in the brain's striatum, which in turn might help therapeutically address pathology that gives rise to psychosis.

For many years, preliminary tests of medicines targeting the muscarinic M1 and M4 receptors suggested that they had excellent potential for reducing schizophrenia's positive, psychosis-related symptoms. The problem has always been side effects: the early candidate drugs had significant side effects in the body's gastrointestinal system, including nausea and vomiting. To potentially overcome this obstacle, developers of KarXT have tested the idea of combining a compound (xanomeline) that stimulates the M1 and M4 muscarinic receptors in the brain with a compound (trospium chloride) that blocks the M1 and M4 receptors in bodily tissue outside the brain, including the gastrointestinal tract. In phase 1 and 2 trials, KarXT appeared to demonstrate antipsychotic efficacy while reducing the frequency and severity of gastrointestinal side effects.

The 252 individuals recruited for the phase 3 trial just reported were drawn from 22 inpatient sites in the U.S.; all had experienced a recent worsening of psychosis warranting hospital admission. The average participant was about 46 years old; about three-fourths were Black. On a scale of symptom severity (called PANSS, the Positive and Negative Symptom Scale), the average participant scored close to 100 (the scale ranges from 30 to 210). Participants went through a "wash-out" period from prior antipsychotic medications that lasted in most cases for one week. Then, participants were randomly assigned to receive KarXT or placebo twice a day for 5 weeks. Those receiving KarXT were started on a dose of 50mg of xanomeline and 20mg of trospium twice daily for 2 days, then 100mg and 20mg of the two drugs, respectively, from days 3-7. Beginning on the 8th day, dosing was flexible, and increased to 125mg/30mg twice daily if tolerated by the patient, otherwise the dose was reduced to the 100mg/20mg level. Nearly all participants were able to tolerate the maximum dose after day 8 for the duration of the trial.

After 5 weeks, those in the KarXT group had significant reductions in both positive and negative schizophrenia symptoms, as measured by the PANSS assessment tool. From an average total symptom score of about 98, the typical participant had a 21-point reduction after 5 weeks on KarXT, compared with an 11.6-point reduction in the placebo group. On separate "subscales" measuring positive or negative symptoms, KarXT was also superior to placebo. KarXT-treated patients had a reduction of nearly 7 points compared with about 4 points in the placebo group. Negative symptoms declined 3.4 points in the KarXT group vs. 1.6 points in the placebo group. In the KarXT group, 55% had an overall symptom reduction of 30% or greater, compared with 28% in the placebo group.

Side effects were present, as they are with virtually all medications, but were considered comparatively mild to moderate by the researchers. Noting KarXT's apparent ability to significantly reduce both positive and negative schizophrenia symptoms as well as the reports on side effects, the researchers concluded that KarXT "has the potential to be the first of a new class of effective and well-tolerated antipsychotic medicines."

The team looked forward to results from additional clinical trials now in progress. Among other things, these may help assess whether the observed decline in negative symptoms in the KarXT patients was a direct result of the medicine's mechanism of action or might be related in part to its ability to reduce positive symptoms. Also, trials in progress will explore over the longer-term the drug's impact as adjunctive treatment on patients with ongoing positive symptoms due to only a partial response to currently available antipsychotics.

Study of One Psychedelic Drug Suggests How It Might be Modified to Eliminate Psychedelic Effects While Retaining Therapeutic Ones

Basic Research; Next-Generation Therapies: PTSD, Anxiety, Depression

Story Highlights:

A detailed study of how the psychedelic drug 5-MeO-DMT interacts with receptors for the neurotransmitter serotonin has suggested that a modified version of the drug that targets the serotonin 1A receptor may not generate psychedelic effects while preserving some of the potential therapeutic effects attributed in various studies to psychedelics in depression, PTSD, and other illnesses.

Journal:
Nature
May 8, 2024

Among those studying psychedelic drugs to treat psychiatric illnesses, there is new research exploring the possibility that structurally modified versions of these agents may reduce or eliminate their powerful mind-altering psychedelic effects while preserving some or all of their potential therapeutic effects.

Reporting in the journal *Nature*, a team co-led by Daniel Wacker, Ph.D., of the Icahn School of Medicine at Mount Sinai, and Dalibor Sames, Ph.D., of Columbia University, used a powerful kind of microscopy called cryogenic election microscopy (cryo-EM) in concert with medicinal chemistry and experiments in living mice to study with great precision at the level of molecules and individual atoms how various psychedelics and approved drugs interact with two kinds of cellular receptors for serotonin.

Lyonna F. Parise, Ph.D., a 2022 BBRF Young Investigator, and **Scott J. Russo, Ph.D.**, a member of BBRF's Scientific Council and a 2008 and 2006 BBRF Young Investigator, were members of the research team.

Serotonin is a neurotransmitter that is ubiquitous in the brain. It engages with individual cells at a variety of receptors, some located on the surface of cells, others within them. The "family" of serotonin-specific receptors includes types 1A and 2A, which were the focus of the new study. The serotonin 2A receptor is an important receptor for excitatory neurons in the brain, and also happens to be the primary target for several psychedelic drugs, including LSD and psilocybin (the active ingredient in "magic mushrooms"). Those drugs are called "agonists" of the 2A serotonin receptor—they bind to it and activate intracellular signaling.

It is widely believed that LSD, psilocybin, and other psychedelics called tryptamine hallucinogens exert both their hallucinogenic and therapeutic effects when they bind at the serotonin 2A receptor. But those and some other psychedelic compounds also engage with a variety of other receptors, including the serotonin 1A receptor. One such psychedelic is 5-MeO-DMT (sometimes called "five methoxy," "bufo," or "toad venom"), a toxin found in the glands of a toad found along the Colorado River. It's very similar to the powerful psychedelic DMT (the active ingredient in ayahuasca), and like it and others, is being considered for possible use in certain psychiatric conditions. A small study conducted recently in Mexico with U.S. Special Forces Veterans tested 5-MeO-DMT in concert with the psychedelic ibogaine for relief of acute PTSD and depression. Both are Schedule I substances banned for human use in the U.S.

As noted by the researchers, the type 1A serotonin receptor is a validated target of several FDA-approved drugs, including anti-anxiety and anti-depressant agents (buspirone

Lyonna F. Parise, Ph.D.

Icahn School of Medicine at Mount Sinai
2022 BBRF Young Investigator

and vilazodone). Yet, they say, "little is known about how psychedelics engage it, and which of their effects are mediated by this receptor." Hence, their study, which sought to perform a detailed structural and functional exploration of the mechanisms through which several "classical" tryptamine psychedelics as well as 5-MeO-DMT and several prescription drugs bind to and activate the 1A serotonin receptor at the molecular and atomic levels.

In a mouse model of depression, they also tested a compound different but structurally analogous to 5-MeO-DMT that selectively targets the 1A serotonin receptor. One of the implicit questions they sought to shed light on was whether a drug targeting the 1A receptor alone, i.e., that did not engage the 2A receptor, might still generate psychedelic effects, and whether it would still generate therapeutic effects (lowering anxiety and depression) ascribed to some psychedelics that bind primarily at the 2A receptor.

Because the serotonin 2A receptor "is responsible for the visual and other sensory disturbances elicited by classical psychedelics, it is typically assumed that these receptors also mediate therapeutic effects."

But, says the team, "there is currently no clinical evidence to support this hypothesis," while evidence generated in animal studies "is mixed."

The team tested the 5-MeO-DMT analogue compound in mice subjected to a kind of chronic stress called "social defeat," which leads the animals to avoid social interaction and to cease caring about seeking treats (analogous to anhedonia in people). The compound, which other experiments showed was a highly selective agonist of the serotonin type 1A receptor, "rescued" these deficits, the team reported, a finding with "potential implications for the therapeutic effects" of 5-MeO- class compounds in treating human psychiatric illnesses perhaps including depression, anxiety, and PTSD.

Other parts of the study generated data supporting the idea that both the 1A and 2A serotonin receptors are involved in stress-coping mechanisms on both a psychological and cellular level; the role of the 1A receptor in stress resilience; and the previously reported antidepressant effect of drugs that specifically target the 1A receptor in animals.

There was also preliminary evidence that the 5-MeO-DMT-analogue targeting the 1A receptor that was tested in mice "lacked the preclinical indications of classical psychedelic effects [the "head-twitch response" is one such sign in rodents], which suggests that some of these compounds may not be hallucinogenic while retaining therapeutic effects."

The team described how the configuration of tiny three-dimensional spaces in receptors called subpockets—highly specific to type 1A vs. 2A serotonin receptors—"determine both the potency and efficacy" of tryptamine hallucinogens at both receptors. This, they said, "provides a structure-guided framework that enables the development" of tryptamine psychedelic analogues "with finely tuned pharmacological activities and varying degrees of selectivity" for 1A and 2A serotonin receptor binding. Synthesizing and testing such compounds will be the subject of future studies.

Importantly, the team also suggested that FDA-approved medicines buspirone, vilazodone, and the antipsychotic aripiprazole, all of which target the type 1A receptor, engage with it differently than 5-MeO-DMT, generating signaling that is distinct from that produced when the psychedelic docks at the receptor to generate signaling outputs. These differences, as well as engagement of other receptor targets, probably accounts for the different effects of these medicines compared with the 5-MeO-DMT analogue tested in the socially defeated mice, the researchers said.

This research was co-led by Daniel Wacker, Ph.D., of the Icahn School of Medicine at Mount Sinai, and Dalibor Sames, Ph.D., of Columbia University.

Experiments Point to Possible Next-Gen Drug Therapies for Bipolar Disorder, Including for Lithium Non-Responders

Basic Research; Next-Generation Therapies: Bipolar Disorder

Story Highlights:

Researchers used stem cell-based technology to discover two ways to reduce hyperactivity in neurons, a characteristic problem in bipolar disorder (BD). The research suggests activation of Akt, a signaling pathway, and AMPK, a protein complex, could be targets for next-gen therapeutics for BD.

Journal:
The Lancet
June 1, 2024

Long-term use of mood-stabilizing drugs has long been a cornerstone of clinical treatment for bipolar disorder (BD). Lithium, which has been in use longer than any other, can be highly effective. In particular it can prevent or reduce the intensity of episodes of mania. Symptoms of mania include hyperactivity, euphoria or highly elevated mood, rushed speech, poor judgment, reduced need for sleep, aggression, and anger.

But lithium doesn't help every patient; in fact, only about 1 patient in 3 responds to it. Among those who do, an important added benefit is that suicide and overall mortality rates are significantly reduced. But toxicity has been associated with lithium administration over the long-term, and its use has been replaced in some patients with drugs not originally approved to treat bipolar disorder, including anticonvulsants such as valproate and lamotrigine.

Given lithium's well-documented ability to be of great benefit to a sizeable subset of BD patients, researchers have taken great pains to discover why it works for them: how the drug affects the central nervous system, at the level of cells, networks, and circuits. The answer has remained elusive, but new research co-led by BBRF grantees and published in the journal *Lancet* not only sheds light on the drug's mechanism of action, but also points to novel therapeutic approaches for patients, including those who do not respond to lithium.

2022 BBRF Young Investigator **Anouar Khayachi, Ph.D.**, of McGill University, Canada, is first author of the new paper, and part of a Canadian team that includes co-leaders **Guy A. Rouleau, M.D., Ph.D.**, a 2010 BBRF Distinguished Investigator, Austen J. Milnerwood, Ph.D., and **Martin Alda, M.D., FRCPC**, 2020 BBRF Colvin Prize winner and a 2003 and 1999 BBRF Independent Investigator.

Among the many insights about lithium's mechanism of action is a recent finding by members of the Canadian team that in neurons of the mouse cerebral cortex, long-term exposure to lithium decreased the flow of sodium ions, reduced firing rates, and lowered the range of calcium ion levels. Ions are atoms that carry an electric charge; their flow into and out of neurons is one of the ways in which the activation of brain cells is regulated.

In the current research, the team used a stem cell-based technology called iPSC (induced pluripotent stem cell). Cells—in this case, blood cells—are harmlessly sampled from individuals both with the illness under study as well as healthy controls. In the lab, these cells are gown in culture and brought back to a stem cell-like state, then re-programmed to re-develop as neurons. The team grew cultures of this kind from 5 BD patients who were responsive to lithium, 4 who were not responders, and 5 age and sex-matched healthy controls (all participants in the study were male).

Anouar Khayachi, Ph.D.

McGill University
2022 BBRF Young Investigator

The point of such research is to discover processes in cells from patients that differ from those in cells from controls. In this case, the BD patient-derived neurons enabled further comparison—between cells derived from patients who were lithium responders and those who weren't.

One important observation made in prior work by the team as well as other researchers was replicated in this work: all of the patient-derived neurons, regardless of the donors' lithium response status, displayed hyperexcitability. When these cells were treated with lithium over 7 days, the hyperactivity was reversed in cells derived from lithium responders, but, as expected, it was not reversed in cells from lithium non-responders.

The next step was to conduct many different kinds of tests on these three sets of neurons grown in culture dishes. Many things were revealed. First: In cells grown from lithium responders—in which hyperactivity was reversed with lithium treatment—the team noted changes in the ability of positively charged sodium ions to flow into and out of the cells, relative to cells grown from lithium non-responders.

Additional experiments that included analyses of protein activity and gene expression revealed that the potentially therapeutic effect of lithium on neurons derived from lithium responders was associated with a specific intracellular signaling pathway, called the Akt signaling pathway. Neurons (and other cells) regulate their survival and growth, in part, via this important pathway.

Additional experiments demonstrated that a compound that activates the Akt pathway mimicked the effect that lithium has on neurons—it reverses their hyperexcitability—but only in neurons grown from patients who responded to lithium, not in those from non-responders. Among the implications of this discovery is that it may make sense to develop and test Akt pathway activators to treat mania in bipolar patients. If the activity of such an agent was as therapeutically beneficial as that of lithium, and the agent was found to be less toxic or have fewer long-term side effects, it might be considered as a replacement for lithium.

Another key finding from the team's experiments also has therapeutic implications. In all BD patient-derived neurons grown in culture—neurons from both lithium responders and non-responders—activation of a protein complex called AMPK (AMP-activated protein kinase) reduced heightened neural network activity that seems to be characteristic in BD. AMPK is an energy sensor inside of cells, a major cellular regulator of lipid and glucose metabolism. Targeting AMPK in neurons might be a strategy to address neuropathology in lithium non-responders and responders alike.

One approved AMPK activator, metformin, is taken by millions of people. People with BD have a 2-fold increased risk of type II diabetes, and insulin resistance is present, the team notes, in about 50% of patients, "which might correlate with disease severity/progression." There has been some suggestion that lithium exerts therapeutic effects in BD via its impact on insulin signaling. Akt and AMPK are both also involved in insulin signaling and the development of insulin resistance. One study has found that metformin improved clinical outcomes in BD patients, not only lowering insulin resistance but improving mood symptoms as well. This preliminary finding "and the results of our work here support use of AMPK activation for BD," the team said, although, of course, this will remain a hypothesis until considerable additional research is performed.

The team noted the main limitation of their study. Studying cells grown in culture in the lab, even those derived from BD patients, are by definition only suggestive, as they were conducted outside the context of the full human (or any animal) system. Reversal of hyperactivation in neurons derived from patients needs to be tested in living organisms—but at this point, no animal model is available to test it.

Nevertheless, the reported experiments suggest specific alterative strategies for treating BD, and provide "a framework for a personalized drug screening platform to accelerate development of alternative therapeutic strategies for BD," the team wrote. Personalized medicine for BD, if realized, could potentially address the frequent lag between diagnosis and therapy selection, as well as significantly reduce the risk of suicide, they said.

Team Develops an Innovative, Implantable Ultrasound Device to Stimulate Neurons in Deep-Brain Regions

New Technologies: Deep-Brain Stimulation

Story Highlights:

Researchers developed and tested a tiny, implantable neurostimulation device that uses ultrasound to modify the activity of neurons deep in the brain. The neural stimulator, called ImPULS, could become "a potent neuromodulatory tool" for therapeutic applications ranging from major depression to Alzheimer's.

Journal:
Nature Communications
June 4, 2024

A research team led by 2018 BBRF Young Investigator **Canan Dağdeviren, Ph.D.**, of the Massachusetts Institute of Technology, reports in *Nature Communications* that it has designed, developed, and successfully tested a tiny, implantable neurostimulation device that uses ultrasound to modify the activity of neurons deep in the brain.

Although still very much an experimental device, their neural stimulator, called ImPULS, in the team's view has promise to become "a potent neuromodulatory tool" for therapeutic applications in people in illnesses ranging from major depression to Alzheimer's. It may also prove useful in basic research on the brain.

ImPULS stands for "implantable piezoelectric ultrasound stimulator." Ultrasound consists of sound waves that vibrate at greater than 20,000 cycles per second (20 kHz), a frequency that is very close to the upper limit of human detection. Many animals can hear in ultrasound wavelengths—from dogs and cats at the lower end to dolphins at the higher end. Ultrasonic waves with much higher frequencies are used for a wide range of medical applications, perhaps the most familiar being the visualization of the human fetus during pregnancy.

ImPULS is not the first device that uses ultrasound to stimulate the brain and alter the activity of neurons. Ultrasound has also been used, so far on a limited basis, to stimulate the brain non-invasively. In transcranial-focused ultrasound (tFUS) treatments in depression, Alzheimer's and epilepsy, low-intensity ultrasonic waves are transmitted through the skull. Unlike the most common form of non-invasive neurostimulation, transcranial magnetic stimulation (TMS), which uses magnetism rather than sound waves to alter neuronal activity, tFUS has the advantage of being able to reach much deeper into the brain. It is thought ultrasound exerts its effects by affecting the tiny pores called ion channels that regulate the electrical activity of neurons. tFUS beams can be precisely focused (on the scale of millimeters), and penetrate several centimeters into regions far "beneath" the brain's cortex, which lies immediately below the skull. Structures in the deeper subcortical regions include those such as the hippocampus and amygdala that play a central role in mood, memory, and learning.

Yet, as the MIT-led research team notes, "ultrasound, when transmitted from outside the human skull, faces significant scattering and reflection." This can cause the stimulation of brain areas beyond the therapeutic target(s), and in some cases can potentially cause damage to the brain. These unintended "off-target" impacts are among the chief motivations for the MIT team's work. Dr. Dagdeviren's 2018 Young Investigator grant supported her work on developing a

Canan Dagdeviren, Ph.D.

Massachusetts Institute of Technology
2018 BBRF Young Investigator

new, implantable interface that could precisely target areas of the brain known to be involved in Parkinson's disease. The current project is related to that effort, in that it also seeks to develop and test a device that can be surgically implanted in the brain to deliver ultrasound with a specificity and precision that exceeds what is possible in tFUS and other non-invasive ultrasound applications.

Implantable technology to deliver neurostimulation is also not a new idea. Helen S. Mayberg, M.D., a BBRF Scientific Council member, prizewinner, and 3-time grantee, pioneered DBS, or deep-brain stimulation, in the 1990s, to treat refractory depression. DBS involves the surgical implantation of electrodes and a device to supply power for stimulatory pulses. These have had dramatic and enduring results in a limited number of patients. Optogenetics, a technology pioneered by BBRF Scientific Council member and grantee Karl Deisseroth, M.D., Ph.D., uses beams of colored laser light delivered through very thin probes implanted in the brain to alter the activity of specific neurons, but has not been applied in humans in part due to its reliance on genetic manipulation of nerve cells, which is not yet feasible in people.

"A miniaturized, non-genetic platform for localized stimulation is therefore needed to fill the gap for next-generation neural interfaces to reach high standards of safety and longevity," the MIT team says. Some early attempts at making ultrasound devices that fit this description have been proposed, but they may not be suitable for implantation deep in the brain "due to their rigid form factors, material composition, or high power requirements," the researchers say.

ImPULS, the implantable piezoelectric ultrasound device they developed, is innovative in several respects. It has no active electrochemical elements. It is highly miniaturized, engineered at the micron-scale (1000 microns = 1 millimeter), is biocompatible, and uses very little power. Piezoelectricity is the electric charge that accumulates in certain solid materials, such as crystals, certain ceramics, and biological matter, in response to applied mechanical stress. To deliver the ultrasound, ImPULS uses a piezoelectric ceramic material micromachined to be only 30 microns thick, with an active element 100 microns in diameter. It is designed to be implanted in deep-brain regions, where its emission of ultrasound energy alters the behavior of adjacent neurons. In tests, the device resisted significant electrical and mechanical degradation over a period of days and did not raise the temperature of brain tissue during ultrasound generation beyond safe levels.

For the initial tests described in their paper, the ImPULS device was connected to an external printed circuit board via a special cable. The extremely thin probe whose implanted 100 micron-wide tip delivers the ultrasound energy was used in the laboratory to excite neurons in a preserved slice of mouse hippocampal tissue. Then, implanted deep in the brain of an anesthetized mouse, ImPULS was used in the living setting to prompt neurons to express a specific gene called c-Fos. Perhaps most intriguing, ImPULS was used in living mice to stimulate neurons that release dopamine in a part of the brain called the substantia nigra pars compacta. Careful application of ultrasound enabled the team to modulate dopamine release over a specific period of time. In Parkinson's disease, large numbers of dopamine neurons in this region at the back of the brain die or cease to function.

The team says the fabrication process enables them to scale ImPULS devices to target larger areas of the brain, if wanted. In future studies, they seek to gain finer control of neural stimulation and evaluate potentially distinct effects such as excitation vs. inhibition in a variety of cell types, neural circuits and brain regions. The team also hopes to produce versions of the device that can deliver ultrasound carrying greater energy. They will also study the durability of the device, hoping to demonstrate that it can survive a month-long implantation. Still other research will try to adapt the technology for specific basic research projects seeking to better understand brain function.

"We believe this implanted ultrasound stimulation device can be developed into a versatile tool for both basic systems neuroscience research and potential therapeutic applications," the researchers said.

The research team included **Steve Ramirez, Ph.D.**, 2016 BBRF Young Investigator.

Network Connectivity Patterns in High-Risk Pre-Adolescents Correctly Predicted Depression Symptom Onset 2 Years Later

Diagnostic Tools/Early Intervention: Depression

Story Highlights:

Imaging scans from over 1,700 of the 11,000+ children enrolled in an ongoing study of brain development through adolescence revealed connectivity patterns, in the scans of healthy 9- and 10-year olds with parental history of depression, that predicted the onset of depression symptoms only 2 years later.

Journal:
Developmental Cognitive Neuroscience
June 4, 2024

Researchers have used functional brain imaging data to discover biomarkers that have the potential to predict early-onset depression in pre-adolescent youths with a family history of depression.

Dylan G. Gee, Ph.D., a 2015 BBRF Young Investigator at Yale University, led a team that made use of data collected by an important NIMH-funded study called the Adolescent Brain Cognitive Development (ABCD) Study. Between 2016 and 2018, a diverse cohort of 11,878 young people from around the nation were recruited for ABCD, which was set up to track their developing brains from pre-adolescence through the end of adolescence. The brain is extremely plastic during these years, in which both neurobiological and social changes are associated with increased vulnerability to developing psychiatric problems.

Taylor J. Keding, Ph.D., a 2023 BBRF Young Investigator, and **Jutta Joormann, Ph.D.**, a 2006 BBRF Young Investigator, were members of the research team in the new study. Bailey Holt-Gosselin was first author of the team's paper appearing in the journal *Developmental Cognitive Neuroscience*.

Dr. Gee and colleagues used a subset of the vast—and still growing—multi-dimensional ABCD database that included 559 children who had no psychiatric symptoms or history at the time of their enrollment, but did have at least one parent with a history of major depressive disorder. Resting-state functional brain imaging scans of these children, enrolled at ages 9 to 10, were compared with scans made of 1,203 children enrolled at the same age who had no psychiatric history and no parental history of depression. The children in the first group were considered, for purposes of the study, at high familial risk for depression, while those in the second group were considered at low familial risk.

Youths who have a parent with depression have a 3- to 5-fold increased risk for developing depression themselves, the research team noted. This, along with the increased vulnerability experienced by all young people for development of psychopathology, "highlights the pressing need to identify predictive neural markers for development of depression prior to the onset of adolescence, especially among children already at high familial risk," the team said.

Evidence from past studies suggests that familial depression risk manifests itself via atypical development of neural circuits implicated in reward and emotion processing, even in children with no history of depression symptoms in pre-adolescence. But the evidence for children is relatively sparse and it is not clear how atypical circuitry emerges over time or where precisely in key brain regions it appears.

Dylan G. Gee, Ph.D.

Yale University
2015 BBRF Young Investigator

Past studies based on resting-state functional brain scans of high familial risk youths prior to the beginning of adolescence have often lacked comparison data from children with low familial risk. Also, the age range of participants in prior studies has been comparatively large, e.g., ages 8–14 or 8–17 years. Dr. Gee and colleagues made certain to limit the age range in their study to 9-10, with the aim of searching for biomarkers that predicted onset of depression symptoms just 2 years later, at ages 11–12. This near-term outcome data was already in the ABCD database.

"Pre-adolescence is a particularly useful window to identify pre-existing neural vulnerability markers," the team said. Vulnerability markers that may be present prior to the emergence of depression "are valuable because this knowledge can lead to early identification of vulnerable youth," who, if treated promptly, stand to have better outcomes.

Each of the 1,762 youths whose data was examined in the study, like all children enrolled in the ABCD study, completed four 5-minute resting-state fMRI scans when they entered the study. Resting-state fMRI measures connectivity in brain circuits while an individual is not focused on a particular mental task. Dr. Gee and colleagues used a variety of methods to spot functional connectivity patterns involving areas involved in processing reward and emotions: the amygdala, putamen, nucleus accumbens, and caudate regions of the brain.

They were able to identify a number of potentially important patterns in the scans. Specific functional connectivity patterns between the amygdala and striatal regions and visual and sensory-sensorimotor networks were found to be predictive of depression 2 years later.

These patterns—again, seen in scans of preadolescents without a diagnosis of a psychiatric disorder at ages 9 and 10—appeared to the team to be potential biomarkers for depression onset by ages 11–12, particularly for youth with a family history of depression.

"The majority of depression-predictive functional connectivity patterns involved regions within visual and sensory/sensorimotor networks which typically mature earlier in development," the researchers pointed out. "Association networks are still undergoing significant development within the age range of our sample. By contrast, sensorimotor networks may have reached greater relative maturity by this age, and therefore may be more likely to differentially shape future [depression] symptom development as a function of familial risk status." It is possible, they speculated, that as neurodevelopment progresses within association networks, these networks "may begin to be more predictive of future symptoms at older ages (e.g., ages 14–18)."

For this reason, the team suggests future studies with the ABCD data investigate later-developing functional connectivity patterns and their possible relation to development of depression symptoms at older ages. They said that being able to predict symptoms at later time points following the pre-adolescent scan (5 or 6 years after) is an important goal for future research, as is consideration of how environmental factors such as early-life adversity affect risk profiles for the emergence of depression.

In identifying vulnerability factors that can be discerned in children before adolescence begins, the current study is a step toward early treatment and perhaps one day prevention of depressive disorders in vulnerable youth.

Researchers Develop 'Mood Instability' Measures to Re-Think How Best to Care for Bipolar Disorder Patients

Diagnostic Tools/Early Intervention; Next-Generation Therapies: Bipolar Disorder

Story Highlights:

New evidence calls into question the assumption in clinical medicine that periods between low and high mood in bipolar disorder are ones of "normal" mood. The finding of considerable "mood instability" between episodes of depression and mania/hypomania could lead to future efforts to treat such mood fluctuations to improve quality of life for patients.

Journal:
Nature Mental Health
August 8, 2024

Research led by 2022 BBRF Young Investigator **Sarah H. Sperry, Ph.D.**, of the University of Michigan, has led to her team's development of an innovative method for assessing the course of bipolar disorder (BD) in those who have been diagnosed.

The new method, which focuses on assessing mood instability in individual patients—frequently and over an extended period—could result, if it were widely adopted, in what Dr. Sperry and colleagues call "a paradigm shift in monitoring outcomes" in BD.

Accurate diagnosis of bipolar disorder is often delayed, Dr. Sperry and colleagues note in their new paper appearing in *Nature Mental Health*, "and even when diagnosed properly, efficient treatment options remain stagnant." It is standard practice, they explain, to think of BD as a "relapsing disorder," in which distinct episodes of depression, mania, or hypomania alternate with periods of normal mood. The deep lows of depressive episodes stand in contrast to the extreme highs of mania in Bipolar I or the somewhat lesser highs of hypomania in Bipolar II.

"Relapsing" is an important term and point of reference for the research by Dr. Sperry and colleagues. As BD is usually thought of, in between depressive episodes on the one hand, and episodes of either mania or hypomania on the other hand, there are periods of "remission," which are conceived as those periods of time when "euthymic" or normal mood prevails—neither qualifying as depressed nor manic/hypomanic. These periods can be of widely varying duration, long or short, depending on the patient.

But, say Dr. Sperry and colleagues, including senior author on the new paper, **Melvin G. McInnis, M.D., FRCPsych** (a 1999 BBRF Independent Investigator and 1992 Young Investigator), the picture is changing because, in recent years, more and more attention has been devoted to studying people with BD over time—what are called longitudinal studies—and assessing them repeatedly over short time periods. There are now wearable digital technologies that enable monitoring of certain behavioral patterns minute by minute and hour by hour. Assessments in the new study did not employ such technologies, but rather depended on patients' self-assessments every 2 months.

In the study that gave rise to their new "paradigm" for assessing BD, Dr. Sperry and colleagues used data from 603 people collected over 10 years or longer for each individual. These people had been enrolled in the Prechter Longitudinal Study of Bipolar Disorder (PLS-BD), and included 385 people diagnosed with BD, 71 with other or nonaffective diagnoses, and 147 with no history of psychiatric diagnosis and none among first-degree relatives. The participants were

Sarah H. Sperry, Ph.D.

University of Michigan
2022 BBRF Young Investigator

typically in their 30s at study entry and White; about 60% were female.

What made this study distinctive was the repeated monitoring of participants, every 2 months, over 10 or more years. The so-called PROMS (patient-reported outcome measures) included assessment questionnaires for depression, mania/hypomania, anxiety, and overall mental and physical functioning.

The scores generated by all of these questionnaires were used to calculate the variation in each measure for each participant, combining results over rolling 1-year periods, as the study progressed. Advanced statistical calculations enabled the team to establish three broad "thresholds" for assessing the degree of variations in symptoms—labeled "mood (in) stability"—over the entire cohort. Low, moderate, and high instability were identified.

The purpose in designing the study in this way was to test the team's hypothesis about a new way of assessing what each patient with BD experiences over the course of time. Are "remissions" between "episodes" the best way to characterize what happens to most people with BD?

This question has great bearing on what kind of care patients receive. Current gold-standard clinical care of BD aims to reduce symptoms or achieve remission in both depressive and manic/hypomanic episodes, as well as to prevent such episodes from occurring where possible. Dr. Sperry has devoted her young career to investigating whether some or even many BD patients experience important mood shifts—significant "mood instability"—even in those periods usually thought of as "between" episodes. Evidence of such instability would call into question the standard assumption in clinical medicine that the "in-between" periods are ones of essentially "normal" mood, what psychiatrists call euthymia. If there is considerable mood instability "in between" episodes of diagnosable depression and mania/hypomania, how might that affect the care BD patients receive and their outcomes?

With the hypothesis that "alternative ways to measure change in BD [status] based on mood instability are needed," the team did arrive at what they consider a "clinically meaningful instability score that is simple to calculate and easy to interpret." They were also able to identify low, moderate, and high instability thresholds, based on individual scores, and used these classifiers to try to predict how individual patients fared both mentally and physically over time.

First, compared with participants in the study who had psychiatric issues other than BD as well as healthy controls, those participants with a BD diagnosis consistently had higher 1-year rolling "mood instability" scores. The effects were largest with respect to symptoms of depression and mania/hypomania, and smaller but still significant for anxiety. Those with BD, compared with those with other psychiatric issues, had larger instability variations in their scores within the 1-year time horizons, indicating to the team that this specifically reflects the trajectory of BD.

Regarding the power of the scores to predict: those participants with higher rolling 1-year instability scores in the measure of depression symptoms tended, if they were in the moderate- and high-threshold subgroups, to have worse mental and physical health functioning. Other questionnaire-based assessors of variations in individual mood status over rolling one-year periods were useful, in various ways, for predicting other outcomes for BD patients as the months and years of the study passed.

Broadly speaking, the team said, their results "provide guidelines for practical clinical monitoring in the daily patient-care setting, and offer an innovative strategy for outcomes assessments."

The team said it might then be possible for research to tackle the question of how to reduce mood instability—fluctuations over comparatively short periods of time within each patient—as a way of improving their outcomes, which include not only mental and physical functioning, but also feelings of well-being, cognition, interpersonal relationships, and occupational outcomes.

Evidence Grows of the Effectiveness of Rapid-Acting Brain Stimulation to Treat Bipolar Depression

Next-Generation Therapies: Bipolar Disorder

Story Highlights:

New research is providing additional evidence of the effectiveness and safety of SAINT, a rapid-acting non-invasive brain stimulation therapy, to treat people suffering from bipolar depression.

Journals:

Williams et al:
Journal of Affective Disorders,
August 16, 2024
Brain Stimulation,
March 4, 2024

Sheline et al:
JAMA Psychiatry,
July 10, 2024

Published research is providing additional evidence of the effectiveness and safety of rapid-acting non-invasive brain stimulation therapy to treat people suffering from bipolar depression.

Bipolar disorder (BD) involves fluctuations in in mood and energy that can result in severe cognitive and functional impairment. High-energy moods (mania or less intense hypomania) can be treated with lithium or anticonvulsants. Depressive moods are treated with a variety of medications. Although patterns can vary greatly from patient to patient, on average, bipolar disorder patients are in the "depressive phase" of the illness between 70% to 80% of the time, and a significant number do not respond satisfactorily or cannot tolerate drug therapies which include mood stabilizers, antidepressants, and antipsychotic medicines. There is also the issue of testing potential drug therapies one by one to see by trial and error which (if any) are effective. Each trial takes weeks and costs the patient more time.

The emerging evidence regarding use of rapid-acting non-invasive brain stimulation to treat bipolar depression is of particular importance given the greatly elevated risk of suicide among adults with BD (estimated at 10%–15% or higher). The new evidence is about a protocol called SAINT (Stanford Accelerated Intelligent Neuromodulation Therapy), pioneered by BBRF grantee and prize winner **Nolan R. Williams, M.D.**, of Stanford University.

As Dr. Williams and colleagues reported beginning in 2018, SAINT, non-invasively delivering 90,000 magnetic pulses to modify cortical activity over the course of only 5 days, has had remarkable efficacy in small initial trials, enabling a majority of patients with severe, refractory depression to achieve remission in just days. SAINT was approved for commercialization by the FDA for use in depression early in 2024.

SAINT uses an accelerated form of iTBS (aiTBS: accelerated intermittent theta-burst stimulation), which delivers five times as many magnetic pulses to patients than conventional TMS (transcranial magnetic stimulation) in a course of therapy. TMS, pioneered by BBRF Scientific Council member and past grantee Mark S. George, M.D. and others and approved by the FDA in 2009, is usually given five times a week in 38-minute sessions over 6 weeks. In SAINT, over a 5-day course, ten stimulation sessions are given each day separated by about an hour.

SAINT is individually targeted. A resting-state functional MRI scan of the patient's brain made prior to treatment provides an optimized location at which to focus the stimulation. Dr. Williams and colleagues aim the pulses at a spot on the scalp above the left dorsolateral prefrontal cortex (DLPFC)

Nolan R. Williams, M.D.

Stanford University
2024 BBRF Colvin Prize
2019 BBRF Klerman Prize
2018, 2016 BBRF Young Investigator

Yvette I. Sheline, M.D.

University of Pennsylvania
BBRF Scientific Council
2005, 2002 BBRF Independent Investigator
1998 BBRF Young Investigator

that, when stimulated, generates a specific neural response in a brain area much farther below the scalp called the subgenual anterior cingulate cortex (sgACC). Precise targeting, Dr. Williams and colleagues believe, can enhance the therapeutic impact in patients receiving SAINT.

The approach has been highly effective in treating patients with unipolar depression (i.e., patients with a major depression diagnosis, as opposed to BD, which also involves episodes of mania/hypomania).

In July, BBRF Scientific Council member and three-time grantee **Yvette I. Sheline, M.D.**, and colleagues at the University of Pennsylvania, reported on a randomized, blinded aiTBS clinical trial involving 24 patients currently experiencing a depressive bipolar episode that had not responded to prior treatment attempts. That trial generated "a large antidepressant effect" in patients who received the therapy, whose depression scores "were significantly lower" after 5 days of treatment. Five of 12 patients receiving the active treatment had achieved a remission at that point. That trial utilized an identical aiTBS stimulation approach to SAINT and a similar resting-state fMRI targeting approach.

Now, in the *Journal of Affective Disorders*, Dr. Williams and colleagues at Stanford and Johns Hopkins Universities have reported similarly impressive results with SAINT in treatment-resistant bipolar depression. SAINT was not paired in this "open-label" trial with an alternate or placebo treatment; all 10 patients knew the treatment they were receiving. Each patient was carefully monitored for possible "emergent" mania symptoms, which in past conventional rTMS trials had been observed in rare cases.

All of the patients were able to receive all of the planned treatment sessions over the 5 days of the trial. There were no serious adverse side effects including an absence of emergent mania symptoms, and no negative impact on cognition. Importantly, SAINT appeared to work just as powerfully as it had in the past for unipolar depression patients.

The participants, who were rated with moderate to severe bipolar depression that had resisted treatment in the current episode, had an average reduction of about 17 points on a depression scale called MADRS. (The typical participant had entered the trial with a score of about 30.) Immediately following the end of the 5-day course of treatment, half the participants had experienced a response (defined as a reduction in depression symptoms of 50% or more); 40% qualified for remission (in essence, the absence of depression symptoms). The remission rate grew to 60% one month following the completion of treatments.

All of the participants in the SAINT trial were diagnosed with bipolar I depression—they had been diagnosed on the basis of experiences of depression and full mania. Several months earlier, another small open-label SAINT study conducted by Dr. Williams and Stanford and Johns Hopkins colleagues included 2 patients with bipolar I (depression and mania) as wells as 5 patients diagnosed with bipolar II (depression and hypomania—a less intense form of mania). As reported in the journal *Brain Stimulation*, this study also generated impressive results: large decreases in depression scores, remissions in some patients, and an absence of severe side effects, including an absence of emergent hypo/mania.

Having now shown powerful preliminary results (in terms of safety, tolerability, feasibility, and efficacy) with individuals with moderate and severe bipolar depression—patients with both bipolar I and bipolar II diagnoses—Dr. Williams and colleagues in both recent publications say the time is right for much larger, double-blind, placebo-controlled trials for SAINT in bipolar depression, with larger and more diverse patient samples needed to confirm both efficacy and safety.

While there are "a few first-line treatment options for bipolar depression," they noted in one of their recent papers, "none are rapid-acting." SAINT, if one day approved for depression in BD, would address both "the time constraints posed by 6-week conventional rTMS course and the need for fast-acting treatments for acute suicidality in high-risk populations," they noted.

The Sheline team included **Nicholas L. Balderston, Ph.D**., 2021 BBRF Klerman Prize, 2021 and 2018 BBRF Young Investigator; and **Robin Cash, Ph.D.**, 2020 BBRF Young Investigator. Among co-authors of one the Williams papers was **Peter Zandi, Ph.D., MPH, MH**S, a 2004 BBRF Young Investigator.

A Possible Biomarker for Cocaine Misuse and a Novel Treatment for Cocaine Addiction Based on Compound in Rosemary

Diagnostic Tools/Early Intervention; Next-Generation Therapies: Addiction

Story Highlights:

Researchers found that activity of certain neurons in the brain's globus pallidus can predict behavioral responses to cocaine in mice. This potential biomarker also suggests a possible basis for novel treatment of cocaine and other substance abuse: administration of carnosic acid obtained from rosemary extract.

Journal:
Neuron
August 16, 2024

Researchers led by 2017 BBRF Young Investigator **Kevin T. Beier, Ph.D.**, have discovered a way to predict individual behavioral responses to cocaine in mice never exposed to the drug, and have also found that carnosic acid, which is found in extract from the herb rosemary, can reduce volitional cocaine use in mice by reducing activity in a key brain circuit that controls cocaine-induced behavioral changes.

Dr. Beier, of the University of California, Irvine, is among researchers who in recent years have studied the system that regulates release of the neurotransmitter dopamine from a region called the ventral tegmental area (VTA). Dopamine release from cells in this area has been implicated in all phases of substance misuse (not only cocaine), from the initial rewarding effect to withdrawal and ultimately to compulsive drug-seeking.

But as Dr. Beier and colleagues note in their new paper, appearing in the journal *Neuron*, "it is precisely because the dopamine system is central to so many functions that it has proved to be a poor target to combat substance abuse." Given dopamine's ubiquity in the brain, rather than try to regulate the dopamine system as a whole as a way of modifying drug addiction, researchers including Dr. Beier and colleagues have turned to the idea of modulating signaling in particular dopamine subcircuits.

In prior research, Dr. Beier's team demonstrated the role of a key subcircuit centered on dopamine-releasing cells in the VTA that contributed to some of the later stages of substance misuse including withdrawal and reinstatement of use after forced cessation. In the team's new experiments just reported, they sought to map circuits that control the earliest stages of substance use disorder (SUD)—those that mediate drug reward as well as the urge to take the drug.

While most SUD research has focused on several brain regions involved in reward and aversion processing including the VTA, nucleus accumbens, and medial prefrontal cortex, Dr. Beier's team has focused on a less-explored region called the globus pallidus externus (GPe), which appears to play an important role in mediating behavioral changes that occur following use of an addictive drug like cocaine.

The question in the new study was which areas of the brain controlled individual differences in behavioral response to cocaine. While cocaine is an addictive drug, not everyone who uses cocaine develops an SUD; Dr. Beier's team was interested in whether individual differences in behavioral responses to cocaine could be predicted prior to repeated use of cocaine. Through a series of experiments in living mice, the team was able, first, to implicate the GPe

Kevin T. Beier, Ph.D.

University of California Irvine School of Medicine
2017 BBRF Young Investigator

"as the central mediator" in cocaine reward as well as in sensitization to the drug (responding more strongly to each subsequent drug exposure). Beyond this, they were able to show that by dampening the activity of parvalbumin (PV)-containing cells in the GPe, they could reduce volitional cocaine intake in mice. This likely occurred through modulating activity in a subset of dopamine cells in the VTA that critically regulate cocaine reward. PV is a protein whose presence is used to distinguish a particular subset of neurons in the brain.

Importantly, the researchers identified a specific mechanism that appeared to be essential in getting this response: they "dampened" GPe cell activity by activating proteins called KCNQ3 and KCNQ5. These are proteins that help regulate the flow of charged molecules (ions) of potassium into and out of nerve cells. The flow of ions like potassium is one of the essential ways that nerve cells regulate their activity—whether and how often they fire.

Interestingly, the experiments demonstrated that in cocaine-naïve mice, levels of firing activity of PV-containing GPe cells correlated directly with how rewarding a mouse found a subsequent cocaine dose to be. Cocaine-naïve animals with high levels of activity in such cells were more susceptible to long-lasting behavioral effects of cocaine than those with low levels of activity.

This result provided a rationale to test whether artificially lowering the activity level in PV-containing GPe cells would lower the behavioral response to cocaine, including the desire of animals to self-administer it when offered. This proved to be the case. The effect was the same in mice of both sexes and was thought by the team to occur via the blocking or lowering of reward from taking the drug.

There were two important takeaways. One is that measuring the baseline activity of PV-containing cells in the GPe is a potential biomarker for cocaine sensitivity—perhaps in people, as in mice. This is important, says Dr. Beier, because "only a subset of people is vulnerable to developing substance-use disorder, but we cannot yet identify who they are. If globus pallidus cell activity can effectively predict behavioral responses to cocaine, it could serve as a biomarker for the most vulnerable, which could be an important method for reducing dependence and ultimately, substance misuse."

The second major takeaway was that the method used to lower the activity of PV-containing GPe cells—administration of carnosic acid obtained from rosemary extract—is a potential novel treatment for cocaine-use disorder and perhaps for other substance use disorders. As Dr. Beier notes, "there are no effective therapeutics for dependence on psychostimulants like cocaine. Our study deepens our understanding of basic brain mechanisms that increase vulnerability and provides a foundation for development of new interventions."

The team noted: "Carnosic acid has [previously] been reported to exhibit wide-ranging health benefits, demonstrating anti- inflammatory, antiviral, anti-obesity, anti-carcinogenic, and anti-depressive properties, and generally shows promise as a neuroprotective agent, including against Alzheimer's and Parkinson disease. However, to our knowledge, this is the first report of its potential as an anti-addictive agent. As such, we should note that much remains unknown about carnosic acid's effects on the brain, both acutely and long term."

Translation of results in rodents to humans is a major undertaking. The next steps in the research include thoroughly assessing any negative side effects of carnosic acid, and determining optimal dosages and timing of treatments. This would precede any tests of efficacy in people. The team is also interested in testing carnosic acid's effectiveness in reducing the desire for other drugs.

The research team included **Jason Aoto, Ph.D.,** 2016 BBRF Young Investigator.

tDCS Non-Invasive Brain Stimulation Fares Well in Trials for PTSD and Major Depression

Next-Generation Therapies: PTSD, Depression

Story Highlights:

Results of two recently published clinical trials co-led by BBRF grantees demonstrate the versatility and potential effectiveness of transcranial direct current stimulation (tDCS), low-power form of non-invasive brain stimulation, in reducing symptoms of PTSD and major depressive disorder.

Journals:

Fu et al:
Nature Medicine,
October 21, 2024

van 't Wout-Frank et al:
JAMA Psychiatry,
March 6, 2024

Results of two recently published clinical trials demonstrate the versatility and potential effectiveness of transcranial direct current stimulation (tDCS) in reducing symptoms of psychiatric illness, specifically, in PTSD and major depressive disorder. BBRF grantees co-led both trials, which were entirely independent of one another.

tDCS is a form of non-invasive brain stimulation. It is less powerful than the most widely used form of non-invasive stimulation, TMS (transcranial magnetic stimulation) and rapid-acting variants such as SAINT (Stanford Accelerated Intelligent Neuromodulatory Therapy).

tDCS applies a weak direct current (0.5 to 2 milliamperes) to the scalp via two electrodes, typically held in place by rubber headbands or embedded in a cap worn by the patient. The direct current of tDCS is thought to alter the excitability of brain cells beneath the scalp, but does not directly trigger nerve cell firing or inhibition as in TMS. Unlike some forms of iTBS such as the SAINT protocol, tDCS is not rapid-acting.

In *JAMA Psychiatry*, a team co-led by **Noah S. Philip, M.D.**, a 2024 BBRF Distinguished Investigator, and **Mascha van 't Wout-Frank, Ph.D.**, a 2010 BBRF Young Investigator, both of Brown University, reported encouraging results of a double-blinded, randomized, placebo-controlled trial using tDCS to treat U.S. military veterans with warzone-related PTSD.

At the VA Providence (RI) Healthcare System, 54 veterans who had chronic PTSD and warzone-related exposure were recruited for the trial.

These individuals were randomized into two groups: 26 received active tDCS which was delivered during six 25-minute sessions of virtual reality-based exposure therapy sessions given over a period of 2 to 3 weeks. tDCS was delivered at 2 milliamperes via a rubber band over the forehead, with the anode targeting the brain's ventromedial prefrontal cortex (vmPFC). Twenty-eight participants received a placebo ("sham") version of tDCS that was designed to be indistinguishable from active tDCS; no electricity was delivered to the scalp. All 54 participants received the active/sham tDCS while they were receiving a form of virtual reality (VR) exposure that was designed to be immersive yet not based specifically on each participant's traumatic experience(s); rather it was designed to incorporate "broadly shared traumatic experiences."

Active tDCS simultaneously delivered with VR exposure, relative to the "sham" version, did yield "significantly meaningful" reductions in PTSD symptoms, measured by a standard, self-report scale—improvements that were observed to increase over time. Those in the active tDCS group experienced enhanced psychophysiological habituation to VR warzone cues, which the team suggested may indicate that the combined treatment facilitated learning and memory, and helped patients both

Mascha van 't Wout-Frank, Ph.D.

Brown University
2010 BBRF Young Investigator

Cynthia H. Y. Fu, M.D., Ph.D.

King's College London, UK
2006, 2002 BBRF Young Investigator

anticipate their fears and learn how to extinguish them. PTSD symptoms continued to improve over time in the active tDCS treatment group over the month following the end of treatments. The results at 3 months were harder to interpret due to dropouts from the participant group, lowering the statistical power of the numbers. Nonetheless, the active tDCS treatment also showed significant and meaningful improved social and occupational functioning at 3 months. Adverse effects were mild and typical for tDCS—most often, tingling or itching and skin redness under the electrodes.

Given that both parts of the active treatment (tDCS during VR) are inexpensive and "accessible," the team said that if the current results are replicated in much larger groups, this brief tDCS-augmented VR intervention might be widely applied, including perhaps in home-based use.

The second recently reported tDCS trial explored home-based tDCS in the treatment of people diagnosed with major depressive disorder. In a fully remote, phase 2, randomized, placebo-based study, researchers tested a 10-week home-based course of tDCS. Leaders of the study included **Cynthia H. Y. Fu, M.D., Ph.D.**, a 2006 and 2002 BBRF Young Investigator; **Allan H. Young, M.D., Ph.D.**, a 2000 BBRF Independent Investigator; and **Jair Soares, M.D., Ph.D.**, a 2002 BBRF Independent Investigator and 1999 and 1997 Young Investigator.

The team reported results of their trial in *Nature Medicine*. They recruited 174 participants, average age in the late 30s, and equally divided them into two groups. One received 5 self-administered active tDCS treatments per week for 3 weeks, then 3 sessions weekly for 7 weeks. The other group received a sham version of the same treatments. Then, in a second 10-week period, all participants received active tDCS. Each tDCS session lasted 30 minutes. The anode of the device was placed over the location on the scalp corresponding with the brain's left dorsolateral prefrontal cortex (DLPFC); the cathode was placed over right DLPFC. tDCS was delivered at 2 milliamperes, the same strength as in the PTSD trial.

tDCS is amenable to self-administered home use, the team said, since it has proven both portable and safe in many past tests.

The participants in the trial were at least moderately depressed—their average score on a standard scale called HDRS was 19 (below 16 is considered "mild" and above 24 "severe"). About one-third were not receiving current treatment; two-thirds were taking antidepressant medication and about 15% were in psychotherapy.

The group receiving the active tDCS treatment experienced "a significant improvement" in depression symptoms as measured by the HDRS score; the average decrease in score was 9.4 points and the score after 10 weeks was 9.5, on average. Those receiving placebo also improved: an average decrease of about 7 points and a score after week 10 of about 11.6. But the response rate to the active treatment was also significantly greater: nearly 60% for those receiving tDCS compared with about 38% for the placebo group. 45% in the active group had a remission after 10 weeks, compared with about 22% in the placebo group.

The significantly better results in those receiving active tDCS was also reflected in other depression rating scores as well as in a score based on patient self-reports. Meaningful efficacy was seen in both first-episode depressed participants as well as those with chronic and treatment-resistant major depression.

The second 10 weeks of the trial, in which all participants received active treatment, helped to keep as many participants as possible in the trial until the conclusion. While there were no serious adverse effects of active tDCS, active stimulation was associated with higher rates of skin redness, irritation and dry skin compared with sham stimulation.

In the view of the researchers, the 10-week course of tDCS as tested against placebo in this trial is evidence supporting its consideration as a first-line treatment in the future, although under the assumption that any home-based treatments will include ongoing safety monitoring by consulting medical personnel.

A Strategy to Sharply Blunt Addictive Reward From Opioids While Retaining Their Pain-Relieving Properties

Basic Research; Next-Generation Therapies: Addiction

Story Highlights:

Researchers administered the drug JZL184 in mice to raise levels of 2-AG, a naturally occurring neuromodulator in the endocannabinoid system. This greatly reduced reward from opioids but had no impact on opioids' pain-relieving properties, suggesting a possible adjunctive therapy to reduce opioid addiction in pain patients.

Journal:
Science Advances
November 29, 2024

Since the 1990s, deaths due to opioid overdoses have become a leading cause of death for Americans under age 50. While a portion of these deaths are due to recreational use of opioids, many others can be traced to people who became dependent after being prescribed FDA-approved opioid-based drugs, typically for the relief of acute or chronic pain.

Now, a team including BBRF Scientific Council member and three-time BBRF grantee **Francis S. Lee, M.D., Ph.D.**, of Weill Cornell Medicine, and collaborators Anjali Rajadhyaksha, Ph.D., and Arlene Martinez-Rivera, Ph.D., now at Temple University, report finding a way to significantly blunt the rewarding properties of opioids while preserving their analgesic (i.e., pain-relieving) properties. The new research appears in the journal *Science Advances* and could indicate a potentially new therapeutic strategy for opioid-related pain treatments. Four other BBRF grantees were members of the team.

The widespread prevalence of opioid use disorder and the high mortality associated with opioid misuse in connection with medical treatment for pain "has highlighted the urgent need to develop non-opioid analgesic alternatives," write Dr. Lee and colleagues. But "an additional strategy," they note, "is to develop adjunctive treatments that can specifically attenuate the rewarding but not the analgesic properties of opioids."

The team tested its approach in mice undergoing various behavioral tasks tied to opioid rewards. The team's approach was to pharmacologically enhance the brain's endocannabinoid system. This system, which is naturally occurring, helps regulate a wide range of functions in the body ranging from cognition to appetite, mood, memory, and pain, among others. Two endocannabinoid compounds, neuromodulators usually referred to by their chemical abbreviations 2-AG and AEA, interact with cannabinoid receptors called CB1 in cells throughout the body. These receptors are widespread in the central nervous system and play key roles in regulating the release of neurotransmitters such as dopamine.

One key place in the brain where 2-AG and AEA interact with CB1 receptors is in the circuit linking the ventral tegmental area (VTA) and the nucleus accumbens (NAc), which are central to the brain's reward system. This circuit is at the core of the opioid reward response. When a person (or mouse) ingests an opioid, dopamine levels in the NAc increase markedly, an action that has been directly linked with the feeling of reward that occurs after taking the drug.

Much research has been devoted to understanding how the endocannabinoid system works, and how it is perturbed by chronic or acute drug use. Cannabis from plants targets the CB1 receptor. Abuse of

Francis S. Lee, M.D., Ph.D.

Weill Cornell Medicine
BBRF Scientific Council
2010 BBRF Independent Investigator
2005, 2002 BBRF Young Investigator

cannabis is thought to dysregulate the body's endocannabinoid system, with various potential impacts including dependency.

Studies of the interplay between the endocannabinoid system and opioid reward has mainly focused on two enzymes (called catabolic enzymes) that tightly regulate the level of the two endocannabinoid molecules, 2-AG and AEA. One of these enzymes, called MAGL, breaks down 2-AG. Another, called FAAH, breaks down AEA. Previous research has indicated that inhibiting these two enzymes has distinct and sometimes opposing effects on opioid reward, highlighting the complexity of these systems.

Drs. Martinez-Rivera, Rajadhyaksha, and Lee set out to systematically study and clarify the specific impact of raising 2-AG and AEA levels in the brain upon the rewarding effect of opioid intake. Using a MAGL inhibitor called JZL184, the team conducted experiments throughout the mouse system as well as in a specific brain region. They found that raising levels of 2-AG greatly reduced opioid reward in various opioid reward tests, including in mice trained to self-administer oxycodone intravenously. Interestingly, using a different compound to raise levels of the other endocannabinoid, AEA, had no effect on morphine reward.

Further experiments focused on the circuitry known to be involved in reward. When the researchers blocked MAGL in the mouse brain's VTA, they found that the rewarding effects of morphine were reduced. They also observed that a decrease in reward was linked to lower activity of the NAc and lower levels of dopamine.

In short, the experiments demonstrated that 2-AG selectively dampens the rewarding effects of opioids—by disrupting signaling between the VTA and NAc. Equally important, while JZL184 substantially reduced opioid reward, two separate tests demonstrated that it had no effect on the analgesic properties of the opioids tested.

For this reason, the team concluded that their findings "provide a compelling rationale for developing a new class of adjunctive endocannabinoid-based treatments that could dissociate the rewarding and analgesic properties of opioids." In other words, they say, it may be possible to develop a drug similar to JZL184 that can be taken alongside an opioid to treat chronic or acute pain while greatly reducing (or even eliminating) the risk of addiction.

Developing a drug treatment that alleviates pain without the addictive side effects of opioids could be a major step toward addressing one key component of the opioid crisis, the team said. "By targeting the brain's reward system without affecting pain relief, we may be able to create a treatment that provides the benefits of opioids while avoiding their addictive potential--an exciting step forward," said Dr. Rajadhyaksha.

The research team included **Conor Liston, M.D.**, Ph.D., 2013 BBRF Young Investigator; **Virginia M. Pickel, Ph.D.**, 2001 BBRF Distinguished Investigator; **Lisa A. Briand, Ph.D.**, 2015 BBRF Young Investigator; and **Kristen Pleil, Ph.D.**, 2017 BBRF Young Investigator.

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46 Chairs of Psychiatry & Neuroscience Departments
45 Members of the National Academy of Medicine
13 National Institutes of Health Chiefs & Directors
7 Members of the National Academy of Sciences
3 Recipients of the National Medal of Science
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2024 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.

We welcome our newest members.

Cecilia Flores, Ph.D.
McGill University

Erika Forbes, Ph.D.
University of Pittsburgh
2014 Independent Investigator Grantee
2006 Young Investigator Grantee

Mazen Kheirbek, Ph.D.
University of California San Francisco
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2017 Young Investigator Grantee

Daniel Lodge, Ph.D.
University of Texas Health Science Center at San Antonio
2009, 2006 Young Investigator Grantee

Tom Neylan, Ph.D.
University of California S an Francisco
1994 Young Investigator Grantee

Rachel Yehuda, Ph.D.
Icahn School of Medicine at Mount Sinai
2016 Distinguished Investigator Grantee

"Each year, we grow the Council by adding a few new members who are committed to our mission, and importantly, who are expert in the new 'next generation' technologies being proposed and who are actively investigating the neurobiology of serious mental illness."

— Dr. Judith Ford, President of the BBRF Scientific Council

BBRF 2024 GRANTEES

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

BY THE NUMBERS SINCE 1987

As of February 2025

AWARDED TO SCIENTISTS

$462+ MILLION

GRANTS

6,707

GRANTEES

5,605

UNIVERSITIES & MEDICAL CENTERS

605

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. Judith Ford, 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 2024 Distinguished Investigator (DI) Grants

165
APPLICATIONS

10
GRANTS AWARDED

$1MILLION
AWARDED IN DISTINGUISHED INVESTIGATOR GRANTS

The BBRF **Distinguished Investigator Grants** provide support for 10 experienced investigators conducting neurobiological and behavioral research. These grants are funded by the generous support of the WoodNext Foundation, a component fund administered by Greater Houston Community Foundation.

Recipients of the $100,000, one-year grants are awarded to established scientists who are pursuing particularly innovative research projects which may provide new approaches to understanding or treating psychiatric illness. These grants are among the most competitive in mental health research and demonstrate the power of investigator-initiated research to bring out new and creative ideas.

The 2024 DI's are exploring new frontiers in understanding a wide range of neuropsychiatric disorders, including autism, anxiety, bipolar disorder, schizophrenia and the potential connection between mental illness and cannabis use.

"It is wonderful to see the relaunching of this very important component of BBRF's research portfolio. The DI award program serves a unique niche by supporting established investigators to explore high-risk but also high-yield ideas. We are delighted with the slate of DI award winners this year. The new awards were selected from a large group of highly competitive applications and will support exciting and innovative lines of research consistent with BBRF's mission to better understand and ultimately treat severe mental illness."

Eric J. Nestler, M.D., Ph.D.
Chair of the BBRF Distinguished Investigator Grant Committee

2024 BBRF Distinguished Investigator Grants by Illness

One of these grantees is listed under multiple categories as their grant project is relevant to more than one illness.

ADDICTION / SUBSTANCE USE DISORDERS

Karin Johanna Hendrika Verweij, Ph.D.
Basic Research

ANXIETY DISORDERS

Elizabeth A. Phelps, Ph.D.
Next-Generation Therapies

Carmen Sandi, Ph.D.
Next-Generation Therapies

AUTISM SPECTRUM DISORDERS

Laura Lee Colgin, Ph.D.
Basic Research

BIOLOGY OF THE BRAIN

This project focuses on how the brain works

ADAPTIVE DECISION MAKING

Kate M. Wassum, Ph.D.
Basic Research

BIPOLAR DISORDER

Mary L. Phillips, M.D., M.D. (Cantab)
Basic Research

DEPRESSION

Noah Stephen Philip, M.D.
Next-Generation Therapies

Carmen Sandi, Ph.D.
Next-Generation Therapies

PTSD

Sanjay J. Mathew, M.D.
Basic Research

SCHIZOPHRENIA

Dorit Ben-Shachar, Ph.D., DSc,
Basic Research
Next-Generation Therapies

Peter Penzes, Ph.D.
Diagnostic Tools/Early Intervention

"WoodNext is very proud to support innovative research via the Brain & Behavior Research Foundation Distinguished Investigator Grants with a total commitment of $5 million across five years. We recognize that scientific research is the key to discovering new pathways to understanding and treating psychiatric illnesses. The ground-breaking work of the Distinguished Investigator Grant recipients will bring hope and healing to people and families impacted by mental illness. We applaud these scientists for their extraordinary dedication, innovation, and leadership."

Nancy Chan, *Executive Director of the WoodNext Foundation*

BBRF Young Investigator Grants in 2024

748 YOUNG INVESTIGATOR APPLICATIONS

150 YOUNG INVESTIGATOR GRANTS AWARDED

$10.4 MILLION AWARDED IN YOUNG INVESTIGATOR GRANTS

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.

Co-Chairs of the Young Investigator Grant Selection Committee

Judith M. Ford, Ph.D.
Professor, Department of Psychiatry
University of California, San Francisco
President, BBRF Scientific Council
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 Young Investigator Grants are the crowning jewel of BBRF. They provide crucial funding for our rising stars in clinical and basic science who are at the frontier of understanding, probing and developing new therapies for mental illness. The awards allow these investigators to explore innovative approaches and generate preliminary data for larger grants from other sources, including the National Institutes of Health (NIH). Moreover, these competitive and prestigious BBRF grants are highly regarded by granting agencies. Years later, grantees regularly express their gratitude for having received Young Investigator awards, citing how influential these grants were in their career development."

— Dr. Suzanne Haber

2024 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

Laika Aguinaldo, Ph.D.
Diagnostic Tools/Early Intervention

Miguel Barretto-García, Ph.D.
Basic Research

Erin Campbell, Ph.D.
Next-Generation Therapies
Basic Research

Yifeng Cheng, Ph.D.
Basic Research

Kauê Costa, Ph.D.
Basic Research

Priscila Dib Goncalves, Ph.D.
Basic Research

Yang Li, Ph.D.
Basic Research

Carolina Luft, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Peter Manza, Ph.D.
Next-Generation Therapies

Freddyson Martinez-Rivera, Ph.D.
Basic Research
Next-Generation Therapies

Suzanne Nolan, Ph.D.
Basic Research

Brenden Tervo-Clemmens, Ph.D.
Basic Research

Yvan Vachez, Ph.D.
Basic Research

Terril Verplaetse, Ph.D
Basic Research

Heather Webber, Ph.D.
Next-Generation Therapies

Natalie Zlebnik, Ph.D.
Basic Research

ATTENTION-DEFICIT HYPERACTIVITY DISORDER (ADHD)

Ryan Doan, Ph.D.
Basic Research

Cassondra Eng, Ph.D.
Next-Generation Therapies

Zhongzheng Fu, Ph.D.
Basic Research

Masashi Hasegawa, Ph.D.
Basic Research

Jaekyoon Kim, Ph.D.
Basic Research

Hannah Lapp, Ph.D.
Basic Research

Martin Munz, Ph.D.
Basic Research
New Technologies

Heather Snell, Ph.D.
Basic Research
New Technologies

ANXIETY DISORDERS

Elisa Baek, Ph.D.
Basic Research

Liam Barry-Carroll, Ph.D.
Basic Research

Johnathan Borland, Ph.D.
Basic Research

Daniela Calvigioni, Ph.D.
Basic Research

Simon Chang, Ph.D.
Basic Research

Kevin Clancy, Ph.D.
New Technologies
Next-Generation Therapies

Austin Coley, Ph.D.
Basic Research

Jacob Crouse, Ph.D.
Diagnostic Tools/Early Intervention
Basic Research

Camila de Avila Dal'Bo, Ph.D.
Basic Research

Alessandro De Nadai, Ph.D.
Basic Research

Mario Fernandez, Ph.D.
Basic Research

Zachary Harvanek, M.D., Ph.D.
Basic Research

Maryam Hasantash, Ph.D.
Basic Research

Ann Iturra Mena, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Charline Kambrun, Ph.D.
Basic Research

Arielle Keller, Ph.D.
Diagnostic Tools/Early Intervention

Esther Klingler, Ph.D.
Basic Research

Laura Luyten, Ph.D
Basic Research

Kahlilia Morris-Blanco, Ph.D.
Basic Research

Laura Quinones Camacho, Ph.D
Basic Research

Divyangana Rakesh, Ph.D.
Basic Research

Natale Sciolino, Ph.D.
Basic Research

Ourania Semelidou, Ph.D.
Basic Research

Joseph Stujenske, M.D., Ph.D.
Basic Research
Next-Generation Therapies

Jiandong Sun, Ph.D.
Basic Research

Hwei Ee Tan, Ph.D.
Basic Research

Najah Walton, Ph.D.
Basic Research

Lauren White, Ph.D.
Diagnostic Tools/Early Intervention

Ye Wu, Ph.D.
Basic Research

Mingmin Zhang, Ph.D.
Basic Research

Qiancheng Zhao, Ph.D.
Basic Research

Yangzhi Zhu, Ph.D.
Diagnostic Tools/Early Intervention
New Technologies

AUTISM SPECTRUM DISORDER (ASD)

Gabriela Bodea, Ph.D.
Basic Research

Caitlin Clements, Ph.D.
Diagnostic Tools/Early Intervention

Marta Cosin-Tomas, Ph.D.
Basic Research

Veronika Dudarev, Ph.D.
Basic Research

Julien Ferent, Ph.D.
Basic Research

Yi Gu, Ph.D.
Basic Research

Charline Kambrun, Ph.D.
Basic Research

Jaekyoon Kim, Ph.D.
Basic Research

Hannah Lapp, Ph.D.
Basic Research

Hsiang-Yuan Lin, M.D.
Basic Research
Next-Generation Therapies

Martin Munz, Ph.D.
Basic Research
New Technologies

Lingdi Nie, Ph.D.
Basic Research

Heather Snell, Ph.D.
Basic Research

Ye Wu, Ph.D.
Basic Research

Xiyu Zhu, Ph.D.
Basic Research

BIOLOGY OF THE BRAIN

These projects focus on how the brain works

ILLNESS-LINKED COPY NUMBER VARIATIONS

Paola Giusti-Rodriguez, Ph.D.
Basic Research

GLIA IN NEURAL DEVELOPMENT

Husniye Kantarci, Ph.D.
Basic Research

NEURAL BASIS OF SELF-MONITORING

Tadeusz Kononowicz, Ph.D.
Basic Research

INDIVIDUAL VARIATION IN BRAIN DEVELOPMENT

Julia Moser, Ph.D.
Basic Research

ACCELERATED BIOLOGICAL AGING

Julian Mutz, Ph.D.
Basic Research

ESTROGEN RECEPTORS IN BRAIN DEVELOPMENT

Nevena Radonjic, M.D., Ph.D.
Basic Research

CEREBELLAR ALTERATIONS DURING ADOLESCENCE

Adrienne Romer, Ph.D.
Basic Research

LATROPHILINS AND SYNAPSE FORMATION

Hamidreza Shaye, Ph.D.
Basic Research

MICROGLIA IN CIRCUIT DEVELOPMENT

Valerie Tornini, Ph.D.
Basic Research

OPIOID RECEPTORS AND VALENCE PROCESSING

Hector Yarur, Ph.D.
Basic Research

BIPOLAR DISORDER

JinYoung Choi, Ph.D.
Next-Generation Therapies
Basic Research

Adam Fijtman, M.D., Ph.D.
Next-Generation Therapies

Masashi Hasegawa, Ph.D.
Basic Research

Maria Koromina, Ph.D.
Basic Research

Leila Nabulsi, Ph.D.
Basic Research

Beier Yao, Ph.D.
Basic Research

BORDERLINE PERSONALITY DISORDER

Miguel Barretto-García, Ph.D.
Basic Research

Andreea Diaconescu, Ph.D.
Basic Research

Masashi Hasegawa, Ph.D.
Basic Research

Natassia Robinson, Ph.D.
Diagnostic Tools/Early Intervention

CHILDHOOD & ADOLESENCE

Laika Aguinaldo, Ph.D.
Diagnostic Tools/Early Intervention

Liam Barry-Carroll, Ph.D.
Basic Research

Caitlin Clements, Ph.D.
Diagnostic Tools/Early Intervention

Jacob Crouse, Ph.D.
Diagnostic Tools/Early Intervention
Basic Research

Christina Cruz, M.D.
Diagnostic Tools/Early Intervention

Alessandro De Nadai, Ph.D.
Basic Research

Elvisha Dhamala, Ph.D.
Diagnostic Tools/Early Intervention

Priscila Dib Goncalves, Ph.D.
Basic Research

Cassondra Eng, Ph.D.
Next-Generation Therapies

Ann Iturra Mena, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Liat Itzhaky, Ph.D.
Diagnostic Tools/Early Intervention

Kiera James, Ph.D.
Diagnostic Tools/Early Intervention

Arielle Keller, Ph.D.
Diagnostic Tools/Early Intervention

Esther Klingler, Ph.D.
Basic Research

Hannah Lapp, Ph.D.
Basic Research

Carolina Luft, Ph.D.
Basic Research

Julia Moser, Ph.D.
Basic Research

Laura Quinones Camacho, Ph.D.
Basic Research

Divyangana Rakesh, Ph.D.
Basic Research

Natassia Robinson, Ph.D.
Diagnostic Tools/Early Intervention

Adrienne Romer, Ph.D.
Basic Research

Brenden Tervo-Clemmens, Ph.D.
Basic Research

Michelle Thai, Ph.D.
Basic Research

Halide Turkozer, M.D.
Basic Research
Diagnostic Tools/Early Intervention

Terril Verplaetse, Ph.D.
Basic Research

Lauren White, Ph.D.
Diagnostic Tools/Early Intervention

Jiahe Zhang, Ph.D.
Next-Generation Therapies

DEPRESSION

Amanda Arulpragasam, Ph.D.
Next-Generation Therapies

Vanessa Babineau, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Miguel Barretto-García, Ph.D.
Basic Research

Mandakh Bekhbat, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Mirjam Bloemendaal, Ph.D.
Basic Research

Johnathan Borland, Ph.D.
Basic Research

Alexei Bygrave, Ph.D.
Basic Research

Simon Chang, Ph.D.
Basic Research

Roselyne Chauvin, Ph.D.
Basic Research

Austin Coley, Ph.D.
Basic Research

Katharine Dunlop, Ph.D.
Diagnostic Tools/Early Intervention

Franco Giarrocco, Ph.D.
Basic Research

Zachary Harvanek, M.D., Ph.D.
Basic Research

Maryam Hasantash, Ph.D.
Basic Research

Zoe Hawks, Ph.D.
Diagnostic Tools/Early Intervention

Sarah Herzog, Ph.D.
Basic Research

Orna Issler, Ph.D.
Basic Research

Liat Itzhaky, Ph.D.
Diagnostic Tools/Early Intervention

Arielle Keller, Ph.D.
Diagnostic Tools/Early Intervention

Bora Kim, M.D.
Next-Generation Therapies

Esther Klingler, Ph.D.
Basic Research

Sangjun Lee, Ph.D.
Next-Generation Therapies

Hsiang-Yuan Lin, M.D.
Basic Research
Next-Generation Therapies

Clara McCarthy, Ph.D.
Basic Research
Next-Generation Therapies

Kahlilia Morris-Blanco, Ph.D.
Basic Research

Suzanne Nolan, Ph.D.
Basic Research

Marco Pagliusi Jr, Ph.D.
Basic Research

Mohammed Mostafizur Rahman, Ph.D.
Basic Research

Divyangana Rakesh, Ph.D.
Basic Research

Soroosh Sanatkhani, Ph.D.
Next-Generation Therapies

Karel Scheepstra, M.D., Ph.D.
Basic Research
Next-Generation Therapies
New Technologies

Tien Hong Stanley Seah, Ph.D.
Basic Research

Urszula Skupio, Ph.D.
Basic Research

Rachel June Smith, Ph.D.
Next-Generation Therapies
Basic Research

Benjamin Spurny-Dworak, Ph.D.
Basic Research
New Technologies

Brian Sweis, M.D., Ph.D.
Basic Research

Hwei Ee Tan, Ph.D.
Basic Research

Michelle Thai, Ph.D.
Basic Research

Nicholas Trapp, M.D.
Diagnostic Tools/Early Intervention

Najah Walton, Ph.D.
Basic Research

Bo Wang, Ph.D.
Diagnostic Tools/Early Intervention

Junsung Woo, Ph.D.
Basic Research

Ye Wu, Ph.D.
Basic Research

Jiahe Zhang, Ph.D.
Next-Generation Therapies

Tingxin Zhang, Ph.D.
Basic Research

Qiancheng Zhao, Ph.D.
Basic Research

Yangzhi Zhu, Ph.D.
Diagnostic Tools/Early Intervention
New Technologies

EATING DISORDERS

Miguel Barretto-García, Ph.D.
Basic Research

Maxime Chevee, Ph.D.
Basic Research

Alexandre Fisette, Ph.D.
Basic Research

Masashi Hasegawa, Ph.D.
Basic Research

Hector Yarur, Ph.D.
Basic Research

OBSESSIVE-COMPULSIVE DISORDER (OCD)

Zhongzheng Fu, Ph.D.
Basic Research

Franco Giarrocco, Ph.D.
Basic Research

Ann Iturra Mena, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Jaekyoon Kim, Ph.D.
Basic Research

Liming Qiu, M.D.
Basic Research
Next-Generation Therapies

OTHER DISORDERS

ALZHEIMER'S

Camila de Avila Dal'Bo, Ph.D.
Basic Research

EPILEPSY

Lingdi Nie, Ph.D.
Basic Research

EPILEPSY

Rachel June Smith, Ph.D.
Next-Generation Therapies
Basic Research

PARKINSON'S DISEASE

Nikolai Gil Reyes, M.D.
Basic Research

STROKE

Kahlilia Morris-Blanco, Ph.D.
Basic Research

TRAUMATIC BRAIN INJURY

Dongtak Lee, Ph.D.
Next-Generation Therapies
New Technologies

POST-TRAUMATIC STRESS DISORDER (PTSD)

Vanessa Babineau, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Liam Barry-Carroll, Ph.D.
Basic Research

Johnathan Borland, Ph.D.
Basic Research

Fenglin Cao, Ph.D.
Basic Research

Kevin Clancy, Ph.D.
New Technologies
Next-Generation Therapies

Xin Deng, Ph.D.
Basic Research

Mario Fernandez, Ph.D.
Basic Research

Thomas Hainmueller, M.D., Ph.D.
Basic Research
Next-Generation Therapies

Zachary Harvanek, M.D., Ph.D.
Basic Research

Maryam Hasantash, Ph.D.
Basic Research

Ann Iturra Mena, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Munir Kutlu, Ph.D.
Basic Research

Joseph Stujenske, M.D., Ph.D.
Basic Research
Next-Generation Therapies

Najah Walton, Ph.D.
Basic Research

Lauren White, Ph.D.
Diagnostic Tools/Early Intervention

PRENATAL BRAIN DEVELOPMENT

Vanessa Babineau, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Marta Cosin-Tomas, Ph.D.
Basic Research

Julien Ferent, Ph.D.
Basic Research

Husniye Kantarci, Ph.D.
Basic Research

Martin Munz, Ph.D.
Basic Research
New Technologies

Nevena Radonjic, M.D., Ph.D.
Basic Research

Ai Tian, Ph.D.
Basic Research

Valerie Tornini, Ph.D.
Basic Research

Yingying Zhang, Ph.D.
Basic Research

PSYCHOSIS

Jacob Crouse, Ph.D.
Diagnostic Tools/Early Intervention
Basic Research

Elvisha Dhamala, Ph.D.
Diagnostic Tools/Early Intervention

Tom Franken, M.D., Ph.D.
Basic Research

Leanna Hernandez, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Katie Lavigne, Ph.D.
Basic Research

Emmanuel Mwesiga, M.D., Ph.D.
Basic Research

Danielle Pratt, Ph.D.
Basic Research

Nikolai Gil Reyes, M.D.
Basic Research

Rik Schalbroeck, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Satoshi Terada, Ph.D.
Basic Research
New Technologies

Ralitsa Todorova, Ph.D.
Basic Research

Halide Turkozer, M.D.
Basic Research
Diagnostic Tools/Early Intervention

Veith Weilnhammer, M.D., Ph.D.
Basic Research

Nicholas Wright, Ph.D.
Basic Research

Beier Yao, Ph.D.
Basic Research

SCHIZOPHRENIA

Samantha Abram, Ph.D.
Next-Generation Therapies

Gabriela Bodea, Ph.D.
Basic Research

Marta Cosin-Tomas, Ph.D.
Basic Research

Henry Cowan, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Jacob Crouse, Ph.D.
Diagnostic Tools/Early Intervention
Basic Research

Elvisha Dhamala, Ph.D.
Diagnostic Tools/Early Intervention

Michael-John Dolan, Ph.D.
Basic Research

Iris Donga Vilares, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Julien Ferent, Ph.D.
Basic Research

Tom Franken, M.D., Ph.D.
Basic Research

Zhongzheng Fu, Ph.D.
Basic Research

Yi Gu, Ph.D.
Basic Research

Leanna Hernandez, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Kevin Kelley, M.D., Ph.D.
Basic Research

Jaekyoon Kim, Ph.D.
Basic Research

Hannah Lapp, Ph.D.
Basic Research

Katie Lavigne, Ph.D.
Basic Research

Martin Munz, Ph.D.
Basic Research
New Technologies

Emmanuel Mwesiga, M.D., Ph.D.
Basic Research

Lingdi Nie, Ph.D.
Basic Research

Suzanne Nolan, Ph.D.
Basic Research

Christopher Parkhurst, M.D., Ph.D.
Basic Research

Danielle Pratt, Ph.D.
Basic Research

Nikolai Gil Reyes, M.D.
Basic Research

Rik Schalbroeck, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Rajyashree Sen, Ph.D.
Basic Research

Satoshi Terada, Ph.D.
Basic Research
New Technologies

Ai Tian, Ph.D.
Basic Research

Ralitsa Todorova, Ph.D.
Basic Research

Halide Turkozer, M.D.
Basic Research
Diagnostic Tools/Early Intervention

Alban Voppel, Ph.D.
Basic Research

Ryan Walsh, Ph.D.
Basic Research

Veith Weilnhammer, M.D., Ph.D.
Basic Research

Frederike Winkel, Ph.D.
Basic Research

Nicholas Wright, Ph.D.
Basic Research

Yingying Zhang, Ph.D.
Basic Research

Xiaoqing Zhou, Ph.D.
Basic Research

Xiyu Zhu, Ph.D.
Basic Research

SUICIDE PREVENTION

Laika Aguinaldo, Ph.D.
Diagnostic Tools/Early Intervention

Brian Albanese, Ph.D.
Diagnostic Tools/Early Intervention

Hongsheng Gui, Ph.D.
Diagnostic Tools/Early Intervention

Sarah Herzog, Ph.D.
Basic Research

Liat Itzhaky, Ph.D.
Diagnostic Tools/Early Intervention

Kiera James, Ph.D.
Diagnostic Tools/Early Intervention

Bora Kim, M.D.
Next-Generation Therapies

Junsung Woo, Ph.D.
Basic Research

2024 Young Investigators: Institutional Affiliations at the Time of the Grant

(in alphabetical order)

Amsterdam University Medical Centers, The Netherlands (2)

Arizona State University

Barcelona Institute for Global Health, Spain

Baylor College of Medicine

Brown University/Ocean State Research Institute, Inc.

Collège de France, France

Columbia University (8)

Dell Medical School, University of Texas at Austin (3)

Emory University

Feinstein Institute for Medical Research/Northwell Health

Harvard University

Harvard University/Boston Children's Hospital (2)

Harvard University/Brigham and Women's Hospital, Inc.

Harvard University/Massachusetts General Hospital (4)

Harvard University/McLean Hospital (3)

Henry Ford Health System

Icahn School of Medicine at Mount Sinai (2)

INSERM, France (3)

Johann Wolfgang Goethe University, Germany

Johns Hopkins University

Karolinska Institute, Sweden (2)

King's College London, UK

King's College London/ Institute of Psychiatry, UK

Krembil Research Institute/University Health Network, Canada

Leuven University, Belgium

Makerere University, Uganda

McGill University, Canada (2)

McLean Hospital (4)

Medical University of Vienna, Austria

Memorial Sloan-Kettering Cancer Center

Michigan State University

Nanyang Technological University, Singapore

National Institute of Mental Health (NIMH/NIH) (3)

National Institute of Neurological Disorders and Stroke (NINDS/NIH)

National Institute on Alcohol Abuse & Alcoholism (NIAAA/NIH)

NeuroCenter Magendie U1215 (INSERM), France

New York University (2)

Northeastern University

Northwestern University

Paris-Saclay University, France

Pontifical Catholic University of Rio Grande do Sul, Brazil

Research Foundation for Mental Hygiene, Inc./ Nathan Kline Institute (2)

Research Foundation for the State University of New York, Upstate Medical University

Rowan University

Rutgers University

Stanford University (4)

Terasaki Institute for Biomedical Innovation

Texas A&M University

Trinity College, Dublin, Ireland

Tufts University (2)

Unity Health Toronto, Canada

Université du Québec à Trois-Rivières Canada,

University of Alabama at Birmingham (2)

University of Alberta, Canada

University of Bordeaux, France

University of British Columbia, Canada

University of Calgary/The Hospital for Sick Children, Canada,

University of California, Berkeley

University of California, Los Angeles (6)

University of California, Riverside

University of California, San Diego

University of California, San Francisco/ Gladstone Institutes

University of California, San Francisco/San Francisco VA Medical Center

University of Connecticut

University of Florida (2)

University of Iowa (2)

University of London/Maudsley Hospital/ King's College London, UK

University of Minnesota (5)

University of Newcastle, Australia

University of North Carolina at Chapel Hill (2)

University of Notre Dame

University of Pennsylvania (3)

University of Pennsylvania/ Children's Hospital of Philadelphia

University of Pittsburgh (3)

University of Queensland, Australia

University of Regensburg, Germany

University of São Paulo, Brazil,

University of Southern California (2)

University of Sydney, Australia

University of Texas Health Science Center at Houston

University of Texas Southwestern Medical Center at Dallas (2)

University of Toronto/Centre for Addiction and Mental Health, Canada (2)

University of Toronto/University Health Network, Canada,

University of Washington

Vanderbilt University (3)

VIB-KU Leuven Center for Brain & Disease Research, Belgium

Virginia Polytechnic Institute and State University

Washington University, St. Louis

Washington University School of Medicine (2)

Weill Cornell Medical College

Western Washington University

Yale University (2)

Yale University/Yale University School of Medicine (2)

2024 BBRF EVENTS

“The Quest for Healthy Minds” January 11, 2024

Dr. Judy Genshaft,
BBRF Board Secretary

Geoffrey Simon,
BBRF Chairman of the Board

Dr. Judy Genshaft, President Emerita of the University of South Florida and BBRF Board Secretary, hosted an event at the Judy Genshaft Honors College, where attendees learned about BBRF’s unique model for advancing the frontiers of brain science.

The BBRF video, also entitled “The Quest for Healthy Minds,” highlights how BBRF’s support for brain research is paving the way to new treatments and methods of prevention.

The video features four groundbreaking scientists—now world leaders in mental health research—who were supported by BBRF early in their careers, when they were struggling for funding.

Watch it here:
https://www.youtube.com/watch?v=qh0QWi2zTBs

Scientific Council Dinner Honoring Herbert Pardes, M.D.

The 2024 BBRF Scientific Council Dinner celebrated and honored the extraordinary life of Herbert Pardes, M.D., a dynamic leader in psychiatry and mental health, and Founder and President of the BBRF Scientific Council.

Dr. Pardes was a champion of mental health research. He believed it was vital to encourage and support talented scientists in their work to expand the frontiers of neuropsychiatric research, which he fervently believed was our best hope for ending the immense suffering caused by mental illness.

Dr. Pardes' legacy in the field of psychiatry is unparalleled. He played leadership roles in psychiatric care, neuroscience research, medical education, and health care policy. BBRF remains dedicated to building upon Dr. Pardes' extraordinary legacy of expanding the frontiers of psychiatric research, with the same passion and commitment he lovingly shared with us for 37 years.

The evening also celebrated the BBRF Klerman & Freedman Prizes and honored five young researchers for their significant findings related to suicide prevention, PTSD, substance-use disorders, autism, brain biology, and therapeutic drug development. These Prizes recognize exceptional clinical and basic research conducted by BBRF Young Investigator Grantees.

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 prizewinners are selected by special committees of the Foundation's Scientific Council.

Top Photo: Geoffrey Simon, Dr. Elizabeth Goldfarb, Dr. Christina Kim, Dr. Erin Gibson, Dr. Juliet Beni Edgcomb, and Dr. Jeffrey Borenstein.

Bottom Photo: Geoffrey Simon, Chairman of the BBRF Board, Dr. Judith Ford, President of the BBRF Scientific Council, and Dr. Jeffery Borenstein, BBRF President & CEO.

ANNUAL KLERMAN PRIZE FOR EXCEPTIONAL CLINICAL RESEARCH

Juliet Beni Edgcomb, M.D., Ph.D.
University of California, Los Angeles

HONORABLE MENTION

Elizabeth V. Goldfarb, Ph.D.
Yale University

ANNUAL FREEDMAN PRIZE FOR EXCEPTIONAL BASIC RESEARCH

Christina K. Kim, Ph.D.
University of California, Davis

HONORABLE MENTIONS

Erin Gibson, Ph.D.
Stanford University

Hugo A. Tejeda, Ph.D.
National Institute of Mental Health

STRIVING TOWARD CURES THROUGH RESEARCH

Scientist Spotlight with: **Christina K. Kim, Ph.D.**

2024 BBRF Freedman Prize for Exceptional Basic Research

2021 BBRF Young Investigator Grant

Dr. Kim seeks to develop new methodologies for recording and perturbing neuronal activity in animal models, specifically in brain regions implicated in neuropsychiatric diseases. Specifically, her lab engineers technologies to identify molecular biomarkers in neurons modulated by novel therapeutic drugs for treating depression and anxiety. She aims to determine which specific neurons and molecules can be targeted to improve disease symptoms. The hope is that an understanding of the targeted cellular mechanisms of therapeutic drugs in the brain can serve as the basis for designing modifications to these agents to increase their specificity and reduce their undesired side effects.

WHAT IS THE CURRENT STATE OF YOUR BBRF-FUNDED RESEARCH?

This first half of our BBRF-funded Research is described in a completed manuscript currently under revision at a journal. The second half has been presented in its entirety at Society for Neuroscience, and is currently being prepared for submission as a manuscript.

WHAT DID YOUR BBRF GRANT MEAN TO YOU?

The BBRF Young Investigator Grant gave me the freedom to try a risky but fruitful new project, which is now the foundational basis of many other ongoing projects in my lab. Without this support, it would have been challenging for us to launch this exciting research direction in our lab.

"I am so grateful to be receiving the Freedman Prize from BBRF. It is a wonderful recognition that comes right at the beginning of my career. This award empowers me to continue pursuing challenging and risky problems in neuropsychiatric disease research."

WHAT HAS BBRF SUPPORT MEANT TO YOUR CAREER?

BBRF support has sparked my career as a technology developer studying neuropsychiatric disorders. This early support guided my independent lab's current research program and vision..

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

People living with mental illness can teach us so much about these disorders, and what aspects of these illnesses are the most challenging to cope with. Their resilience is the inspiration for why we as researchers continue to seek answers in the lab. Through interactions with people living with mental illnesses, I'm able to share knowledge and hope with those who need it. These interactions also motivate me to think deeply about the purpose and motivation of our lab's research.

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

In the best possible scenario, we hope that our research reveals an understanding of how certain treatments work to combat symptoms of mental illness in patients. By understanding the targeted cellular mechanisms of these drugs in the brain, we can then begin to design modifications to them to increase their specificity and reduce their undesired side effects. The ultimate hope is to improve the day-to-day lives of patients and their families.

Scientist Spotlight with: **Juliet Beni Edgcomb, M.D., Ph.D.**

2024 BBRF Klerman Prize for Exceptional Clinical Research

2020 BBRF Young Investigator Grant

Dr. Edgcomb seeks to develop a set of rules for clearly identifying children and adolescents with suicide-related symptoms from within noisy and complex electronic health record (EHR) data. This would be a significant step forward in suicide prevention. She has worked with an EHR "training set" that includes data from 400 children and adolescents, to identify those presenting to healthcare providers with suicidal thoughts and behaviors. The aim is to identify the predictive variables best defining each pertinent phenotype and to produce a predicted probability of each suicide-related phenotype for each child.

" This is an extraordinary honor that deeply humbles and inspires me. It is a recognition not just of individual efforts, but of collaborative spirit and dedication that drive the field of psychiatric research. This accolade is a powerful reminder of our shared purpose and energizes my commitment."

WHAT IS THE CURRENT STATE OF YOUR BBRF-FUNDED RESEARCH?

The project developed methods to detect suicide and self-harm from electronic health records of 600 children receiving emergency care. Results demonstrated a gap in detection with commonly used strategies and the potential of new computational methods to enhance detection. We since expanded this project to develop and validate methods to detect suicide-related visits among 3400 children. Preliminary findings from this larger study reveal disparities in detection by age, sex, and race/ethnicity with existing strategies, and the potential for medical record phenotyping to attenuate disparities in detection of populations at-risk and in need.

WHAT DID YOUR BBRF GRANT MEAN TO YOU?

Receiving a BBRF Young Investigator Grant was not only a tremendous honor but also has been vital catalyst for my research. This grant represents both recognition and support from the scientific community for my work in exploring innovative approaches to understanding mental health. I am deeply grateful for this opportunity to make a difference in the lives of those affected by mental illness.

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

Every encounter offers me a deeper understanding of the unique struggles and strengths of young people facing mental health challenges. These conversations not only deepen my empathy but also continuously refine my approach to treatment and prevention.
On a personal level, as a parent, these interactions remind me of the resilience of the human spirit and the impact of empathy. They reinforce my commitment to destigmatizing mental health issues and advocating for better mental health resources. .

WHAT HAS BBRF SUPPORT MEANT TO YOUR CAREER?

My project was honored with the Steven, Sally, and Isabella Grimes Investigator Award through the BBRF Young Investigator Grant. This dedication by a specific family instilled purpose in my work, and connected discovery and hope. While BBRF support tangibly made possible my research, it also reinforced a conviction that this work can transform care for children and save lives.

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

Labor-intensive manual chart reviews, diagnostic codes, and literal term searches remain the predominant mode of detecting suicidal behavior among children, limiting sample size to detect variation in care and access to life-saving suicide prevention interventions. By advancing strategies to efficiently detect need at scale, my goal is to reduce the time between research innovation and clinical practice.

STRIVING TOWARD CURES THROUGH RESEARCH

2024 INTERNATIONAL MENTAL HEALTH RESEARCH SYMPOSIUM

BBRF held its annual International Mental Health Symposium at the Kaufman Music Center in New York City, which was simultaneously live-streamed. Six 2024 BBRF Outstanding Achievement Prizewinners gave presentations on topics that included identifying risk factors for psychosis prior to the first appearance of symptoms; a cognitive neuroscience approach to understanding circuits and symptoms in psychosis; exploration of mechanisms in breakthrough rapid-acting therapies for mood disorders; and the investigation of differences in early social development in relation to their clinical and translational implications.

The symposium also featured a presentation from Franca Ma-ih Sulem Yong, the winner of the 2024 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 YouTube channel:
https://bbrfoundation.org/blog/2024-international-mental-health-research-symposium-presentations

Standing L to R: Dr. Carol Tamminga, Dr. Jeffrey Borenstein, Franca Ma-ih Sulem Yong, Dr. Cameron Carter, Dr. Deanna Barch, Dr. Nicole Karcher, Dr. Chistopher McDougle, Dr. Nolan Williams, and Geoffrey Simon. Photos by Chad David Kraus.

Presentations

Identifying Risk For Developing Psychosis So We Can Promote Prevention

Deanna M. Barch, Ph.D.
Washington University in St. Louis

BBRF LIEBER PRIZEWINNER FOR OUTSTANDING ACHIEVEMENT IN SCHIZOPHRENIA RESEARCH

Identifying Risk Factors For Early Psychosis Spectrum Symptoms

Nicole Karcher, Ph.D.
Washington University in St. Louis

BBRF MALTZ PRIZEWINNER FOR INNOVATIVE AND PROMISING SCHIZOPHRENIA RESEARCH

Breakthrough Rapid-Acting Therapeutics: Exploring Efficacy and Mechanisms in Treatment-Resistant Mood Disorders

Nolan R. Williams, M.D.
Stanford University

BBRF COLVIN PRIZEWINNER FOR OUTSTANDING ACHIEVEMENT IN MOOD DISORDERS RESEARCH

New Horizons for Child Psychiatry From Research on Individual Differences in Early Social Development

John N. Constantino, M.D.
Pediatric Institute, Children's Healthcare of Atlanta
Emory University

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

The Psychopharmacology of Childhood-Onset Neuropsychiatric Disorders Across the Lifespan

Christopher J. McDougle, M.D.
Massachusetts General Hospital / Harvard Medical School

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

A Cognitive Neuroscience Approach to Understanding Circuits and Symptoms in Psychosis

Cameron S. Carter, M.D.
University of California, Irvine

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

Navigating My ADHD Through Self Art Therapy

Franca Ma-ih Sulem Yong
Founder, Afrogiveness Movement

PRIZEWINNER, BBRF PARDES HUMANITARIAN PRIZE IN MENTAL HEALTH

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The Pardes Humanitarian Prize in Mental Health

The 2024 Pardes Humanitarian Prize in Mental Health was awarded on October 25th and honored **Franca Ma-ih Sulem Yong** for serving as an extraordinary advocate for tolerance, forgiveness, mental health, and human fraternity. She is a champion of mental health rights and a leading force for healing in Africa.

Dr. Jeffrey Borenstein and Franca Ma-ih Sulem Yong

The 2024 Honorary Pardes Humanitarian Prize in Mental Health was awarded to the **Graham Boeckh Foundation** for serving as a catalyst for transformational changes that significantly improve the lives of people living with, or at risk of, mental illness.

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.

(L–R): Tony Boeckh, Raymonde Boeckh, Dr. Jeffrey Borenstein, and Ian Boeckh

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.

PREVIOUS PRIZE WINNERS

2023
SPECIAL OLYMPICS INTERNATIONAL
Honorary Tribute: Henry Jarecki, M.D.

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 25, 2024

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

Deanna M. Barch, Ph.D.

Washington University, St. Louis

BBRF Scientific Council Member
2013 BBRF Distinguished Investigator
2006 BBRF Independent Investigator
2000, 1995 BBRF Young Investigator

RUANE PRIZE

Outstanding Achievement in Child & Adolescent Psychiatric Research

John N. Constantino, M.D.

Pediatric Institute, Children's Healthcare of Atlanta
Emory University

MALTZ PRIZE

Innovative & Promising Schizophrenia Research

Nicole Karcher, Ph.D.

Washington University, St. Louis

Christopher J. McDougle, M.D.

Massachusetts General Hospital / Harvard Medical School

1997 BBRF Independent Investigator
1994, 1990 BBRF Young Investigator

COLVIN PRIZE

Outstanding Achievement in Mood Disorders Research

Nolan R. Williams, M.D.

Stanford University

2019 BBRF Klerman Prize for Exceptional Clinical Research
2018, 2016 BBRF Young Investigator

GOLDMAN-RAKIC PRIZE

Outstanding Achievement in Cognitive Neuroscience

Cameron S. Carter, M.D.

University of California, Irvine

BBRF Scientific Council Member
2007 BBRF Distinguished Investigator
2001 BBRF Klerman Prize for Exceptional Clinical Research
1997, 1994 BBRF Young Investigator

Healthy Minds

The Emmy nominated television series, ***Healthy Minds with Dr. Jeffrey Borenstein,*** produced by BBRF, 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.

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

Season 10 will be available to watch in the Fall at: https://www.pbs.org/show/healthy-minds-with-dr-jeffrey-borenstein/

SEASON 9 EPISODES:

Episode 1 — Metabolic Psychiatry

A ketogenic diet focused on increased protein and decreased carbohydrates has shown positive results for patients with bipolar disorder, epilepsy, and schizophrenia. *Guests: Jan Ellison Baszucki, mother of bipolar patient, now funding research as President, Baszucki Group; Judith M. Ford, Ph.D., Professor of Psychiatry, University of California, San Franciso.*

Episode 2 — Update on COVID and Mental Health

A follow up to the 2022 season of "Healthy Minds" explores some potential long-term effects of COVID including depression, anxiety, psychosis, and brain fog, as well as treatments for these conditions. *Guest: Maura Boldrini, M.D., Associate Professor of Psychiatry, Columbia University Vagelos College of Physicians and Surgeons and Director, Quantitative Brain Biology Institute (Brain QUANT).*

Episode 3 — Helping People Who Are Homeless, Part 1

A model program in Boston offers a holistic approach to clinical care for the homeless built around a street team bringing mental health resources directly to those most in need, including case workers, psychiatrists, and a recovery coach who has experienced being homeless. *Guest: Katherine Koh, M.D., Assistant Professor of Psychiatry, Harvard Medical School and Street Psychiatrist, Boston Health Care for the Homeless Program.*

Episode 4 — Helping People Who Are Homeless, Part 2

Research to improve clinical care and positive outcomes for the homeless population includes understanding risk factors for homelessness, including the need for mental health support during transitions out of the military, jail, and foster care. *Guest: Katherine Koh, M.D., Assistant Professor of Psychiatry, Harvard Medical School and Street Psychiatrist, Boston Health Care for the Homeless Program.*

Episode 5 — Post-Traumatic Stress in Children and Adolescents

Topics covered: PTSD looks different in children and adolescents than in adults; What factors contribute to trauma's long-term effects?; unique treatments for youth including eye movement desensitization and reprocessing (EMDR); the need for suicide prevention awareness after trauma in young people's lives. *Guest: Ryan Herringa, M.D., Ph.D., University of Wisconsin Health Professor in Children and Adolescent Psychiatry, University of Wisconsin School of Medicine and Public Health.*

Episode 6 — Prenatal Choline and Brain Health

The nutrient choline has been shown to support fetal brain development, and supplements taken during pregnancy may lead to improved concentration and attention spans in childhood as well as a decreased risk of schizophrenia for these children later in life. *Guest: Robert Freedman, M.D., Department of Psychiatry, University of Colorado School of Medicine.*

Episode 7 — Diagnosis and Treatment for Subtypes of Depression

New research using brain scans and biological markers has revealed areas of connectivity in the brain that can make diagnosis and treatment of the various types of depression more efficient and effective and identify the fundamental mechanisms that make moods change. *Guest: Conor Liston, M.D., Ph.D., Professor of Neuroscience and Psychiatry, Weill Cornell Medicine.*

Episode 8 — Treatment of Early Psychosis

Coordinated care including early intervention, education, a team of medical experts, and a strong support system of family as well as peers with shared experience can increase positive outcomes for young people after a first psychotic episode. The leader of the "On Track New York" program, a doctor and sibling of an early onset patient herself, explores the advances in understanding and treating adolescents and young adults experiencing hallucinations and other symptoms. *Guest: Lisa Dixon, M.D., Professor of Psychiatry, Columbia University Vagelos College of Physicians and Surgeons.*

Episode 9 — Mental Health, Obesity and Diabetes

Research that looks at mental health holistically has revealed that half of all patients with depression or bipolar disorder patients are diabetic or pre-diabetic, leading to a new perspective on symptoms and treatment regarding insulin and brain function. *Guest: Roger McIntyre, M.D., FRCPC, Professor of Psychiatry and Pharmacology, University of Toronto, Canada*

Episode 10 — ADHD: What You Need to Know

Demystifying the symptoms of Attention-Deficit Hyperactivity Disorder and understanding the variety of ways ADHD presents in young people including differences in which aspect of the disorder is manifested, and best advice for caregivers to help young people lead successful lives after diagnosis. *Guest: Stephen P. Hinshaw, Ph.D., Distinguished Professor of Psychology, University of California, Berkeley, and Professor of Psychiatry and Behavioral Sciences, University of California, San Francisco.*

Episode 11 — Education and Opportunities for People with Neurodiversity

A model academic program in Florida addresses the needs of neurodiverse students, founded by parents looking for resources. LiFT (Learning Independence for Tomorrow) Academy serves kindergarten through 12th grade, and LiFT University Transition Program is a four-year post-secondary transition program for students who have completed high school for continued academics, career readiness, and life skill training. *Guest: Keli Mondello, co-founder and Chairman of the Board, Learning Institute for Tomorrow (LiFT).*

Episode 12 — Anxiety in Youth

Diagnosing and treating anxiety in childhood and adolescence can decrease the risk of developing depression and other mental disorders later in life. Advice for parents to recognize the differences between normal worries and anxiety, and the impact of outside factors including bullying and social media. *Guest: Daniel Pine, M.D., National Institutes of Health Distinguished Investigator.*

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 2024:

PTSD: The Brain Basis of Susceptibility
Tuesday, January 9, 2024
Nathaniel G. Harnett, Ph.D.
McLean Hospital
Harvard Medical School

Developing Biological Markers to Improve Clinical Care in Autism
Tuesday, February 13, 2024
James McPartland, Ph.D.
Yale School of Medicine

Detection of Suicide-related Emergencies Among Children Using Real-world Clinical Data
Tuesday, March 12, 2024
Juliet B. Edgcomb, M.D., Ph.D.
University of California, Los Angeles

Enhancing Positive Emotions to Prevent Depression in Youth
Tuesday, April 9, 2024
Autumn Kujawa, Ph.D.
Vanderbilt University

Optimizing Deep Brain Stimulation for Obsessive-Compulsive Disorder
Tuesday, May 14, 2024
Allison C. Waters, Ph.D.
Icahn School of Medicine at Mount Sinai

Neurocognitively-Defined Subtypes in Bipolar Disorder: A Path to More Personalized Treatments
Tuesday, June 11, 2024
Katherine E. Burdick. Ph.D.
Brigham and Women's Hospital
Harvard Medical School

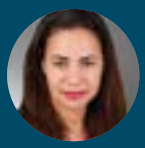
Motivational Modulation of Cognitive Control in ADHD
Tuesday, July 9, 2024
Kimberly S. Chiew, Ph.D.
University of Denver

Timing is Everything: Repeated Neurocognitive Assessments Can Help Us Understand Symptom Patterns in Bulimia Nervosa
Tuesday, August 13, 2024
Laura A. Berner, Ph.D.
Icahn School of Medicine at Mount Sinai

New Diagnostic Tools to Predict Symptom Improvements in Personality Disorders
Tuesday, September 10, 2024
Jenna M. Traynor, Ph.D.
Harvard Medical School

Toward Brain-Based Prediction of Recovery: How Neuroimaging Can Help Combat the Substance-Use Epidemic
Tuesday, October 8, 2024
Sarah W. Yip, Ph.D., MSc
Yale School of Medicine

Intergenerational Effects of Adversity on Mind-Body Health: Pathways Through the Gut-Brain Axis
Tuesday, November 12, 2024
Bridget Laura Callaghan, Ph.D.
University of California, Los Angeles



Investigating the Genetic and Biological Mechanisms That Predispose to Early-Onset Psychotic Illnesses
Tuesday, December 10, 2024
Catherine Astrid Brownstein, Ph.D., M.P.H.
Harvard Medical School, Boston Children's Hospital

BBRF EDUCATIONAL WEBINAR

ADHD: What You Need to Know – A Q&A with Dr. Stephen Hinshaw

This special webinar was broadcast via Zoom on December 4, 2024.

Teachers, school counselors and other educational professionals are on the front line in helping students with mental health challenges and can be among the first to notice when a child is struggling. In their pivotal relationships with students and families, these professionals are often in a position to match resources with needs. By expanding their knowledge of mental health issues experienced by young people, school faculty and staff may be better able to intervene earlier and make referrals for evaluation and assistance which can positively impact student well-being, performance and family life.

This conversation provided educators with a deeper understanding of Attention-Deficit Hyperactivity Disorder and the variety of ways ADHD presents in young people. BBRF President & CEO, Dr. Jeffrey Borenstein and Dr. Stephen Hinshaw discussed the different ways the disorder manifests, differences in presentation between boys and girls, and provided advice for caregivers and educators as to how to help young people lead successful lives after diagnosis.

This webinar is available on the BBRF website:
https://bbrfoundation.org/event/adhd-what-you-need-know-qa-dr-stephen-hinshaw

THANK YOU TO OUR DONORS

Your support this year enabled us to fund over $11 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 $462 million to fund more than 6,700 grants to more than 5,600 leading scientists around the world.

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

Dr. Herbert Pardes Tribute Fund

In Tribute and Memory of Herbert Pardes, M.D. and his continued legacy, importance, and leadership of the BBRF Scientific Council.

Herbert Pardes, M.D., President of the BBRF Scientific Council from its inception in 1987, passed away on April 30, 2024, at the age of 89. In 1987, Dr. Pardes was among the small group of patient advocates, physicians, and researchers who founded the National Alliance for Research on Schizophrenia and Depression (NARSAD), renamed the Brain & Behavior Research Foundation (BBRF) in 2011.

Today BBRF's Scientific Council is comprised of 192 members that include leaders in neurobiology, neuroscience, clinical care, psychology, and psychiatry. These esteemed individuals have recommended more than 6,700 grants for funding and are highly regarded in their respective fields. They have a profound understanding of psychiatric research funding, a deep knowledge of how the scientific community operates and are the bedrock of the organization.

In honor of Dr. Pardes' remarkable legacy and his unparalleled contributions to the field, a fundraising campaign for the Scientific Council was launched with the goal of supporting BBRF's mission and continuing the important work that Dr. Pardes dedicated his life to.

Dr. William Byerley (Scientific Council Member Emeritus) generously matched donations up to $125,000 from the Scientific Council members.

Below is a listing of BBRF Scientific Council Members who raised $195,360:

Ted Abel, Ph.D.
Deanna M. Barch, Ph.D.
Peter F. Buckley, M.D.
William F. Byerley, M.D.
Cameron S. Carter, M.D., M.S.
Jacqueline N. Crawley, Ph.D.
Lisa Beth Dixon, M.D., M.P.H.
Drs. Judith M. Ford and Daniel Mathalon
Dr. and Mrs. Alan Frazer
Dr. and Mrs. Robert R. Freedman
Anthony A. Grace, Ph.D.
Dr. Suzanne N. Haber and Mr. William L. Thomson
Jonathan A. Javitch, M.D., Ph.D.
Dilip V. Jeste, M.D.
Drs. John H. Krystal and Bonnie Becker
Dr. and Mrs. George Lantos
Drs. James F. Leckman and Hannah H. Leckman
Ellen E. Lee, M.D.
Dr. and Mrs. Robert H. Lenox
Helen S. Mayberg, M.D.
Dr. and Mrs. Herbert Y. Meltzer
David A. Morilak, Ph.D.
Dr. and Mrs. Eric J. Nestler
Thomas C. Neylan, M.D.
Patricio O'Donnell, M.D., Ph.D.
Dr. Daniel S. Pine and Ms. Judith Brazen
Dr. and Mrs. Robert M. Post
Gerard A. Sanacora, M.D., Ph.D.
Joanna E. Steinglass, M.D.
Jeremy M. Veenstra-VanderWeele, M.D., Ph.D.
Drs. Myrna M. Weissman and James C. Frauenthal

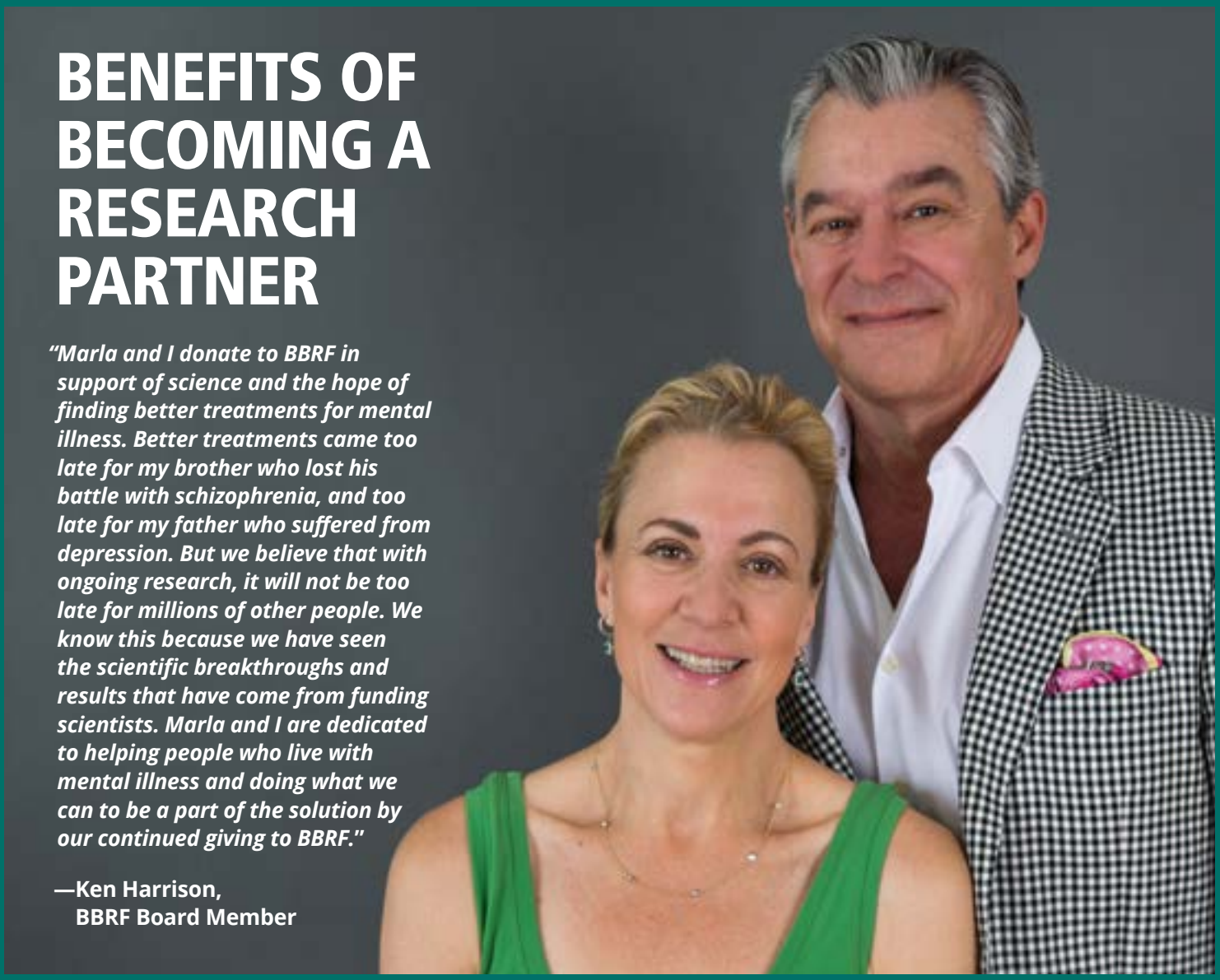
In 2019, the **Moritz Hilder Innovative Brain Research Fund** was established by the Trustee of the Jane Hilder Harris Trust with an endowment gift of $3.5 million. It was created to preserve and honor the memory of Moritz Hilder, the father of the late Jane Hilder Harris.

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 2024, the Endowment provided $199,852 for the funding of six PTSD research projects.

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

BENEFITS OF BECOMING A RESEARCH PARTNER



"Marla and I donate to BBRF in support of science and the hope of finding better treatments for mental illness. Better treatments came too late for my brother who lost his battle with schizophrenia, and too late for my father who suffered from depression. But we believe that with ongoing research, it will not be too late for millions of other people. We know this because we have seen the scientific breakthroughs and results that have come from funding scientists. Marla and I are dedicated to helping people who live with mental illness and doing what we can to be a part of the solution by our continued giving to BBRF."

—Ken Harrison,
BBRF Board Member

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

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Jon Altschul Investigators

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Young Investigator
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University of Michigan

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ANONYMOUS
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Young Investigator
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Bob & Rita Elmen Family Foundation Investigator

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University of Saskatchewan

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McLean Hospital - Harvard University

Young Investigator
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Yale University

Young Investigator
Fabian Streit, Ph.D.
Central Institute of Mental Health, Mannheim - University of Heidelberg

Young Investigator
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McLean Hospital - Harvard University

ANONYMOUS
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McLean Hospital - Harvard University

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Icahn School of Medicine at Mount Sinai

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Young Investigator
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Columbia University

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Katowitz Investigators

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Trinity College, Dublin

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Jia Guo, Ph.D.
Columbia University

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Rosalie & Daniel Katz Family Investigators

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Massachusetts General Hospital - Harvard University

Young Investigator
Aiste Lengvenyte, M.D.
University of Montpellier

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Kenny Family Investigator

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Oregon Health & Science University

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Jeanne Marie Lee Investigators

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Marta Cosin-Tomas, Ph.D.
Barcelona Institute for Global Health

Young Investigator
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Young Investigator
Cassondra M. Eng, Ph.D.
Stanford University

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Joana Ferreira, Ph.D.
Center for Neuroscience and Cell Biology (CNC)

Young Investigator
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University of California, San Francisco - The J. David Gladstone Institutes

Young Investigator
Sayan Nandi, Ph.D.
Howard University

Young Investigator
Kirsten E. Schoonover, Ph.D.
University of Alabama at Birmingham

Young Investigator
Johanna Seitz-Holland, M.D., Ph.D.
Brigham and Women's Hospital, Inc. - Harvard University

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Molly Lou Foundation Investigators

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Indian Institute of Science

Young Investigator
Shokouh Arjmand, Pharm.D.
Aarhus University

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Merjan Family Investigator

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Victor M. Luna, Ph.D.
Temple University

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New York University

Young Investigator
Xinzhu Yu, Ph.D.
University of Illinois Chicago

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The Pfeil Foundation Investigators

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Marjolein E.A. Barendse, Ph.D.
Erasmus Medical Center - University Medical Center Rotterdam

Young Investigator
Daniel S. Barron, M.D., Ph.D.
Brigham and Women's Hospital, Inc. - Harvard University

Young Investigator
Jennifer J. Donegan, Ph.D.
Dell Medical School, University of Texas at Austin

Young Investigator
Anna J. Finley, Ph.D.
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Natalie Cizik
Jill Cochran
Alison P. Cohen
Dr. Thomas B. Coles, Jr. and Mrs. Nancy A. Coles
Kellie I. Cook
Cookie
Shawna Copeland
Courtney S. Crane
Colin J. Crawford
Andrew M. Curry
Joe D.
DEAD RKGK
Stephen P. Dean
Jeffrey DeBono
Zac Deckard
Burak Deniz
Amit Desai
Bill DeSiena, LCSW
Dokibird
Patrick Doncov
William Duffy
Quan Duong
Lauren R. Duran
Eastside Management
Dr. Christopher Eginton and Mrs. Lindsay Eginton
EJ Eisenberg
JR Eisenberg
Lee Eisenberg
Chris Ellis
Hande Erçel
Matt Festa
Kristi A. Fitz
Judith Fleschner
Lajpat R. Gandhi, M.D., MB, BS
Emilie J. Gannon
Irving Getz
Joseph and Sarah Goodman
Darlene Graham
Thomas D. Green
Bryan M. Greene
Sarina Gwirtzman
The Hagen Family
Layla Hamedi
Bruce Harlow, Sr. and his unconditional love for his three children
John Kennedy Harrison II
Eric and Madeline Hauser
Zachary Hendricks
Hize'NLowz, LLC
Natalie Hoague
Dr. David P. Hoffman and Mrs. Susan M. Hoffman
Jacob Hoffman
Mark Holloway

Honor Tributes (continued)

Nicola and Erin Holloway
Keith Hopkinson
Paul Horelick
Samuel Hurley
Angela Hurtado
Kenneth Hyacinthe
Louis Innamorato, C.P.A.
Bob Jackson
Julia and Chris
Mariel Kagan
David Kahl
Jared W. Kaplan
Karen's birthday
Joe Karner
Robert Karner and Mary Chung
Miriam E. Katowitz, C.P.A.
Joel and Elinor Katz
Ken
Kerry
Christopher Kilbourne
Peter Kirk
Brian Kirkpatrick, M.D.
Nicholas Kirkpatrick
The Kirsics: Michael, Lisa, King Khaya and Chloe Superstar
Elisabeth D. Kline's beloved children
Jack Lee Knicely
Alexander Kocinsky
Karl F. Korpela
Howard and Shelley Kristol
John H. Krystal, M.D.
Jaren M. Kuester
Mr. and Mrs. Moon Kwok Kwong
Danielle D. Ladensack
Ian Laidlaw, LCSW-R
The Laitman Family
Skylar Lake
Martin Langer
Sarah Leonard-James
Jill D. Lewis-Spector, Ed.D.
Lisa
Lisa's birthday
Kelsey Loughlin
Daniel Maahs
Jeff Macdonald
Maci's birthday
William Maier
Robert Malone
The Winner of the Manifold Charity Poker Tournament
J. John Mann, M.D.
John D. Marangiello
Jared and Carley Margolis
The Maybank Family
Ian M. McElheny
Teresa McInerney
Jeffrey McKnight
Patrick McWilliams
Ingrid Mesa-Gibbons
Michael
Timothy Miller
The Morisi Family
Andrew L. Morrison, M.D.
Melissa Mortensen
Anne K. Muellerleile
Grace Nagaur
The Recipients of the Navient Team Leadership Award
Natalie Newcamp
Tiffany Nguyen
Nita's birthday
Diane M. Novellino
Paul O'Connell
Ella Orloff
Nieves A. Ortiz
Mary Pat and John Osterhaus
The Osterhaus Family
Gloria Palma-Olsavsky
Patrick W. Parker
Sallie M. Parker
The Patient
Dr. David Penn-Soskin
Eleanor Perry-White
John and Brittney Pfafflin
Alberto Piazza and Ada Ruata
Patricia H. Porter
Robert M. Post, M.D.
James and Donna Potter
Paul Pozniak
Stephen Quentzel, M.D.
Ryan T. Rauscher
Katherine (Kat) R. Raveling
Madeline Reed-Johnston
Andy Reynolds
Min J. Ro
Kimberly A. Rock
Harry Rollings
Faith Rothblatt
Michael R. Rothman
Ann Royall-Bolton
Tyler J. Rutherford
Mary C. Salpukas
Mark A. Satterthwaite, M.D.
Sawyer
Charles Scharfman
Dr. Brett Shurman
Dr. and Mrs. Mark Silver
Geoffrey A. Simon and Andrea R. Graham
Helen B. Simpson, M.D., Ph.D.
Arron N. Smith and Sean D.O. Smith
Eugene (Gene) Smith
Grace Smith
Richard and Sue Smith
Kathryn A. Snow, LCSW
Kenneth H. Sonnenfeld, Ph.D., J.D.
Alan J. Spandorf
Kathy Stephens
Fiona Stewart, Ph.D.
Greg Stickel
Jean L. Stone
Gregory Strauss, Ph.D.
Dr. Darcy Strobel
Sarah Stroh
Peter J. Tarr, Ph.D.
Taylor
The King
James C. Thies
Kristen Tizon
Arlene Tracy
Alex Tramiel and Sonia Miller
John Tyler and Caroline Kelly
Unexplainable (the podcast)
Jasmine S. Van Regenmortel
Juliana Venegas and Brent McBride
Michael Vollmar, M.D.
Les Wallin
Jennifer Ward
The Wei Family
Robert and Frances Weisman
The Wells Family
Dr. Austin J. Wertheimer and Ms. Caryl I. Goodman
The White Family
Brooke Wilken
Mike Yamanaka
Michael Yelenosky
Young Basic Science Researchers
Alex Zeigler
Stuart Zeigler
Zachary Zelman
Howard L. Zenker
Raleigh Zimmerman

Memorial Tributes

A loved one
Kathleen (Kathy) M. Abaray
Sue Abiad
John (Jed) B. Abrams
William F. Abrams
Alexis (Lexi) N. Adams

Memorial Tributes (continued)

Cecilia J. Adamski
Joyce Adelman
Aria N. Albertelli
Allison J. (Smith) Alfonso
Michael Alice
Darryl Allen
Janice M. Allison
Adrienne L. Anderson
Susan (Sue) K. Anderson
Angel
Edward Arvey
Zerqa Atiq
Christopher J. Awn
John J. Awn, Jr.
Arthur Aylem
Margaret R. Bachman
Gregory T. Bak
Kiara Baker
Anna Balek
Scott Balgemann
James (Jim) F. Balistreri
Mary Jane (Hauersperger) Banta
Cynthia L. Barazowski-Kassel
Neil Gregory (The Warrior) Barber
Richard (Ricky) B. Barfuss
Rebecca Bartlett's beloved family
Scott R. Bartlett
Stephen D. Bartusiak
Judith A. Baskin
Brian A. Bass
Leroy Baumgartner
David Beckel
Patricia (Pat) A. Bell
Jim Bennefield
John Bennefield
Chester Charles Bennington (Linkin Park)
Benny
Angelique N. Benrahou
Timothy J. Berchin
Dr. Deepak Bhasin
Stephen J. Binder
Victor Biondo
Dorothy F. Birdwell Maughan
Phyllis J. (Kratovil) Birk
Heather A. Bleick
John H. Blomquist
Kirk D. Boise
Oliver R. Bolger
Dennis L. Bond
Joanne M. Borkowicz
Betty Jane Boydstun
Dustin (Bo / Dbo) W. Bozarth
Michael C. Bragg
Robert Branch
Amy J. Brandl's beloved brother
Randall Brandt Smith
David Breuer
Elise Bridwell
Catherine (Cathie) A. Brodie
Reina Brody
Joan Y. Brooks
Daniel S. Brown
Elyce A. Brown
Beverly Brownlee
Bruce Brownlee
Corey A. Brunk
Elaine C. Bryant
James C. Bryant
Walter W. Bundschuh
Peter R. Bunning
Dorothy L. Burgess
Colleen M. Burke
Evan P. Busbey
The Butler Families
Patrick M. Caires
Brent M. Cameron
Tyler J. Cannon
Susana Cardenas
Dennis J. Carlton
Jeffrey T. Carlton
Em (Scout) Marie Carr
Tara L. Carrilli-Johnson
Richard M. Casey
Suzanne L. Castellani
Cynthia (Cindy) M. Cates
Catherine
Victoria E. Catral
Mary T. Cavanaugh
Adam R. Cavender, M.D.
Cynthia L. Chabay-Rosen
Patrick J. Chaloux
Dale R. Cherney
Matthew Cherney
Vizia Chinthala
Allison A. Chlan-Fellner
John A. Chomis
Andrew S. Christie
Kevin A. Christoffers
Heather Christopher
Kristina Cicoria, M.D.
Anthony (Tony) Cifelli
Jenna Clemenz
Justin W. Cline
Valerie L. Collar
Alexander B. Conley
Cynthia Cook
Ryan J. Cook
Grace Cook-Blasingame
David Cooper-Claire
Jordan Cooper-Halpern
Cara E. Corin
Cameron M. Cosentino
Christopher M. Cowan
Prudence Cox
John R. Craig
Jacob W. Crowder
Joan (Joanie) M. Curtin
Cynthia Dalao Bagatelas
Colter Dallman
Richard D. Daly
Cynthia (Cindy) A. Dankberg
Ross M. DaPrato
Sarah N. Darby
Dan M. David
James (Jim) L. Davidson III
Greg Davis
Terrence (King Terry) A. Davis
Decker J. Davison
Dr. Margarida Magalhães Dederick
Joseph Dell
Tyson J. Dent
Henry (Hank) J. Deutschendorf II
Nathaniel J. Dickinson
John Dilanni
Brandon Dixon
Nicholas (Nick) A. Dobbert
Una G. Donnelly
John Donovan
Stephen G. Doochin
August (Gus) E. Doskey
Loraine (Lory) J. Dryden
Michael Ducate
Michael A. Ducate
Jack E. Duffy
Dylan J. Edwards
Mark D. Edwards
Ronald (Ronnie) O. Edwards
Major Lawrence J. Elliott (USAF Retired)
Paula J. Erdelyi
Erica
Catherine (Catie) A. Etier
Marian (Molly) P. Everdell
Terri Everett's Home
Erik S. Fallahi
Garrett R. Farr
Alex D. Farren
Jan A. Fawcett, M.D.
Nathan Feinstein
Dorothy B. Feit
Hettie Feldman
James (Alec) A. Fentum

Memorial Tributes (continued)

Catherine Finn
Helga Fischbach
Nicholas D. Fischer
Ruth H. Flick
Raymond J. Flood, Jr.
Edwin (Ted) H. Flowers
Kathleen Foglino
Helen C. Fontana
Jacob J. Fox
Frank
Astral (Andrew) B. Freeman
Gail W. French
Robert (Bob) O. Friedel, M.D.
H. Bruce Friedrich
Rose Marie Friedrich
Jason C. Frisco
Jerrice A. Fritzlen
David Fuller-Cowles
Carl Fulmer
Stanton E. Gagel
Diane N. Galba
Mary Gallagher
Daniel (Danny) L. Gallegos
Ines Garcia
Erika N. Garlisch
Richard V. Garrett
Olive (Lehmer) Garrett Frye
Susan Getz
Steven H. Gibson
Erin M. Gilmer
Wendy A. Giurleo
Daniel J. Glorioso
Isabella (Izzy) Glorioso
Brian D. Go
George Goldman
Rochelle B. Goldman
Dr. Patricia Goldman-Rakic
Zachary Golub-Rosen
Ethel G. Gordon
Francey Gordon
Ted A. Gorman
Ellen Gottfried
Grace
Adam J. Grainawi
William F. Grant, Sr.
Andrew N. Gray, Ph.D.
Jacob A. Gray
Norma Gray
Susan J. Gray
Michael (Mike) J. Gredlein
David E. Green
Rose K. Greenberg
Andrew J. Greenfield
Mark J. Greenlee
Mark Greenwood
Matthew J. Gregory
Noah J. Grimes
Joshua E. Grossman
Wilfred L. Guerin
Viola M. Gugliuzza
Thomas S. Gumport
Amanda G. Gurley
John B. Haldeman
Matthew A. Hall
Stephen B. Ham
Kuang-Hwa Han
Ruth Handleman
James (Jim / Jamie) V. Hanna II
John P. Hanus
Matthew (Matt) J. Hardin
Jason R. Hargarten
Andrew Harrison
Dr. William L. Harrison
Cynthia (Cindy) M. Harwell-Paar
Deborah Hawkins
Norma (Levenson) Haynes
Larry Hays
Kevin J. Heald
John A. Heather, M.D.
Bradley R. Heilman
Gary E. Heussler, Ph.D.
Margaret C. Hibbitts
John Hickey
John J. Hoffman V
David J. Holien
Corinne H. Holt
Dr. Philip S. Holzman
Mark J. Hovis' best friend
Rong Huang
Edelgard Hubbauer
Rennie E. Hughes
William (Billy) W. Humphrey
Alan N. Hundert
Richard (Rich) W. Hungate
Darrell K. Hutchins
Irene (Renie) Hutt
Bessie L. Iglehart
Naveed A. Iqbal
Sandra J. Israel
Vivian B. Jackson
Marie Jana's beloved nephew, Brandon
Leslie (Les) W. Janski
Maria C. (Basdekian) Jarusinsky
Jimmy
Michael D. Johnson
Ryan P. Johnson
Ryan B. Joiner
Herbert (Skipp) R. Jones, Jr.
Savit Joshi
Allison Judd's beloved grandma
Andreas (Dre) P. Kahan
Leif Kamara
Mehul Kamble
Michael D. Kamp
Albert W. Kaplan
Gary A. Kaplan's departed grandparents, aunts, uncles, cousins and friends
Jeannette Kaplan
John R. Karatheodoris
Steven M. Karp
Melissa N. Karpf
Kevin J. Kassel
Claude R. Kaufman
Phyllis Kazik
Brian J. Kelly
Tyler S. Kelly
Michael F. Kenigsberg
Kenneth
Patrick (Stacy) W. Kenny
Marci Kerben
James (Jim) J. Kerrigan, Jr., M.D.
Lucas R. Ketelsen
Kevin
Gary D. Kiefer
Paul Killea
Jonathan S. Kinsley
Jill A. Kirby
Brian Kirschner
Steve Knitzer
William F. Koch
Allison (Alli) S. Kohl
Robert Kolozsvary
Steven Korn
Julie A. Kounce
John B. Kramer, Jr.
Olivia L. Kresach
Joseph J. Kroepil, Jr.
Rebecca J. Kruger
Scott Kruse
Michael Krusell Nelson
Kendal (Ken) A. Kucera
Ivan A. Kurcsinka
Gary Kushman
Murray (Murry) Kusmin
Keith Kussman
Shirley A. Kussman's beloved son
Morris Laitman, Ph.D.
Carole Jean Laping
Mary Ann Lapinskas
Misty D. LaRue
Jenna R. Laubach

Memorial Tributes (continued)

Brendan L. Lauderdale
Robert E. Lauer
Paul T. Laun
Henry R. Laveran
Alexander (Alex) P. Lawlor
Edward J. Leahy
Harry R. Leeds
Linda G. Leeds
Thomas (Tommy) J. Leonard
Allison Leong
Jorie Lester-Mark
Stephen (Steve) E. Librizzi
Jacob Lichtenstein
Constance E. Lieber
Samuel A. Lieber
Stephen A. Lieber
Anna Y. Li-Harezlak
Lance Lipton
Frank J. Lodanosky
John (Jack) S. Logue II
Danielle M. Lorenz
Victor G. Lottmann
Betty (Splawn) Lowder
James (Jim) E. Lowder
Joanna Lowry
Thomas A. Luby, Jr.
Gavin Lundin's beloved mother
Barbara Lyons
Matthew C. Lyons
Marissa N. MacLaughlin
Ann Marie Madigan, M.D.
Roman Makuch
Earl P. Malarchick, Ph.D.
Robert T. Malison, M.D.
Merrill M. Manning III
Marco
Alan G. Marer
Dr. Peter Marks
Matthew Marshall
Tara L. Martabano
Elizabeth (Beth) A. Martino
Martyred Jews and those Jews that died in the Holocaust
Dolores Massey-Healy
Michael D. Mattoon
Thomas Matye
Travis Matye
John K. Maxwell, Jr.
Kate McAllister Neely
Michael P. McClain
June E. McDonald
Mark McDonald
Kevin M. McLaughlin
John (Mr. Mc) P. McManus
William Cody Meeks
Abigale O. Merrifield
Daniel Michaels
Apphia Michelich
Ronald Michelich
Mary Ruth Middlebrook
Christopher F. Miehlisch
Andrew (AJ) J. Miller
Lori Miller-Levine
Duncan N. Mitchell
Neil Molberger
Gregory A. Monk
Juan (Joji) Maniquis Montelibano
Jeffrey D. Moon
Irene Moran
Richard Moran
Christa M. Mordino
Frank Morgan
Matthew P. Morgan
Isabelle Moua
Patrick Rory Muldoon
Ronald P. Mullan
Ronald (Ronnie) Mullan
Wyatt L. Murchison
Barbara Nachbar
Martin Nachbar
Paul Nachbar
Lori A. Nahas
Narayan K. Nannaware
Adam Neely
Dale Neilson
Barry J. Nelson
Karen S. Nelson
Paul Nelson
Christopher (Chris) J. Neubecker
Douglas A. Newman
David Nicholas
Emma (Emmie) Kajsa Marie Nicholas
Richard (Toby) W. Nichols, Jr.
Andrew Niskala
Richard (Dick) A. Nunis
Kelly O'Malley-Coogan
Greg Ochs
Emily Odle
Katherine (Katie) R. Olin
Cameron R. Orr
Barbara W. Page
James D. Palmer III
Albert L. Pan
Jennifer Y. Paragano
Herbert Pardes, M.D.
David W. Park
Grayson M. Parker
Josiah E. Parker III
Mark Parr
Georgiana A. (Thorpe) Pasela
William (Billy) F. Paul III
Arthur Peck, M.D.
Randy A. Perdon
Judith Pestronk
Seymour Pestronk
M. Hunter Phillips
Nan E. Phillips
Parker E. Phillips II
William Brodie Phillips
Reed Pickus
Sybil Pierce
Jessica A. Piesman
James (Jim) Piskule III
Aaron R. Pixley
Genesis Pool
Dr. Alex Porter
Brad Powers
Sean P. Pryor
Peppino Puleo
Matthew P. Purcell
Joel M. Purrington
Dylan Quast
Ramez E. Qureshi
Andrew Radcliffe-Watts
Luca Rader
Arthur J. Radin, C.P.A.
Evelyn R. Rafferty
James P. Rafferty, Sr.
Jane (Janie) E. Ragel
Sidharth Ramakrishnan
Christine M. Rancatore
Steve Ratner
Cassandra (Cassie) N. Ray
Anthony M. Razziano
Russell J. Razziano
John Reason
Steven (Stevie) R. Reid II
Vito Rella
Jesse I. Remez
Samuel (Sam) D. Rendall
Michael Reschke
Chris Reuter
Dr. Ernie Rhamstine
John J. Rice, M.D.
Paul F. Richards
Caasi A. Rich-Vogel
Christopher Ricker
William L. Ricketts
Priscilla Riddick-Trotter
Dorrit L. Rippchen
David A. Rock

Memorial Tributes (continued)

David P. Rogers
Brian Rorick
Thomas Rorick
Alan G. Ross
Christina (Chrissy) Rossi
Matthew S. Rothman
Kevin Rubinstein
Irwin Rutland
John G. Ryan
Patricia (Patty) L. Ryan
Edward (Eddie) M. Saenz
Edward Salvucci
Carol Sanchez
Anton E. Schanz
Terry Scheffler
Delbert (Del) J. Scheid
David L. Schmidlap
Lee R. Schoolmeesters
Karen L. Schuetz
Nora E. Schuster
Ellen Schusterman
Jeffrey A. Schwartz
Judith Schwartz
Norma Schweitzer
David J. Scott
Kelly M. Scruggs
Michael Seeds
Ezra L. Seegull
Kerry W. Self
Kenneth F. Selig
Timothy B. Sennott
Gary S. Sevitsky
Ratilal C. Shah
William D. Shannon
Daniel Sharkey Esrick
Megan L. Sharp
Conor F. Sharpe
Harriet Shetler
Sylvia Shick
Benjamin L. Silver
Rochelle (Shelly) Simms
Marcia Simon-Kaplan
Kurt Simpson
John T. Sinnott
Helen Siporin
Marianthi (Bavas) Skiadas
Patrick D. Skosnik, Ph.D.
Lilia Slodownik-Dalkoff
Edmund Smith
Edward J. Smith
Jeff C. Smith
Robert (Bob) H. Smith
William H. Smith II
Ann Smith-Sovey
James (Bert) Snider
Candace Snyder
Donald E. Snyder
Joseph (Drew) A. Sobotka
Marilyn (Lynn) J. Sobotka
Sierra Soler
Betty Jean Sonny
Michael J. Sonny
Chris E. Sorensen
Kelly L. Sorensen
Nathan A. Soukup
Gena Spaulding
Brad M. Speiser
Jeffrey T. Sramek
Doretha St. Clair
Ian J. Stancato
Brian A. Stapleton
John (Jack) A. Stapleton
Gregory L. Starling
Tyler R. Starling
Nathan B. Steinbach
Edward G. Steinmetz, Jr.
Robert Steinpreis
Marlene H. Steiskal
Kolne M. Stella
Florence Stern Weisman
Amy M. Strahan
Alfred F. Strakshus
Steven (Scott) Stripling
Mary J. Strub-Caulkins
Ruth S. Sweeney
Andrew Taddy
Cheryl J. Tallant
Melanie D. Tannenbaum
Dino B. Taraschi
H. Vonn Taylor
Lyle Taylor
Nancy Teas-Hardie
William Theos
Thomas
Camilla (Cammie) L. Thompson
Susan Thorpe
Yolanda E. Thorpe
Patrick C. Tierney
Zachary (Zach) Tolchin
Troy Torres, Jr.
Leanne Townsend
Mario A. Tozzi, Jr.
Victoria (Tori) Trang Cook
Jeffrey S. Trott
Virgina Trunzo
Donald Trybula
Alexandros Tsaoussis-Maddock
Amanda E. Tucci
Robert (Bob) T. Tucker
Mackenzie E. Tynan
Logan Upton
Maple Valiente
Brett A. Van Vort
Corey R. Varga
Charles Varkoly
John M. Vergo
Alysia P. Vesley
Harriet Vicente
James (Jim) E. Vlach
Kurt J. von Boeselager
Gregory Von Burg
Paul Von Burg
Wm. F. Wagner, Jr.
Capt. Anders (Andy) G. Wallin II
Tordis Wallin
John (Jack) Wallisch
Catherine (Katie) R. Walsh
Thomas Walsh
Sharon Wang
Robert (Bob) L. Waterhouse, Sr.
Susan Weems
Sifei Wei
Colleen E. Westbrook
Robert M. Wetzel
Michael G. Wieman
Jon K. Wilbur
Judith (Judy) Wildfeuer
Gertrude Wildgruber
Robert (Bob) L. Wildt
Mark Williams
Wesley Willis
Betty J. Winberg
Barbara A. Winkler-Monsanto, M.D.
Douglas Wistner
Farrell J. Wolfson
Ryan M. Woodland
Robert T. Woods
Surekha Yadawad
April A. Yahiro
Mary Ann Yahiro
Catherine (Kitty) L. Yelenosky
Kenneth L. Yocom
Anna Zarski
Mary Katherine Zartman
Peter W. Zartman
Jieru Zhao
Edward N. Ziegler
Gladys P. Ziegler
Barry L. Zimmerman
Joseph (Joe) J. Zito
Stan Ziubrzynski

Financial Summary*

Consolidated Statement of Financial Position

		DECEMBER 31, 2024	DECEMBER 31, 2023
ASSETS			
Current Assets			
Cash and cash equivalents		$38,084,529	$26,905,317
Investments, at fair value, current portion		11,991,311	14,629,688
Contributions receivable		96,425	415,480
Pledges receivable, current portion		4,895	7,470
Prepaid expenses and other assets		302,557	230,236
	Total Current Assets	**50,479,717**	**42,188,191**
Pledges receivable, net, less current portion		9,738	9,738
Assets held in charitable remainder trust		2,300,206	1,885,836
Fixed assets, net		8,085	9,684
Investments, at fair value, less current portion		8,947,449	8,947,449
Right-of-Use Asset		716,139	125,457
	Total Assets	**$62,461,334**	**$53,166,355**
LIABILITIES AND NET ASSETS			
Current Liabilities			
Accounts payable and accrued expenses		$52,931	$75,069
Grants payable		15,014,698	14,887,604
Operating lease liability, current portion		342,373	148,179
Accrued compensation		71,023	48,077
Annuities payable		970,084	826,641
Charitable gift annuities payable		12,780	13,599
	Total Current Liabilities	**16,463,889**	**15,999,169**
Operating lease liability, net, less current portion		528,260	—
	Total Liabilities	**16,992,149**	**15,999,169**
Net Assets			
Without donor restrictions		34,274,112	26,492,489
With donor restrictions		11,195,073	10,674,697
	Total Net Assets	**45,469,185**	**37,167,186**
	Total Liabilities and Net Assets	**$62,461,334**	**$53,166,355**

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

Consolidated Statement of Activities

		YEAR ENDED DECEMBER 31, 2024	YEAR ENDED DECEMBER 31, 2023
SUPPORT AND REVENUE			
Contributions		$15,589,932	$14,613,749
Special events, net		42,878	89,157
In-kind contribution of services		2,054,955	2,048,285
Bequests		3,472,060	5,022,129
Net realized and unrealized gains on investments		3,698,514	3,929,558
Net appreciation of assets held in charitable remainder trusts		414,370	184,399
Dividend and interest income		1,462,573	908,154
	Total Support and Revenue	**26,735,282**	**26,795,431**
EXPENSES			
Program Services			
Research grants and awards		10,772,382	9,232,426
Scientific advancement		2,397,371	2,309,627
Program support		2,513,497	2,352,532
	Total Program Services	**15,633,250**	**13,894,585**
Supporting Services			
Fundraising**		989,718	897,389
Administration**		1,810,315	1,629,752
	Total Supporting Services	**2,800,033**	**2,527,141**
	Total Expenses	**18,433,283**	**16,421,726**
Change in Net Assets		8,301,999	10,373,705
Net Assets, beginning of year		37,167,186	26,793,481
	Net Assets, end of year	**$45,469,185**	**$37,167,186**

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

BRAIN & BEHAVIOR
RESEARCH FOUNDATION
Awarding **NARSAD** Grants

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