BRAIN & BEHAVIOR RESEARCH FOUNDATION

2025 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,

We are pleased to report that the Brain and Behavior Research Foundation (BBRF) has increased its funding of mental health research grants. Due to major shifts in government research funding, BBRF has increased the number of Young Investigator Grants by an additional 10%. BBRF is nimble and able to take bold action because it receives no government funds. BBRF remains the world's largest private funder of mental health research grants. It is the solid support of BBRF's generous donors that makes this possible. **Philanthropic support for mental health research has never been more essential.**

In 2025 the Brain and Behavior Research Foundation awarded more than $12 million to support research across a wide range of brain disorders. Since 1987, BBRF has invested $476 million in grants to more than 5,700 scientists worldwide—each dedicated to discovering better treatments, cures, and methods of prevention for mental illness.

Throughout 2025, BBRF-funded breakthroughs were published in leading psychiatric and medical journals. This annual report highlights 15 *Leading Research Achievements by BBRF Grantees, Prizewinners & Scientific Council Members for 2025*—advances that underscore the Foundation's vital role in improving the lives of individuals and families affected by mental illness.

Every BBRF grant recipient is selected by our distinguished Scientific Council, a group of 191 world-renowned scientists who rigorously review each application to identify the most promising and innovative research. This past year, the Council evaluated more than 1,000 grant proposals, up 12% from 2024. It awarded 165 Young Investigator Grants—supporting two years of research at $35,000 per year—and 10 Distinguished Investigator Grants, each providing $100,000 for one year of research.

In July, BBRF hosted its annual *Scientific Council Dinner* where we presented the prestigious Klerman & Freedman Awards. These prizes recognize exceptional clinical and basic research conducted by BBRF Young Investigator Grantees. They represent the future of mental health research that is at the heart of BBRF's mission.

In October, BBRF held its *International Mental Health Research Symposium*, where our five Outstanding Achievement Award winners shared their innovative research in schizophrenia, bipolar disorder, pediatric mood and anxiety disorders, and cognitive neuroscience. All symposium presentations are available to view on the BBRF website: https://bbrfoundation.org/event/international-mental-health-research-symposium

Later that evening at the *Annual Awards Dinner* BBRF recognized recipients of the 2025 Outstanding Achievement Prizes, along with the Pardes Humanitarian Prize in Mental Health.

BBRF continues to share the latest advances in mental health research through *Brain & Behavior Magazine*, as well as in our weekly "*eNews*" newsletter, which is available free of charge to donors, the scientific community, and the public. These platforms highlight important discoveries from BBRF grantees, awardees, and Scientific Council members.

The Foundation's free monthly webinar series, *Meet the Scientist*, connects audiences with leading global experts who present their latest findings in brain and behavior research.

BBRF also produces the award-winning public television series "Healthy Minds," broadcast on PBS stations nationwide and also available online at pbs.org. The program delivers valuable insights into psychiatric conditions, treatments, and emerging scientific breakthroughs. Season 10 is currently available to watch online at bbrfoundation.org/healthy-minds-tv, and Season 11 is expected to premiere on PBS this fall.

In addition, BBRF is proud to continue to support several initiatives aimed at strengthening mental health awareness in journalism and public discourse, including The Carter Center's Mental Health Journalism Fellowship Program and Mental Health Parity Newsroom Collaborative, The Poynter Institute's Mental Health Reporting Project, and MindSite News.

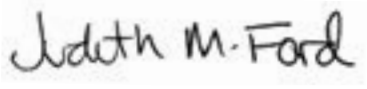
We are continually inspired by the scale and impact of the discoveries made possible through the support of our generous donors. Our shared vision—a world free from the burden of mental illness—depends on the partnership between dedicated donors and the exceptional scientists selected by the BBRF Scientific Council. While meaningful progress continues, much work remains ahead.

As always, 100% of your contribution for research directly supports research, as our operating expenses are covered by separate foundation grants.

At a time of heightened uncertainty in mental health research funding, your steadfast commitment is more important than ever. Together, we will continue to advance innovative research that leads to new discoveries, better treatments, and lasting hope for those affected by mental illness.

Sincerely,

Jeffrey Borenstein, M.D.
President & CEO



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

Geoffrey Simon
Chair, Board of Directors

2025 **LEADING RESEARCH ACHIEVEMENTS** BY FOUNDATION GRANTEES, PRIZE WINNERS, & SCIENTIFIC COUNCIL MEMBERS

Proof-of-Concept Test for an RNA-Based Therapy to Prevent Effects of a Devastating Autism-Related Gene Mutation

Next-Generation Therapies: Autism, Schizophrenia, Neurodevelopmental Disorders

Story Highlights:

Researchers have reported progress in developing an RNA-based therapy (using "antisense" technology) to treat neurodevelopmental disorders, including some autism spectrum disorders (ASDs).

Journal:
Molecular Therapy
March 5, 2025

Researchers led by a BBRF grantee have reported progress in developing an RNA-based therapy to treat neurodevelopmental disorders, including some autism spectrum disorders (ASDs).

RNA, like DNA, is a fundamental biological material. In the nucleus of cells, messenger RNAs (mRNAs) carry instructions encoded in DNA to cellular factories outside the nucleus called ribosomes, which assemble the many thousands of proteins that are the basis of all living structures and which perform myriad roles in living organisms.

There are many other types ("species") of RNA that perform indispensable roles in our cells but are not directly connected to protein synthesis (these are "non-coding" RNAs). Among these are various kinds of RNA molecules that help to regulate how, when, and where genes are expressed.

The new research, appearing in the journal *Molecular Therapy,* reports efforts by a team led by **Marta Biagioli, Ph.D.,** a 2015 BBRF Young Investigator at the University of Trento, Italy, to design and synthesize a long non-coding RNA (lncRNA) molecule able to increase protein production associated with a target mRNA. This molecule—referred to by an acronym, SINEUP—was then tested in experiments to provide "proof of concept" for its therapeutic application in neurodevelopmental disorders caused by mutations in a gene called CHD8.

Among the many gene mutations implicated in ASDs, those occurring in CHD8, while affecting a relatively small number of patients, are among the strongest ASD risk factors. When present in an individual, CHD8 mutations can have a catastrophic impact, by causing a loss of function in the protein encoded by the CHD8 gene. Although we have two copies of every gene in the nucleus of most of our cells, in some cases, including that of CHD8, when one copy of a gene is mutated or missing, normal function is not possible (not enough of the protein is made). In the case of the CHD8 gene, two functioning copies are, in fact, needed to generate the required amount of protein for normal cellular physiology.

The pathology associated with disabling CHD8 mutations or deletion of one copy of the CHD8 gene causes dysfunction in gene expression, specifically, the process by which the information in genes is transcribed into mRNA. In the emerging brain, this results in alterations in the development of neurons, with many possible clinical consequences, including macrocephaly, enlarged head size. These effects are seen in a subset of people with ASDs.

Dr. Biagioli's BBRF Young Investigator grant was devoted to developing an RNA approach to ameliorating pathologies caused by loss or dysfunction of one copy of the CHD8 gene.

Marta Biagioli, Ph.D.

University of Trento, Italy
2015 BBRF Young Investigator

The method employed by her team makes use of a synthetically constructed antisense lncRNA, called a SINEUP molecule. "Antisense" refers to a molecular sequence that is complementary to that of a specific RNA molecule in cells that one wishes to target. The portion of the lncRNA molecule that is "antisense" relative to a specific target mRNA makes it act like an extremely precise homing device—an excellent property for a prospective therapeutic molecule.

The expectation was that the binding of the SINEUP lncRNA to its target, CHD8 mRNA, would lead to increased production of the CHD8 protein. This approach is a way of addressing the insufficiency of CHD8 protein in the neurons of people with a devastating CHD8 gene mutation that disables one copy of the gene.

Dr. Biagioli and colleagues tested their SINEUP RNA in test tubes, applying them in human-derived neural progenitor cells that were created from human cell samples, using stem-cell technology. They were also tested in fibroblasts—cells found in connective tissue—that were similarly generated from human cell samples, in this case, from cells donated by ASD patients with CHD8 mutations. These cells had aberrantly reduced levels of the CHD8 protein prior to the test. Application of the SINEUP lncRNA had the effect of raising CHD8 protein levels one and a half times—levels thought to be sufficient to support normal function.

Other tests indicated that the SINEUP RNA molecule, once applied, could reverse dysregulation of the transcription process through which the message of the CHD8 gene is copied into messenger RNA format. Application of the SINEUP RNA also reversed changes in epigenetic regulation that are seen when the CHD8 gene is mutated. Finally, tests were performed in a living organism—in zebrafish, which are often used in tests of genetic technologies. Delivery of the antisense RNA into zebrafish embryos with reduced levels of CHD8 prevented enlarged head size, which had been previously reported in this animal model and resembling the impact of CHD8 mutations in people.

"In conclusion," the team reported, "our data provide experimental evidence toward the development of a state-of-the-art type of RNA-based therapy for neurodevelopmental disorders, with implications beyond the CHD8 syndrome and ASD, and with potential impact for a large repertory of currently incurable neurologic conditions caused by reduced levels of target protein."

Noting that "CHD8 plays a pivotal role during the early phases of human development," the team said that future research will include trying to determine the optimal timing for the delivery of antisense RNA molecules used in the CHD8 experiments. Such molecules, for ethical reasons, cannot be delivered in the human fetus. But the team suggests that a "key developmental window" occurring shortly after birth might provide a time to deliver antisense RNA therapies like the one they tested, with the chance of "still alleviating important ASD phenotypes [i.e., clinical manifestations]."

As the current paper reports a proof of concept, a good deal more experimentation in animal models will be needed before tests in humans of this or optimized versions of the SINEUP antisense lncRNA can be contemplated. ❖

Loss of Pleasure From High-Fat Food That Paradoxically Supports Obesity Is Traced to a Brain Signaling Pathway Involving Neurotensin

Basic Research, Next-Generation Therapies: Eating Disorders

Story Highlights:

New research helps explain a paradox: why individuals acclimated to high-fat food begin to devalue it—get less pleasure from it—yet continue to eat it and become obese. In mice, restoring neurotensin levels in a key brain pathway mitigated high-fat diet-induced changes in eating that supported obesity.

Journal:
Nature
March 26, 2025

Ready access to and excessive consumption of high-calorie foods are widely held to be key contributing factors in the development and progression of obesity. The link has been observed in people but also tested repeatedly in animals.

This research has confirmed that chronic exposure to a high-fat diet "profoundly influences eating behaviors," particularly those driven by the pleasurable (sometimes called "hedonic") properties of food. So note a team of researchers led by two BBRF grantees who have published in the journal *Nature* results of new research on the relationship between the functioning of the brain's reward system and obesity.

It was their aim to investigate how a chronic high-fat diet affects eating behaviors, one aspect of which seems to defy common sense. It has been observed in mice and people that after a period of time, individuals who have become accustomed to eating a high-fat diet begin to exhibit a reduced desire to consume or actively seek high-calorie or high-fat food. Just as curious, this observed tendency actually seems to contribute to the progression of obesity. How to explain this?

Seeking in mouse models to answer "the critical question of how continuous access to calorie-rich foods affects neural circuits involved in feeding and motivation," the team was led by **Stephan Lammel, Ph.D.**, a 2015 BBRF Young Investigator. **Neta Gazit Shimoni, Ph.D.**, whose 2021 BBRF Young Investigator grant was devoted to studying the regulation of hedonic, or pleasure-driven feeding, and its relation to obesity, and Amanda J. Tose, Ph.D., were co-first authors. All are at the University of California, Berkeley.

"The paradox is that we normally think that people with obesity tend to seek out foods that induce pleasure," Dr. Lammel explains. "However, our research suggests that these people actually may experience less pleasure from eating palatable foods. They do not eat less, but the way they eat has changed. They are not eating for pleasure anymore."

In both people and mice, dopamine-producing neurons in the brain's ventral tegmental areas (VTA) that connect to cells in the brain's nucleus accumbens (NAc) have been implicated in the motivational aspects of feeding behavior. Activation of dopamine neurons in the VTA that are connected with the NAc has been pinpointed as centrally associated with the reward that is involved in seeking and consuming food. Even the mere anticipation of reward has the effect of enhancing dopamine-cell firing, which in turn promotes behaviors in which both animals and people seek food rewards.

Conversely, however, past research in mice and people has indicated the

Stephan Lammel, Ph.D.

University of California, Berkeley
2015 BBRF Young Investigator

Neta Gazit Shimoni, Ph.D.

University of California, Berkeley
2021 BBRF Young Investigator

paradox: that chronic exposure to a high-fat diet reduces dopamine activity in this circuit, potentially impairing reward-related processes, but, as noted, actually contributing to obesity. Drs. Lammel, Shimoni and colleagues in past work have shown that by artificially stimulating the pathway connecting the lateral NAc and the VTA, mice could be driven to display robust reward-related behaviors. In the current research, they sought to learn about whether increased activity in the lateral NAc-VTA pathway is associated with hedonic feeding behaviors, and how these may be affected by diet-induced obesity.

The newly reported research demonstrates that chronic consumption by mice of a high-fat diet alters hedonic feeding behaviors and disrupts signaling in the pathway connecting the lateral NAc and the VTA. The signaling disrupted specifically involves neurotensin, a neuropeptide, or signaling protein, found in the brain and central nervous system. The change in neurotensin signaling was observed to have an impact on the progression of obesity.

The team's finding that neurotensin release from neurons in the lateral NAc decreases after mice have grown accustomed to a high-fat diet, in the team's view, "provides a circuit-level explanation for the link between dopamine activity and weight gain, altered feeding behavior, and obesity progression."

As they explain, neurotensin acts through the neurotensin-1 receptor on dopamine neurons to enhance their activity, leading to an increase in dopamine release in the NAc. Dopamine, importantly, is a key regulator of reward learning and motivated behavior. Thus, they say, a reduction in neurotensin release in the lateral NAc induced by a high-fat diet "is likely to diminish" excitation of dopamine neurons, which in turn "reduces the desire to consume high-calorie foods."

The new research may help explain the paradox in which such alterations actually contribute to obesity. "It may be challenging to comprehend," the team notes, why mice (or people) acclimated to rich food begin to devalue acquiring it—yet continue to eat it and become obese.

The answer is not yet clear, but may have to do at least in part with habit. "Obesity is often associated with reduced behavioral sensitivity to changes in the motivational value of hedonic food rewards, including habit-like behavioral control that encourages overconsumption of food," the team wrote. Other possibilities are that when the reward initially attached to rich food is diminished or devalued, this alters an individual's feeding habits and circadian rhythm. Also, it may involve changes in locomotion or reduced "exploration behavior," which may indirectly promote weight gain.

The newly reported research suggests that in mice, at least, restoring neurotensin levels in the lateral NAc-VTA pathway can mitigate high-fat diet-induced changes in hedonic feeding, anxiety, mobility, and food consumption. Each of these factors appears to have a role in obesity progression, the researchers said. "Reduced anxiety in particular may not only improve food consumption behaviors but also enhance overall mental health, which is highly relevant for treating obesity and its comorbidities."

"A high-fat diet changes the brain, leading to lower neurotensin levels, which in turn alters how we eat and respond to these foods," Dr. Lammel said. "We found a way to restore the desire for high-calorie foods, which may actually help with weight management." If this approach can be translated into humans, it could open new avenues for addressing obesity by restoring food-related pleasure in those who lose it, possibly altering unhealthy eating patterns. Such research is already under way in the Lammel lab.

Given the role of high-calorie foods "in driving the obesity epidemic," the team says "targeting neurotensin signaling in the NAc-VTA pathway may offer a promising strategy" to regulate food intake and support healthy weight maintenance—perhaps "without disrupting other essential functions" that are mediated by neurotensin, which in addition to feeding behaviors include the regulation of pain and body temperature. ❖

Team Modifies LSD Molecule to Capture Potential Therapeutic Effects While Limiting Hallucinations

Next-Generation Therapies: Schizophrenia, Bipolar Disorder, Depression

Story Highlights:

Researchers modified the LSD molecule to create a new drug called JRT that appears to have little or no hallucinogenic potential yet retains some of the potentially pro-cognitive therapeutic impact of some hallucinogens, including the potential to address the negative and cognitive symptoms of schizophrenia.

Journal:
Proceedings of the National Academy of Sciences (PNAS)
April 14, 2025

Some psychedelic compounds have been shown to be powerful promoters of growth in atrophied cortical neurons, a fact that has intrigued researchers interested in addressing one of the hallmark pathologies associated with schizophrenia.

Analysis of postmortem brains of people who suffered from the illness have revealed decreased branching of the dendrites that bring signals from other neurons into nerve cells in the cortex; reduced density of the tiny bump-like protrusions along dendrites that are the points of contact for axons projected by neighboring neurons; and abnormally low levels of the proteins that form synapses in cortical tissue. All are manifestations of cortical atrophy.

For various reasons, the pharmacological properties of psychedelics—their hallucinogenic properties—make these drugs particularly inappropriate for people with schizophrenia and psychosis. But the power of some of these same substances to promote neuronal growth in the face of cortical atrophy has inspired some researchers to search for ways to modify psychedelics so as to retain their potentially therapeutic properties and reduce or eliminate their hallucinogenic ones.

In a paper newly published in the *Proceedings of the National Academy of Sciences (PNAS)*, a team led by David E. Olson, Ph.D., at the Institute for Psychedelics and Neurotherapeutics at the University of California, Davis, reports that they have modified the LSD molecule to create a drug dubbed JRT. The new drug proved in a range of experiments to be "an exceptionally potent analogue of LSD," yet with much lower hallucinogenic potential and improved selectivity. The new drug appears to have "the ability to produce a wide range of therapeutic effects." A powerful promoter of growth among cortical neurons, JRT also had strong antidepressant properties in animal tests and showed potential to address the negative and cognitive symptoms of schizophrenia.

The team included three BBRF grantees: **William A. Carlezon Jr., Ph.D.**, a 2007 and 2005 BBRF Independent Investigator and 1999 Young Investigator; **Conor Liston, M.D, Ph.D.**, a 2013 BBRF Young Investigator; and **Alex S. Nord, Ph.D.**, a 2015 BBRF Young Investigator. The new paper's co-first authors were Drs. Jeremy R. Tuck and Lee E. Dunlap.

Schizophrenia's negative and cognitive symptoms are unaffected by both first- and second-generation antipsychotic medicines. (The newly approved schizophrenia drug Cobenfy has shown some effectiveness in reducing both negative and cognitive symptoms, although these properties are still under study.) Antipsychotic drugs block the D2 dopamine receptor in nerve cells and are often highly effective in reducing the positive symptoms of the illness including hallucinations

William A. Carlezon, Jr., Ph.D.

Harvard Medical School
McLean Hospital
BBRF Scientific Council
2007, 2005 BBRF Independent Investigator
1999 BBRF Young Investigator

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

Weill Cornell Medicine
BBRF Scientific Council
2013 BBRF Young Investigator

and delusions. Thought- and reality-distorting symptoms like these are among the principal effects of hallucinogens like LSD, making them especially dangerous for those with schizophrenia, psychosis, or a family history of either.

In theory, modifying a hallucinogen like LSD to be non-hallucinogenic could result in a compound that retains the molecule's growth-spurring and possibly pro-cognitive effects. Such a compound might then be an attractive candidate to address negative symptoms of schizophrenia, which include flat affect, reduced emotional expression, social withdrawal, and difficulty with motivation. Cognitive symptoms include difficulty paying attention, impaired working memory, slowed response time, difficulty handling verbal information, and difficulty switching between tasks.

Dr. Olson's team performed an astonishingly simple modification of the LSD molecule: they swapped the positions of two atoms in LSD to create JRT. Like LSD, the new drug specifically spurs activity at the serotonin 2A receptor. But the structural modification that generated JRT also reduced its potential to generate hallucinations, as both test tube-based and mouse-based experiments indicated.

Dr. Olson commented: "What I think is so interesting about this work is that JRT and LSD have essentially the same molecular shape and weight, yet they have distinct pharmacology thanks to the transposition of those two atoms."

The pharmacology of the various existing approved schizophrenia medicines is complex. But it appeared in the experiments involving JRT that it would not have side-effects associated with existing medicines such as weight gain or sedation. JRT proved to be a partial agonist, or stimulator, of the serotonin 2A receptor, possibly explaining its ability, on the one hand, to promote cortical neuron growth, but on the other hand, failing to cause behaviors in mice that indicate hallucinogenic impact. These include head-twitching behavior, hyperlocomotion, and deficits in prepulse inhibition, a measure of the brain's ability to filter out irrelevant sensory information.

"Despite its lower hallucinogenic potential," JRT in head-to-head comparisons with LSD and the antipsychotic clozapine "demonstrated superior effects on cortical neuron growth, [and] moreover produced a remarkable 46% increase in dendritic spine density" in living mice. Other experiments showed it "completely rescued cortical atrophy" in a particular layer of neurons in the mouse brain.

"These changes in structural plasticity were accompanied by robust antidepressant-like properties and pro-cognitive effects," in tests that included measuring active coping strategies in response to an unavoidable stressor. In a test of chronic stress, the drug reversed anhedonia-like behaviors (inability to seek pleasure). JRT also "promoted cognitive flexibility" in mice performing a "reversal learning task" (a test in which an individual learns to abandon a previously learned behavior and adopt a new one).

The team said JRT was an agent that "can potentially address negative and cognitive symptoms associated with schizophrenia without exacerbating positive symptoms or producing side effects characteristic of current treatments." Much more testing needs to be done, in animals, before human tests would even be contemplated.

More broadly, the researchers believe these experiments highlight the potential of modifying the chemical structures of some psychedelics "to produce analogues with improved efficacy and safety profiles," as in this case they appeared to have discovered a non-hallucinogenic stimulator of cortical plasticity and growth with potential to treat illnesses that cannot be addressed by psychedelics including schizophrenia, psychosis, and bipolar disorder.

Dr. Olson is a co-founder and head of the scientific advisory board of Delix Therapeutics, which was a funder, along with the NIH and other organizations, of the current research. Some other team members also have relationships with Delix and are holders of patent rights for JRT. ❖

Non-invasive Ultrasound Brain Modulation Therapy Shows Potential to Treat Mood, Anxiety, Trauma, Across Diagnoses in Pilot Trial

Next-Generation Therapies: Depression, Anxiety, PTSD

Story Highlights:

A pilot study of low-intensity transcranial focused ultrasound (tFUS)—a form of non-invasive soundwave-based stimulation that can reach deep inside the brain—showed safety and therapeutic potential across diagnoses in 29 patients with a variety of mood, anxiety, and trauma-related disorders.

Journal:
Molecular Psychiatry
April 24, 2025

Over the last two decades, non-invasive brain stimulation, especially rTMS (repetitive transcranial magnetic stimulation), has become a widely used therapy for psychiatric disorders, most especially depression. In recent years, rapid-acting versions have been successfully introduced to treat severe, treatment-resistant major depressive disorder, while in other applications, non-invasive stimulation has been tested to address other conditions, including PTSD and OCD.

In a paper published in *Molecular Psychiatry*, researchers led by **Gregory A. Fonzo, Ph.D.**, a 2019 BBRF Young Investigator at the University of Texas at Austin Dell Medical School, report on a pilot study of low-intensity transcranial focused ultrasound (tFUS), which they tested for safety and therapeutic potential in 29 patients with a variety of mood, anxiety, and trauma-related disorders, as well as in 23 healthy controls.

While rTMS uses magnetic pulses to alter the activity of cortical cells just beneath the skull, tFUS uses focused high-frequency soundwaves to reach areas of the brain that lie beneath the cortex—so-called subcortical areas. rTMS can affect subcortical structures such as the amygdala and hippocampus, but only indirectly, via connections forged by stimulated cortical cells with those structures. In tFUS, there is no cortical intermediary; focused sound waves reach directly into the subcortical brain and can be targeted with considerable precision.

The study performed by Dr. Fonzo and colleagues, who included **Charles B. Nemeroff, M.D., Ph.D.**, a BBRF Scientific Council member, 1997 Selo Prize-winner, and two-time Distinguished Investigator (1996, 2003), focused on tFUS's impact on the amygdala, a subcortical structure centrally involved in the processing of emotions. Hyperactivity in the amygdala is thought to be implicated in a range of psychiatric conditions.

The experiments, in addition to testing an application of tFUS technology, reflect an approach advanced at the National Institutes of Health that encourages researchers to think of psychiatric symptoms across diagnostic boundaries. Called RDoC, or Research Domain Criteria, it regards the amygdala, for example, as a key mediator of "all negative valence subdomains," i.e., brain areas involved in generating negative feelings that include perceptions of acute threat (fear), potential threat (anxiety), sustained threat, loss, and lack of reward. These constructs are interconnected and relate to responses to aversive situations or contexts, and some or all of them may be involved in various mood, anxiety, and trauma-related disorders.

Gregory A. Fonzo, Ph.D.

The University of Texas at Austin
2019 BBRF Young Investigator

The team's premise was: if tFUS is capable of safely and therapeutically modifying the amygdala, specifically in reducing hyperactivity in the structure, it could conceivably be used transdiagnostically—across diagnoses—as a form of therapy. Current therapies including SSRI antidepressants and psychotherapy may act across diagnoses to some important extent. But many who receive these and other therapies don't respond or don't respond fully or in a durable way. Hence, the continuing search for new approaches.

A single application of focused ultrasound ("sonication") has been shown to alter neurobiological function in monkeys which can last over one hour. These and other tests indicate that tFUS alters neuroplasticity, i.e., the ability of neurons to change the strength of their connections, one of the mechanisms though which antidepressants are thought to deliver therapeutic results.

A test of tFUS to inhibit neural activation in the amygdala across a range of mood, anxiety and trauma-related disorders had not yet been attempted. Dr. Fonzo and colleagues recruited 29 patients with such disorders, as well as 23 healthy controls. They conducted a double-blinded, placebo-controlled "target engagement study" in the 52 participants, designed to test whether tFUS could indeed modulate activity in a targeted area, the left amygdala. Afterward, they conducted an unblinded pilot clinical trial in which the 29 participants with psychiatric diagnoses received daily repetitive tFUS (rtFUS) over 3 weeks (5 treatments per week) targeting the left amygdala.

The "target engagement" tests were successful. In two sessions separated by one week, patients and controls received an active tFUS session and a placebo, or "sham," version. The treatment was guided by MRI, and effects were observed when the participants were receiving a functional MRI scan. These experiments showed that active tFUS (versus "sham") reduced activation in the left amygdala, while modulating connectivity between that area and interconnected limbic and prefrontal circuitry. There was considerable variability in the magnitude of such reduction among recipients of tFUS, a result to be taken up in future studies. These tests also established to the team's satisfaction that tFUS as administered was safe and "feasible as an intervention approach." No serious adverse events were reported.

Of the 29 participants in the unblinded pilot clinical trial, most of them in the early 20s, diagnoses were overlapping: 16 had been diagnosed with major depression; 10 with bipolar disorder; 4 with alcohol use disorder; 2 with panic disorder; 23 with an anxiety disorder; and 10 with PTSD. The primary outcome measure was the Mood and Anxiety Symptom Questionnaire—General Distress (MASQ-GD) scale, on which the average participant had a score of about 30 prior to the 3 weeks of tFUS treatments, and 21 following the treatment course, a reduction, the team said, that was statistically significant, despite the small size of the cohort.

"The pilot trial provides initial evidence of safety, feasibility and possible utility of daily rtFUS as a transdiagnostic intervention," the team reported. "We observed a significant reduction in our primary outcome, a general measure of negative-affect symptoms" in the disorders affecting the participants. "Following the entire treatment course the effect size [of the benefit] was moderate-to-large for the primary outcome as well as for several secondary outcomes, including depression and PTSD symptom severity."

The researchers say these results justify a much larger, double-blinded "sham"-controlled clinical trial. Such a trial would not have the disadvantage of the current pilot of lacking a control group—which makes it difficult to assess the possible benefit of the tested rtFUS protocol. Future tests might also explore the dosing—whether sessions 5 days per week are optimal, or if fewer sessions or a different treatment duration might be advantageous. There is also no data yet on the durability of the therapeutic effects of rtFUS, another subject for exploration in future tests. ❖

Sleep Disturbances in Preadolescents Predicted Increased Depression Severity, Self-Harm Behaviors the Following Week

Diagnostic Tools/Early Intervention: Depression, Childhood Mental Health

Story Highlights:

Over the course of a year, researchers obtained evidence that weekly self-reports of trouble sleeping in preadolescents predicted and preceded increased depression severity and engagement in self-harm behaviors the following week in those children.

Journal:
Research on Child and Adolescent Psychopathology
May 14, 2025

For the first time, researchers have obtained evidence establishing a potentially predictive connection between disturbances in sleep and the occurrence of depression and self-harming behaviors in preadolescent children.

Led by 2022 BBRF Young Investigator **Caroline P. Hoyniak, Ph.D.,** of Washington University, St. Louis, the team makes clear what is at stake in their study, in a paper appearing in *Research on Child and Adolescent Psychopathology*. Preadolescence is the transition period from late childhood to adolescence that not only coincides, typically, with the onset of puberty, but is also a time of physical and psychosocial development, marked by "rapid increases in capacity for emotional regulation," albeit in the context of increasing peer pressures and the growing need for autonomy. "This confluence of factors puts preadolescents at heightened risk" for psychiatric conditions, especially mood disorders, particularly among females.

Statistics show that depression significantly increases in prevalence during the childhood-to-adolescence transition. Importantly, child-onset depression is known to increase risk for a "chronic and relapsing course of depression throughout the lifespan." Experiences with self-injurious thoughts and behaviors (SITBs) often co-occur with mood disorders in preadolescence. For reasons still unclear, there has been a rise in the rate of SITBs in preadolescent youth across the last two decades, and suicide is now the second leading cause of death in youths aged 10-14. In preadolescents in whom depression and SITBs co-occur, symptoms tend to be more severe and there is increased risk for functional impairment later in life, including substance use problems and suicidal behaviors.

It is currently quite difficult to predict risk for episodes of depression or SITBs in youth, the team points out. One promising area of research in older individuals has focused on sleep disturbances as a risk factor and potential predictor of subsequent mood episodes. "Sleep disturbances" include trouble falling asleep and frequent waking in the night; sleepwalking and night terrors; as well as disordered breathing during sleep, short total duration of sleep, and daytime sleepiness.

"Given the burden of depression and self-harm behaviors in childhood," the team said, "it is critical to identify predictive factors associated with increased risk for recurrence, [and] sleep disturbances may be one such risk factor."

Noting that 20% of preadolescents experience sleep disturbances, as well as about three-fourths of all youth diagnosed with depression, the researchers set out to explore whether preadolescence might "provide a unique window for exploring the

Caroline P. Hoyniak, Ph.D.

Washington University in St. Louis
2022 BBRF Young Investigator

unfolding nature of the relationships between sleep, depression, and SITBs." Two BBRF Scientific Council members were senior members of the team: **Joan L. Luby, M.D.**, a three-time BBRF grantee and winner of BBRF's Klerman (2004) and Ruane (2020) Prizes; and **Deanna Barch, Ph.D.**, a four-time BBRF grant recipient and winner of the 2024 BBRF Lieber Prize.

Of particular interest to the team were the temporal dynamics of sleep/depression/SITB relationships: how they evolved over time in a sample of preadolescents, many of whom had been diagnosed with major depressive disorder (MDD) in early childhood. They were also mindful of the fact that traditionally, sleep disturbances in adults have often been thought of as symptoms of depression, not an independent risk factor. This may or may not be a useful way of thinking of sleep and depression, whether for adults or young people.

To explore "temporal dynamics and directionality" of sleep/depression/SITB relationships in preadolescence, the team drew in part upon children who had participated in a randomized-control trial at Washington University to study the effectiveness of a form of depression therapy for young children and their parents, called Parent-Child Interaction Therapy--Emotion Development (PCIT-ED). That study enrolled 229 children ages 3–7 from 2014–18.

Some of those children along with others formed the cohort in the current study of 210 children ages 8 to 12 years. These children, 125 of whom had a history of or current SITBs, were asked to complete weekly assessments (via computer or phone) of depression symptoms and SITBs across the course of a year. Of these subjects, 118 completed surveys for at least two consecutive weeks and were included in the analysis for the new paper.

The 118 participants in the final analysis were, on average, 10 years old; 29% female; and 75% white. In addition, 46% of the sample had a current diagnosis of MDD (i.e., diagnosis made in preadolescence), and 87% had either a past or current diagnosis of MDD. About half the cohort had a current diagnosis of an anxiety disorder or phobia, and about 25% had a current diagnosis of ADHD.

The most important finding of the study was that weekly self-reports of trouble sleeping were found to predict and precede reports of increased depression severity and engagement in self-harm behaviors the following week. Also, reports of fatigue predicted increased depression severity the following week.

The researchers indicated a number of possible mechanisms that might be underlying the associations they found. Disturbed sleep often leads to long "awake" periods in bed, which can provide opportunities for rumination—the repetitive contemplation of negative thoughts. Also, sleep disturbances are known to affect the function of brain regions central in emotional regulation; they may help to exacerbate negative moods. Another possibility is that the loss of sleep reduces REM sleep, which is thought to help individuals process and manage negative emotional memories.

The team stressed its finding on linkages between disturbed sleep and near-term increased deliberate self-harm behaviors (although possibly not including suicidal thoughts) appeared to be independent of any impact sleep disturbances had on increased depression severity. This risk "signal," if replicated in larger studies involving a more diverse cohort, could be of great import to doctors caring for preadolescents. Monitoring sleep disturbances, and potentially providing interventions to improve sleep habits, might help prevent self-harm in at-risk preadolescents, the team said.

"Clinicians working with preadolescents with or at risk for depression and/or self-harm behaviors should regularly assess difficulty sleeping and fatigue," they advised. "Measurement-based care including weekly assessments . . . could be incorporated into treatment for depression or self-harm in a brief and cost-effective manner to detect sleep disturbances" when they first appear. Also, the team suggested, "clinicians should consider intervention strategies to help shift sleep earlier (e.g., bedtime scheduling), to increase adherence to consistent daily sleep-wake rhythms, increase sleep durations overall, and target social barriers to sleep (e.g., reducing caffeine and phone use before bedtime)." ❖

Alcohol-Regulating Hormone Delivered in Combination With GLP-1 Drug Has Potential Application in Alcohol Use Disorder

Next-Generation Therapies: Addiction

Story Highlights:

Animal experiments with an analogue of the liver-produced hormone FGF21 indicated how it modulates the brain to curtail alcohol consumption. Its impacts were augmented when combined with a GLP-1 stimulating drug, suggesting a potential future treatment for alcohol use disorder.

Journal:
Neuropsychopharmacology
May 26, 2025

Researchers led by a BBRF grantee report new research extending knowledge about how a naturally occurring hormone called FGF21 helps to regulate alcohol consumption. Using a synthetic analogue of the hormone, they showed in mouse experiments how it appears to impact behaviors relevant in alcohol consumption as well as how it impacts the activation of neurons involved in the initiation and termination of drinking.

The research is of particular interest because FGF21 and the pathways it impacts are targets of interest in the development of new treatments for alcohol use disorder (AUD). A number of medications for AUD are in use today (naltrexone, nalmefene, acamprosate, and topiramate), but their effectiveness varies widely and the search for new treatments continues.

FGF21 is one of a number of peptides (protein fragments) that operate in the body as hormones and play key roles in metabolic health (glucose regulation, insulin sensitivity) and energy balance. These include GLP-1, produced in the gut and the target of weight-loss and diabetes medicines such as Ozempic and Mounjaro. (Others are orexin, produced in the brain, and leptin, generated within fatty tissue.) FGF21 (fibroblast growth factor 21) is generated in the liver in response to various metabolic stressors, including alcohol.

In a paper appearing in *Neuropsychopharmacology*, a team led by 2020 BBRF Young Investigator E. **Zayra Millan, Ph.D.**, of the University of New South Wales, Sydney, Australia, point to three lines of evidence implicating FGF21 in the regulation of alcohol consumption. One is experiments in mice in which induced overexpression of FGF21 as well as pharmaceutical administration of an FGF21 analogue both lead to reduced preference for alcohol in animals. Second, in large-scale genome studies in people, DNA variations affecting the gene that encodes FGF21 and its cellular receptor are statistically associated with alcohol consumption and risk of AUD. Third, studies in which FGF21 signaling is disrupted or blocked result in increased alcohol consumption by laboratory mice.

The latter experiments serve to remind that FGF21 is involved in signaling that normally acts to curtail alcohol consumption. A pathway carrying this signal has been localized in mice, and involves neurons in the basolateral amygdala (BLA) that project to the nucleus accumbens (NAc). Stimulation of the relevant NAc neurons inhibits consumption, while a pause in activation of these neurons is required when an individual initiates and maintains alcohol consumption. How FGF21 affects these NAc neurons was unknown prior to the current study.

E. Zayra Millan, Ph.D.

University of New South Wales, Sydney, Australia
2020 BBRF Young Investigator

Dysregulation of FGF21's normal function could be one way of understanding how chronic and habitual alcohol consumption can lead to AUD. Mammals began consuming alcohol from fermented fruit long before humans developed methods to distill alcohol. It is therefore not surprising that multiple bodily systems in mammals, including humans, evolved over time to sense and regulate alcohol consumption. The prevalence of AUD in humans indirectly suggests that naturally evolved regulatory systems can become dysfunctional (due to genetic and/or environmental factors), removing the evolutionary "brake" on excessive or health-impairing alcohol intake.

In their newly reported research, Dr. Millan and colleagues used an FGF21 analogue called PF-05231023 to confirm FGF21's ability to reduce voluntary alcohol consumption and preference for alcohol (vs. other fluids offered—sweetened water, in the mouse experiments). But the results that are most notable concerned the influence of the FGF21 analogue on behaviors involving alcohol consumption. Notable among these are "approach behaviors," i.e., those an individual takes toward a stimulus perceived to be positive or rewarding. In alcohol consumption, a variety of cues, such as time of day, a specific activity, or suggestion by peers can trigger approach behaviors, i.e, actions required to obtain alcohol.

The team's experiments showed that the FGF21 analogue directly reduced alcohol consumption in male mice, but not females. The reason for the sex specificity is not clear and will be pursued in future studies. The team suggests the difference may be due to sex-specific metabolic effects of synthetic FGF21 (i.e., the analogue, as opposed to the naturally occurring hormone), and/or may be related to a difference in expression of FGF21 receptors in males and females or in liver status relative to diet.

In both sexes, the FGF21 analogue weakened the intensity of responses following the presentation of alcohol-related cues, and also reduced the motivation of individual animals to seek alcohol. Importantly, the drug did not affect the animals' pursuit of sugar when sucrose-related cues were given or when motivation to seek sucrose solution was tested. This is one of several pieces of evidence suggesting to the team that the FGF21 analogue's effects on consumption was reward-specific—it did not perturb the reward response globally.

The experiments also showed that the FGF21 analogue's impact on alcohol drinking in males appeared to be associated with "pre-ingestive evaluative processes and reward palatability." In other words, the hedonic, or pleasure-driven urge to consume alcohol appeared to be altered. This was linked in the team's research with the drug's ability to alter the activity of neurons in the nucleus accumbens—it increased the recruitment of NAc neurons involved in termination of alcohol drinking behavior.

The team administered the FGF21 analogue in concert with a sub-therapeutic dose of a GLP-1 stimulating drug called Exendin-4, which targets signaling between the gut and brain to control metabolism. Their joint administration had the effect of augmenting the impact of the FGF21 analogue on alcohol-seeking behavior. Exendin-4, alone, had no such effect.

To the team, this was evidence of a "complementary and inter-dependent mechanism of action" in FGF21 and GLP-1. "Our findings suggest that combination agonist [i.e., hormone-stimulating] approaches may be of benefit in the treatment of AUD." Such an approach, they note, is already being tested in context of weight loss and insulin sensitivity. ❖

Brain Changes Underlying PTSD Are Revealed in Detailed Analysis at the Single-Cell Level

Basic Research: PTSD

Story Highlights:

A new study shows how the brain in PTSD is altered at the level of genes and single cells. Affected brains showed decreases in communication from inhibitory neurons, possibly accounting for hyperexcitation in the prefrontal cortex. Immune-related microglia were found to be underactive, and cells lining blood vessels were found to be dysregulated.

Journal:
Nature
June 18, 2025

A new study empowered by advances in technology has provided what is likely the most detailed account to date of biological changes that take place in the brain when someone has post-traumatic stress disorder (PTSD). The new findings shed light on PTSD pathology, identify specific and potentially targetable genetic, cell-type, and functional alterations, and also shed light on factors distinguishing brain changes in PTSD and major depressive disorder (MDD).

Led by **Matthew J. Girgenti, Ph.D.**, of Yale University, a two-time BBRF Young Investigator whose 2023 grant award helped support this research, a team that included 8 other recipients of BBRF grants examined a variety of changes at the single-cell level in 111 postmortem brains donated by people in three subgroups: those who had lived with PTSD, those diagnosed with MDD, and those who did not have a psychiatric diagnosis. Of the past BBRF grantees on the team, two are members of BBRF's Scientific Council: its vice-president, **John H. Krystal, M.D.**, and **Kristen J. Brennand, Ph.D.**, both of Yale.

The data that contributed to the team's analysis was derived from over 2 million individual cells from the brain's dorsolateral prefrontal cortex (DLPFC), and specifically from the nuclei of those cells, which harbor the human genome and the regulatory elements that determine how and when genes are expressed. Prior studies have shed considerable light on genetic variations seen in PTSD patients, as well as molecular-level changes in PTSD brains, and have revealed alterations in a number of gene pathways affecting inhibitory neurotransmitter signaling, immunity, and neuroinflammation, as well as signaling in the glucocorticoid system, which processes the stress response that is perturbed in PTSD.

But until recently, it was not possible to study genetic variation in individuals affected by a given disorder at the level of individual cells in brain regions of interest like the DLPFC, which is part of the cerebral cortex and plays a major role in the regulation of emotions. "Advances in genome technologies now enable the study of chromatin assemblies in individual cells," the team noted, referring to the bundling of DNA in the cell nucleus that helps determine which of our ~21,000 genes can be activated and which cannot be at a given moment.

When combined with an analysis of which genes in a cell are being expressed (i.e., activated so as to generate the manufacture of specific proteins), chromatin data can provide the resolution needed to identify how DNA variations associated with PTSD affect the process called transcription—the copying of genetic information to RNA—in individual cells. It is at this fine level of detail that progress can be made in learning

Matthew J. Girgenti, Ph.D.

Yale School of Medicine
2023, 2019 BBRF Young Investigator

how an illness like PTSD or depression alters neurons and the circuits they form, as well as other brain-cell types.

This new capability is especially significant in trying to understand the biological causes and effects of some psychiatric illnesses including PTSD and depression, which, unlike brain diseases like Alzheimer's, are not associated with large-scale pathologies like plaques that can be readily imaged.

"We annotated and censused all major [brain] cell types, including excitatory and inhibitory neurons and non-neuronal cell types," the team reported in the journal *Nature*. "We identified cell type-specific genes that were expressed differentially in PTSD and converging and diverging expression changes between PTSD and MDD." They also "constructed the regulatory landscape" impacting gene expression in PTSD. These and other analyses led to a number of major findings.

Among the postmortem brains with PTSD, the team's investigation revealed notable gene alterations in inhibitory neurons, which "fine-tune" excitatory brain circuits and in this way regulate them, among other things preventing them under normal conditions from firing too much. In brains affected by PTSD and MDD, the team observed a decrease in communication from inhibitory neurons—which may account for hyperexcitation in the prefrontal cortex. Following a traumatic experience, hyperexcitability might give rise to some of the symptoms seen in PTSD, such as hypervigilance or even nightmares.

The immune cells unique to the brain, microglia, were found to be overactive in the MDD brains and underactive in the PTSD brains. The apparent suppression of neuroimmune processes and microglial activity in the PTSD brains "is a finding that seems to differentiate MDD and PTSD," Dr. Girgenti noted, despite a number of previously noted genetic overlaps.

The PTSD brains were found to have genomic changes associated with dysregulation in endothelial cells, which line the blood vessels. This was an unexpected finding. It is known, however, that cortisol, the primary stress hormone, is paradoxically present at unusually low levels in PTSD brains. The team speculated that this previously unknown neurovascular dimension of PTSD, mirrored by high levels of activity in endothelial cells of a previously identified PTSD risk gene called FKBP5, may prove to be a mechanism to compensate for the unusually low cortisol levels.

Taken together, the study "enabled us to identify genes and pathways associated with PTSD pathology." These included stress hormones, immune, and neuroinflammatory mechanisms, in addition to the inhibitory neurotransmitter GABA.

About half of people with PTSD also suffer from MDD. The study helps identify "convergent and divergent molecular effects of both," the team said. But future extensions of this work should include analysis of a greater number of brains from people diagnosed with PTSD but not MDD to further highlight changes that are specific to PTSD. They also noted that like all other studies reliant on postmortem brains, it was impossible to say in this study how other factors among individual donors might have perturbed their findings. One example is the use of medications as well as other substances, which can alter the brain and its functions significantly. A greater number and more diversity among brain donors will aid future studies of this kind, they said. ❖

Addictive Use of Phones, Social Media, & Video Games Is "Common" in Young Adolescents and Linked to Risk for Suicidal Behaviors and Worse Mental Health, Study Finds

Diagnostic Tools/Early Intervention: Depression, Suicide Prevention, Childhood Mental Health

Story Highlights:

Four years of data from a large study of U.S. youths ages 10-14 finds that addictive use of phones, social media and video games is commonplace, and associated with 2 to 3 times higher rates of suicidal ideation and behaviors, and other mental health problems.

Journal:
Journal of the American Medical Association (JAMA)
June 18, 2025

Researchers using 4 years of interim data from a large, ongoing study of mental health and brain development in American children and adolescents have found that "high" or "increasing" addictive use of screen-based activities is not only commonplace, but is also associated with two to three times higher rates of suicidal ideation, suicidal behaviors, and other mental health problems, compared with those with "low" addictive or much weaker habitual screen use.

The new study, published in the *Journal of the American Medical Association (JAMA*), is an important contribution to the vigorous debate about how the advent and ubiquitous use of social media, mobile phones, and video games is affecting young people.

Considerable past research has focused on the potential impact of total screen time on youth mental health. Results have been inconclusive. The new study, while including screen time in its analysis, finds that it is not, by itself, specifically associated with elevated risk for suicidal ideation or behavior or what psychiatrists call internalizing and externalizing behaviors. Rather, the study finds, it is the role that high or increasing addictive use trajectories of screen-based activities play in the lives of young people that can specifically be linked with adverse mental health outcomes, including those associated with suicide.

BBRF Scientific Council member **J. John Mann, M.D.**, a world authority on suicide at Columbia University and the New York State Psychiatric Institute, and the winner of 2022 BBRF Colvin Prize and a 2008 BBRF Distinguished Investigator, was senior member of the team.

The researchers based their study on the most recent release of data from the U.S. government-supported Adolescent Brain and Cognitive Development (ABCD) study, which has recruited over 11,000 youths ages 9 and 10 at 21 U.S. sites. These young people are being followed all the way through adolescence, to the transition to adulthood. Many kinds of measures are being taken periodically in the ABCD study, ranging from brain scans to blood draws to detailed mental health assessments, making possible a great variety of spin-off studies using portions of the total dataset, including the one just reported on screen use.

A total of 4,635 of the ABCD participants completed surveys at their 2-, 3- and 4-year follow-ups after joining the study. These follow-ups included self-reports of screen use and habits as well as self- and parental reports of mental health. Subtracting those with missing information, the cohort analyzed for the current study numbered 4,285 youths, who were 10 years old on average at the study's baseline and 14 at the 4th follow-up.

Establishing "addictive use" for the 3 screen-based modes—social media,

J. John Mann, M.D.

New York State Psychiatric Institute
Columbia University Medical Center
2022 BBRF Colvin Prize
2008 BBRF Distinguished Investigator

mobile phones, and video games—was based on several self-report questionnaires, filled out annually over the 4-year interval monitored in the study. These included questions such as "I feel the need to use social media apps more and more" (1=never, 6=very often); "The thought of being without my phone makes me feel distressed" (1=strongly disagree, 7=strongly agree); and "I play video games so I can forget about my problems" (1=never, 6=very often). All of these have been shown to have high reliability in past studies.

Child and parent reports of suicidal behaviors and suicidal ideation over the prior year were assessed at year 4, using another well-validated questionnaire covering a spectrum of suicide-related outcomes. These included, for ideation: a "yes" reply to either: passive ideation; nonspecific active ideation; specific active ideation; active ideation with intent; or active ideation with plan and intent. Suicidal behavior was indicated with a "yes" reply to any of the following: preparatory actions for imminent suicidal behavior; interrupted attempt; aborted attempt; or actual attempt.

A final analytic component, the Child Behavior Checklist (CBCL), periodically filled out by parents, was used to indicate clinically significant adverse mental health-related behaviors, grouped as internalizing and externalizing.

"This study identified distinct trajectories of addictive use of social media, mobile phones, and video games from childhood to early adolescence, and found links to suicidal behaviors, suicidal ideation, and worse mental health outcomes," the team reported.

For both social media and mobile phones, addictive use trajectories followed 3 different patterns, "and a substantial proportion of youths had addictive use trajectories that increased over the 4 years of observation, starting at age 10," the team said. These patterns of increasing addictive use as the years passed, they noted, "would not have been predicted" based on assessments made at the beginning of the study, and were specifically associated with suicidal behaviors and ideation. "This underscores the potential importance of repeated assessment" of addictive screen use as children enter adolescence, they said.

Video game use was found to follow 2 trajectories, dubbed "high" and "low." These were stable over time, which to the team means that those most at risk might be identified early, without the need for repeated assessment.

Almost 1 in 2 youths had a high addictive use trajectory for mobile phones, and more than 40% had such a trajectory for video games. "Many others had increasing addictive use over the 4-year observation period which ended with high addictive use." Almost 1 in 3 had this "increasing addictive" trajectory for social media and 1 in 4 for mobile phones.

As for how these trajectories affected mental health risk: for social media and mobile phones, both the "high" and "increasing" addictive use paths were associated with 2 to 3 times greater risks of suicidal behaviors or ideation, compared with "low" addictive use trajectories (i.e., not all "addictive" use was linked with increased suicide risk—just "high" or "increasing" addictive use). Also, "high" and "increasing" addictive use of social media were found to be associated with higher internalizing and externalizing symptom scores compared with the "low" addictive use trajectory. The "high" addictive use path for video games was linked with higher risks of suicidal behaviors and ideation and higher internalizing symptoms compared with the "low" addictive use path.

Total screen time was not found in this study to be associated with suicide-related or mental health outcomes, nor did it alter the various findings regarding associations between addictive use trajectories and these outcomes. "Total screen time" and "addictive use" are likely two different constructs, the team said. This is not to say, however, that total screen time is not an important factor in mental health. For example, long periods on the phone or other screen activities are well understood to crowd out sleep, exercise, and face-to-face contact in many users—none of which are healthy. Both constructs are likely important, though in different ways.

The current study will likely lead to additional studies on monitoring the addictive element in the use of electronic devices among youth. It also calls urgent attention to the issue of developing effective preventive and treatment approaches for those youth who do become addicted to their screens. ❖

More Screen Time in Childhood Followed By Depression in Early Adolescence Is Linked to Less Sleep and White Matter Changes

Diagnostic Tools/Early Intervention: Depression, Suicide Prevention, Childhood Mental Health

Story Highlights:

More daily screen time in late childhood (ages 9–10) was associated in a new study with more depressive symptoms in early adolescence (age 13). The screen time/depression relationship was related to short sleep and changes in white matter.

Journal:
JAMA Pediatrics
June 23, 2025

A new study based on data from nearly 1,000 young people, ages 9–13, suggests how the amount of time spent on screens each day—TVs, computers, mobile phones, videogames—impacts depression risk at age 13. More specifically, the study relates screen use to sleep patterns and the structure of the brain's white matter.

2021 BBRF Young Investigator **João Paulo Lima Santos, M.D.**, of the University of Pittsburgh, devoted his grant to research that culminated in the new paper, which appears in the journal *JAMA Pediatrics*. He and colleagues wanted to know whether more screen time could impact sleep duration, and perhaps as a result of that, structural connectivity in the young, developing brain. One way of studying structural connectivity is via the brain's white matter, which largely consists of axons and dendrites that link the human brain's estimated 85 billion neurons. These connective "tracts" are called white matter because of their physical appearance: axons and dendrites are coated with a fatty insulation called myelin, which in microscopes appears off-white in color. Other members of the team included **Amelia Versace, M.D.**, a 2009 BBRF Young Investigator, and **Cecile D. Ladouceur, Ph.D.**, a 2006 BBRF Young Investigator, both also at the University of Pittsburgh.

"Time spent on screens is not necessarily harmful," the team notes in its paper, pointing out that some studies of the question have found a weak or no relation between screen time and youth mental health. Some of these studies have, however, suggested a possible relation between use of screen-based devices and depression, "particularly during windows of developmental sensitivity such as early adolescence."

The new study is predicated on the idea that such a possibility must be explored in greater detail. Sleep is critical for healthy neurodevelopment, "influencing the maturation of brain regions implicated in emotional regulation," they note, "with noteworthy associations in early adolescence involving sleep duration and the brain's gray and white matter." Insufficient sleep and poor sleep quality are already known to raise the risk of depression in adolescents. How screen time might affect sleep and thus, indirectly, the question of depression risk is at the heart of the new study.

The study led by Dr. Lima Santos made use of the remarkable dataset of the NIH's Adolescent Brain Cognitive Development Study (ABCD), which recruited over 11,000 young people ages 9–11 at 21 U.S. sites and is following them through adolescence. The data analyzed in the current study was based on a subsample of 976 young people in the ABCD dataset, measured at two points: at the time of recruitment to the study (in this sample, average age between 9 and 10, i.e., "late childhood"); and at age 13 ("early adolescence").

João Paulo Lima Santos, M.D.

University of Pittsburgh
2021 Young Investigator

Self-reported screen time at both time points was collected using a questionnaire that broke down activity into categories: watching TV and videos ("passive entertainment"); playing videogames ("active entertainment"); and texting, using social media, videochatting ("communication").

Studying white matter structure was a key part of the study. Early in life and through adolescence, white matter undergoes critical changes, including myelination and reorganization of axons and dendrites. External factors including physical injury can alter the process, as can inadequate or irregular sleep. To study white matter, the team used an imaging technology that enabled them to parse specific components affected by sleep and perhaps screen time. Called NODDI (neurite orientation dispersion and density imaging), it has previously established that "short sleep" in early adolescence is associated with decreased "coherence" in white matter tracts implicated in emotional regulation. These include the cingulum bundle, forceps minor, and uncinate fasciculus. These were under scrutiny in the brain scans from the study's sample of 976 young people at the two study time points (ages 9-10 and 13).

After analyzing the data, this is what the team found: first, and perhaps most important, more daily screen time in late childhood (e.g., ages 9–10) was associated with more depressive symptoms in early adolescence (e.g., age 13). The attention to white matter paid off: the team found that the screen time/depression relationship was related to short sleep and diminished coherence of a particular white matter tract type, cingulum bundles. By this, the investigators mean that short nightly sleep duration and a deterioration of white matter organization were factors that correlated with the specific relations observed (with more screen time at age 9 or 10, more depression symptoms at age 13). This is not to say screen use or sleep time caused the white matter deterioration; separate studies must be conducted to prove such a relationship. But the factors do co-occur, this research indicates.

Another finding: 1 hour of daily screen time was associated with shorter sleep; 2 hours with more depressive symptoms, and 3 hours with worse organization of the white matter cingulum bundles.

Regarding "shorter sleep," the team proposed several factors that may lie behind this: blue light from screens may disrupt melatonin, circadian rhythms, and sleep timing. Also, evening screen use, especially if stimulating, can delay bedtime and displace sleep.

Active and passive entertainment were linked with short sleep and white matter deterioration, but screen time devoted to communication did not appear to impact sleep duration, the study indicated. The authors noted that "this suggests that the relationships involving social media in early adolescence may be more nuanced, and components beyond screen time (such as actual content) should be explored." Still, screen time may be found to be more relevant in the years just ahead, the team said, given that there has been an increase in social media use among children aged 8–12 years.

What about parental efforts to restrict screen time? The researchers are skeptical, calling these "impractical, especially for adolescents who rely on screens for other activities such as homework."

"Altogether, the findings show that screens pose potential risks, but engaging parents, educators, and adolescents is essential to develop balanced strategies" that can address screen use and sleep "based on individual needs," the team said.

In the end, the team feels their findings on white matter are the most important in the study: white matter is a key factor, they stressed, in understanding how screen time interacts with sleep to impact depression risk by age 13. "These findings underscore the importance of fostering healthy screen time habits and prioritizing adequate sleep to support emotional and brain development" they said, stressing that sleep habits, in particular, are something that are eminently modifiable to promote mental and physical health, assuming parents and children are willing to work on them. ❖

For the First Time, Researchers Model a Crucial Phase of Early Human Brain Development—the Forging of Connections Across the Developing Spinal Cord

Basic Research: Autism, Schizophrenia, Neurodevelopmental Disorders

Story Highlights:

Using stem cell-based technology to create living models of human brain development, researchers for the first time succeeded in modeling the process through which specialized cells in the neural tube guide axons crossing from one side of the emerging spinal cord to the other, establishing bilateral connectivity.

Journals:
Science
July 17, 2025
Nature
April 9, 2025

A great deal remains to be discovered about how the human brain develops into its mature form, starting with its origins in a tube-like structure that emerges early in embryonic development called the neural tube. From out of this humble tube, the brain and spinal cord grow. The nervous system's process of self-assembly, guided by developmental programs evolved over millions of years and stored in our genes, is one of the great miracles of nature.

The very beginnings of the process are understood in part because they have been observed in animal models of brain development. While suggestive, these models diverge from the human developmental process in important details. In recent years researchers have developed specialized tools to create laboratory models of early human brain development, an achievement that is shedding light not only on how the human brain develops but also on problems and pathologies that perturb peripheral and central nervous system development and result in neurodevelopmental disorders that include autism and schizophrenia.

Now, in the journal *Science*, researchers describe their success in modeling a crucial phase of early human brain development for the first time. The research was directed by **Sergiu P. Pasca, M.D.**, whose work has been supported by 2017 BBRF Independent Investigator and 2012 Young Investigator grants. **Neal D. Amin, M.D., Ph.D.**, a 2021 BBRF Young Investigator, was co-first author on the new paper.

Dr. Pasca and his team are among the leading developers of new technologies that are revealing the secrets of human brain development. He was a pioneer in harnessing stem-cell science to create brain "organoids," structures grown in a lab dish derived from patient blood- or skin-cell samples. Reprogrammed to develop as neurons or other brain cell types, cells in brain organoids wire together and begin to function. When transplanted into living rodents, they integrate with the functioning brain. This provides a window on normal brain development, but also on pathologies that perturb it, at the level of cells and circuits.

The new research relies on a technique Dr. Pasca perfected several years ago, to integrate organoids comprising different brain-cell and central nervous system cell types into larger, more complex entities he calls "assembloids." The aim of the new assembloids created by the Pasca lab was to model the process through which neurodevelopmental guideposts called "organizers" orchestrate cell patterning and axon guidance in the spinal cord of the emerging nervous system.

Some of this biology has been modeled in mammals, leading to fundamental discoveries about how cells within a structure called the

Sergiu P. Pasca, M.D.

Stanford University
2017 BBRF Independent Investigator
2012 BBRF Young Investigator

floor plate help direct neuronal axons to cross the spinal cord's midline. The uniquely human aspects of this process remain mysterious, which led Dr. Pasca and colleagues to build assembloids comprised of three human cell-based organoid types to model the process as it unfolds in people.

The floor plate is a specialized group of cells at the ventral (i.e., bottom) midline of the developing neural tube. The floor plate is transitory group of specialized cells that secrete crucial patterning signals and can guide axons crossing from one side of the spinal cord to the other. This process of "crossing" establishes bilateral connectivity, which is critical for coordinating left-right movements. Problems that crop up during this early process of establishing midline connectivity have been implicated in neurodevelopmental disorders.

The Pasca team proceeded in several steps. First, they developed organoids that resembled the floor plate in humans. These were combined and integrated with separate organoids resembling the human spinal cord. The product of these multiple organoids, once combined, was an entity the team calls "midline assembloids."

Various experiments established that the floor plate organoids faithfully generated patterning and axon guidance signals that are present in the human floor plate and are responsible for guiding axons across the midline and establishing connectivity across the spinal cord.

In an important set of experiments, the team analyzed the "secretome" of the cells comprising the floor-plate assembloids. The secretome is the complete set of molecules secreted by a cell into its surrounding environment, and the basis for interactions among cells of all kinds. The team found 27 genes whose expression profile was markedly different in humans from that seen in corresponding floor-plate cells in the developing mouse brain. These are clues to unique aspects of human brain development.

Using the gene-editing technology called CRISPR, the researchers deleted various of the human-enriched floor-plate genes, and observed the impact on axon guidance directed by floor-plate cells in midline assembloids. This revealed that two genes in particular, called GALNT2 and PLD3, when deleted, significantly impaired axon guidance. Implying that these two genes are critical in the process, it suggests potential sources of pathology relevant to axon guidance in early development. It's just one example of the kind of insights that the "assembloid" approach can generate, since such experiments can never be performed in the human fetus.

The assembloid model reported in their paper "holds potential for additional applications" the team said. "Expanding beyond the spinal cord, assembloids can model midline function in other brain regions such as the corpus callosum, which are often disrupted in neurodevelopmental disorders." The corpus callosum is a large bundle of nerve fibers that connects the right and left hemispheres of the brain, allowing them to communicate and coordinate functions.

Broadly speaking, the team noted, "organizer organoids offer a versatile toolset to advance our knowledge of human cell specification, axon guidance, and evolution, which are crucial to understanding human neurodevelopment and the impact of neurodevelopmental disorders." ❖

Study Sheds Light on MDMA's Prosocial Effects & Lower Abuse Potential

Next-Generation Therapies: PTSD, Depression

Story Highlights:

A new study provides evidence explaining why a form of the psychedelic compound MDMA at low doses has reduced abuse potential compared with other psychedelic drugs and why it appears to generate pro-social effects, potentially useful in treating, for example, PTSD.

Journal:
Molecular Psychiatry
July 24, 2025

For several years, **Robert C. Malenka, M.D., Ph.D.**, a BBRF Scientific Council member and a 3-time BBRF grantee and prizewinner, along with some of his Stanford University colleagues, have been pursuing what they call a "circuits-first approach" to research aimed at better understanding psychedelic drugs and their potential to be useful in the treatment of psychiatric illnesses such as depression and PTSD.

They have urged that by using modern neuroscience tools to "define the [brain-]circuit adaptations that contribute to a drug's behavioral and therapeutic effects, studies can be conducted which could reveal new molecular targets in brain cells or circuits" which can be used as a basis for developing novel versions of psychedelic drugs that have maximum therapeutic impact and cause fewer side effects.

In a newly published paper in *Molecular Psychiatry*, Dr. Malenka, along with senior collaborator Boris D. Heifets, M.D., Ph.D., and a team that included 2023 and 2020 BBRF Young Investigator **Neir Eshel M.D., Ph.D.**, show some of the fruits of the "circuits-first" approach. They closely studied how the drug MDMA (known on the street as "molly" and "ecstasy") exerts its principal effects—some undesirable, some potentially therapeutic—and found separate mechanisms that appear to be responsible for each. Taken together, the results suggest how and why MDMA appears to have lower abuse potential than some other psychotropic drugs, and may have potential for use as an "enactogen," a drug that induces feelings of empathy and emotional openness.

The behavioral effects of MDMA in "assisted therapy" applications tested in small trials in people with PTSD have indicated its characteristic properties: an enhanced sense of emotional connectedness and empathy, along with reduced fear when confronted with aversive stimuli like traumatic memories. But MDMA, an amphetamine analog, is prone to misuse and abuse, which, the team notes, is "an important risk consideration for treating patients with PTSD, many of whom have comorbid substance use disorders."

At the same time, MDMA is not as widely abused as closely related amphetamine drugs, such as methamphetamine. The question the researchers explored in their study was whether MDMA's reduced abuse potential is mechanistically linked to its therapeutic behavioral effects. Prior work by the team helped explain how MDMA's neurochemical mechanism differs from that of methamphetamine. In some respects it is similar: like "meth" and other amphetamines, MDMA has a molecular affinity for the protein that transports dopamine molecules in the brain, called the dopamine transporter (DAT). Like all amphetamines, MDMA amplifies

Robert C. Malenka, M.D., Ph.D.

Stanford University School of Medicine
2010 BBRF Goldman-Rakic Prize
2007 BBRF Distinguished Investigator
1992, 1990 Young Investigator

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

Stanford University School of Medicine
2023, 2020 BBRF Young Investigator

dopamine release in the brain, which generates an intensely rewarding feeling—and is also the reason it can be addictive.

But unlike meth, the researchers were able to show that MDMA also has a high affinity for the protein that transports serotonin in the brain, called the serotonin transporter (SERT). They found that in a major reward center of the brain called the nucleus accumbens (NAc), serotonin release appeared to account for MDMA's prosocial effects in various mouse experiments. This prosocial effect was the result of an interaction between MDMA and SERT, and subsequent activation of one of the many receptors for serotonin in the brain—the serotonin 1B receptor, in cells in the NAc.

In contrast, their experiments and those of other investigators have shown that the nonsocial drug reward evoked by meth—as well as high doses of MDMA—appear to be traceable to dopamine release, in the same brain structure, the NAc. This raises the question of whether and how the specific dopamine- and serotonin-enhancing effects of MDMA in the NAc might be mechanistically related, and whether this might help explain why MDMA at low doses has been observed to have a lower risk of abuse in humans.

The team conducted their experiments in mice. The form of MDMA administered at various dosages (from low to high), called R-MDMA, is a chemical version that has a structural configuration which gives it distinct properties compared to conventional MDMA. Multiple structural forms of MDMA were administered for comparison purposes, as well as methamphetamine and cocaine. Well-validated tests were performed, revealing the addictive properties of drugs based on conditioned expectation of reward, as well as tests in which the social behavior of the animals could be closely observed before and after drug administration, including in animals with transporters for serotonin or dopamine genetically deleted.

One finding was that serotonin released after MDMA administration had the effect of limiting the release of dopamine through activity observed in the NAc.

Further experiments revealed that MDMA's activation of a specific receptor for serotonin in the NAc—the serotonin 2C receptor—actively suppressed dopamine release in that brain structure. This action, the team suggested, may account for MDMA's lower addictive potential: its action lowers the rewarding release of dopamine in the NAc, compared, for example, with the stimulation of dopamine release in an administration of meth.

Other experiments provided evidence for theories about the possible source of MDMA's prosocial effects. The form being tested as a potential drug, R-MDMA, has prosocial effects because R-MDMA is more active at serotonin transporter molecules (SERTs) than at transporters for dopamine (DATs), especially in comparison with the standard form of MDMA, which affects these molecules more evenly.

Importantly, the precise cellular location of the serotonin 2-C receptors in the NAc linked with the drug's limitation of dopamine release and thus its lower abuse potential is still unclear, and should be taken up in subsequent research, the team said. They also cautioned that the simple behavioral tests they used in mice to explore addictive and prosocial behavioral characteristics of MDMA "are unlikely to represent the full range of social behavior and patterns of drug misuse" in people.

Still, the results of their study provide, they said, reason to continue exploring the use of R-MDMA (at low doses) in the clinic for therapeutic purposes in patients with illnesses like PTSD that often do not respond satisfactorily, or over the long-term, to current treatments. ❖

A Way to Repair a "Leaky" Blood-Brain Barrier (BBB) is Tested

Basic Research; Next-Generation Therapies: Schizophrenia, Autism, Neurodevelopmental Disorders, ADHD, Depression, Anxiety

Story Highlights:

Researchers have tested a way to potentially repair the integrity of the blood-brain barrier (BBB), the protective membrane that separates the brain from the bloodstream. Leaks in the BBB have been linked with neurodevelopmental illnesses including schizophrenia and autism.

Journal:
Science Translational Medicine
August 20, 2025

A team of researchers co-led by a three-time BBRF grantee has developed a way to potentially repair the integrity of the blood-brain barrier (BBB), the protective membrane that separates the brain from the bloodstream. Leaks and other problems in the BBB have been linked in past research with neurodevelopmental illnesses such as autism and schizophrenia as well as neurodegenerative conditions such as Parkinson's and Alzheimer's diseases.

The BBB is composed of specialized endothelial cells (ECs) that separate the bloodstream from brain tissue. The barrier they form regulates the passage of substances into and out of the brain, protecting it from harmful toxins and microbes, as well as proinflammatory immune cells, while allowing essential nutrients and signaling molecules to pass into the brain.

Investigators reporting new research in the journal *Science Translational Medicine*, led by BBRF grantee **Stewart A. Anderson M.D.**, and Jorge Ivan Alvarez, Ph.D., both of the University of Pennsylvania, have previously found impairment in the BBB in mice that model the human genetic condition called 22q11.2 deletion syndrome (22qDS). 22qDS involves the deletion of roughly 45 genes on one copy of chromosome 22, and is associated with neurodevelopmental disorders including intellectual disability, autism, and ADHD.

Dr. Anderson, a BBRF Scientific Council member, 2016 BBRF Independent Investigator and 2002 and 1995 BBRF Young Investigator, and another team member, **Adam J. Rossano, M.D.**, Ph.D., a 2023 BBRF Young Investigator, have both conducted grant projects focusing on the role of mitochondria—the ubiquitous energy "factories" that power our cells—in the context of pathologies thought to occur in schizophrenia and psychosis.

"Little is known about the role of the mitochondria in the BBB," the team notes in their paper, although it has been demonstrated that ECs in the BBB have a much higher mitochondrial content than ECs elsewhere in the body (i.e., outside the brain). This suggested to the team that ECs in the BBB may be unusually dependent upon mitochondria.

The new study is driven in part by the prior finding of BBB impairment in mouse models of 22qDS, but also by the previously established fact that among the 45 genes deleted in 22qDS, at least 6 encode proteins that localize to mitochondria. The researchers set out to study whether mitochondrial deficits contribute to BBB dysfunction in 22qDS, and whether this, in turn, might be linked to behaviors seen in people and mouse models of 22qDS—specifically, social deficits.

The team used induced pluripotent stem cell (iPSC) technology in some of their experiments, which enables

Stewart A. Anderson, M.D.

University of Pennsylvania Perelman School of Medicine
BBRF Scientific Council
2016 BBRF Independent Investigator
2002, 1995 BBRF Young Investigator
1999 BBRF Freedman Prize

investigators to study, in cell cultures or following transplantation into animals, pathologies that occur in the human brain. Some of these pathologies are thought to underlie neurodevelopmental disorders such as autism spectrum disorders and schizophrenia. Blood or skin cells are sampled from affected patients and then reprogramed to become stem cells that can be redeveloped as specific cell types such as neurons. Importantly, the new cells retain the disease-linked genetic variations of the original donor.

In this study, the team generated endothelial-like cells from stem cells derived from 22qDS patients who also were diagnosed with schizophrenia, as well as from controls. The researchers then conducted experiments showing mitochondrial impairment, including a deficit in ATP production in these cells. ATP is the main energy transfer molecule in cells that powers cellular and organ functions throughout the brain and body. Mitochondrial impairments were also seen in BBB endothelial cells from mice genetically modeling 22qDS syndrome.

On the basis of these results, the team proposes that mitochondria are indeed dysfunctional in the BBB in 22qDS—and possibly in other disorders in which BBB integrity is impaired and a source of pathology.

A separate set of experiments in both the 22qDS model mice and in the stem cell-derived human BBB cells involved treatment with a drug called bezafibrate. FDA approved, it is used to treat hyperlipidemia (high blood lipid levels), by lowering "bad" cholesterol and raising "good" cholesterol. Treatment with drugs of this type—stimulators of activity at PPAR receptors, which help regulate, among other things, the generation and turnover of mitochondria, have generated positive results in animal-model experiments pertinent to stroke, epilepsy, schizophrenia, and Alzheimer's.

In both in the stem cell-derived BBB and the 22qDS mouse model, bezafibrate improved mitochondrial generation of ATP, and improved measures of BBB "leakiness." It is possible that functional BBB improvement is a direct consequence of the drug's effect, but, the team noted, it is also possible that at least in the 22qDS mouse model, the drug's impacts on LDL cholesterol, reducing triglycerides, improving glucose sensitivity, and decreasing blood glucose levels might also have had influenced BBB function. Further experiments could reveal mechanisms more specifically.

After drug treatment, the team also observed upregulation, i.e., increased expression, of mitochondrial genes in ECs from the BBB in the model mice, another bit of evidence suggesting the drug's BBB-strengthening effects.

Importantly, treatment with bezafibrate also corrected deficits in social memory in the mice modeling 22qDS. This kind of deficit has been previously linked with BBB dysfunction. Again, it is possible, but not certain, that this outcome was influenced by bezafibrate directly affecting neuronal functioning, the team said.

Overall, the researchers suggest their experiments indicate that mitochondrial dysfunction in the BBB influences both BBB integrity and behaviors that may result from breaches, and "identify a potential therapeutic mechanism by which the BBB may be targeted in these conditions." ❖

New Research Underlines the Importance of Recognizing Mood Instability Between 'Major Episodes' in Bipolar Disorder

Diagnostic Tools/Early Intervention: Bipolar Disorder

Story Highlights:

New research underlines the importance of "mood instability" occurring between major mood episodes in bipolar disorder. 481 patients followed over 5 years fell into three distinct mood instability subgroups, "high," "moderate," and "low," each predicting future clinical and functional outcomes.

Journal:
Nature Mental Health
September 22, 2025

A research team led by 2022 BBRF Young Investigator **Sarah H. Sperry, Ph.D.,** of the University of Michigan, has published results of a study of 481 people diagnosed with bipolar disorder (BD). They underline the importance of "mood instability" occurring between the major mood episodes that have traditionally provided a foundation for describing and treating the illness.

There are three diagnostic categories for bipolar disorder indicated in the DSM-V manual used by most psychiatrists. Those diagnosed with Bipolar I have one or more depressive and manic major mood episodes; those with Bipolar II have one or more episodes of depression and hypomania, the latter a less intense but equally important form of mania. A third category, BD NOS (Not Otherwise Specified) is characterized by mood episodes that don't meet criteria in BD I or BD II for intensity or duration.

This diagnostic schema has provided "the foundation of BD research and clinical care," Dr. Sperry and colleagues note in their new paper, "and has focused on characterizing and treating episodes [of depression or mania/hypomania], identifying predictors of episode relapse, and the short- and long-term consequences of episodes."

However, the researchers point out, "increasing evidence suggests that bipolar disorders are associated with mood instability, even outside the context of [major] mood episodes."

Dr. Sperry, in part with the support from her Young Investigator grant, has for a number of years devoted her research to developing and testing mood instability measures that might reveal the extent and importance of between-episode mood fluctuations. The new paper, which appears in *Nature Mental Health,* reports results that led the team to identify three subgroups of BD patients based specifically on mood instability. Each of the subgroups has potential predictive power regarding long-term outcomes and implications for how patients are assessed and treated, the investigators said. The team also included: **Melvin G. McInnis, M.D.**, 1999 BBRF Independent Investigator and 1992 Young Investigator; and **Ivy F. Tso, Ph.D.**, 2018 BBRF Young Investigator.

The team defines mood instability as "frequent and/or intense fluctuations in mood over time." While mood fluctuations "inherently underlie the [existing] diagnostic criteria of BD," they stress that mood instability "also includes shifts in mood symptoms at subsyndromal levels." By this, they mean that some mood shifts may not reach the duration or intensity levels that qualify them as "major" episodes, but may nevertheless have a very real impact on the experience that patients have with the illness, and on how they fare over time. Among other things, the attention to "subsyndromal" mood shifts between major episodes calls into question the assumption made by some that in between major episodes,

Sarah H. Sperry, Ph.D.

University of Michigan
2022 BBRF Young Investigator

most BD patients are "euthymic," i.e., living in a state of emotional stability and well-being.

The team made use of data from one of the largest long-term studies of BD, the Prechter Longitudinal Study of Bipolar Disorder (PLS-BD). They aimed to characterize and identify subgroups among 481 individuals based on mood instability measured by standard self-report-based instruments every 2 months over 5 years. They also used machine learning to identify various risk factors noted at baseline (when each participant joined the study) that might predict each participant's mood instability status and their clinical and life-functioning outcomes in the 6th year after recruitment.

The cohort studied, mean age 41, was largely White (90%) and female (72%). 71% had a BD I diagnosis; 22% BD II; and 7% BD NOS.

Analysis revealed that participants fell into three distinct subgroups of mood instability, "high," "moderate," and "low." Over 78% were in the high or moderate groups, suggesting to the team that mood instability is a non-trivial factor in understanding the patient experience.

Of the many potential risk factors identifiable at "baseline," the team found seven that most reliably "predicted" outcomes (in retrospect, from the perspective of the investigators, who could not know at the outset which, if any, would have any predictive value 6 years later). The seven factors, in order of descending predictive power, were: neuroticism; sleep quality; childhood emotional neglect or physical abuse; stimulant abuse; age when hypomania first occurred; and number of depressive episodes. (The leading predictive factor, neuroticism, is used by psychiatrists to describe individuals with a tendency to experience negative emotions, such as anxiety, depression, anger, and self-doubt.)

"All seven of these factors can easily be measured across development and treatments, highlighting their utility as early risk markers," the team wrote.

They also found significant differences in clinical and functional outcomes across the three mood instability subgroups, with high mood instability correlating with the most severe outcomes in year 6. Compared with those in the low instability class, those in the high instability class had worse family and work functioning, poorer mental health functioning, and higher suicidal ideation in year 6. Being in the high instability group also predicted worse family functioning and suicidal ideation compared with those in the moderate group. Being in the moderate group, in turn, predicted worse family functioning compared with those in the low instability group. Also, the results indicated that the more chronic or remitting that depression is in BD patients, "the more unstable mood tends to be going forward."

"These results highlight the central role of mood instability in BDs, regardless of subtype or symptom severity," the researchers concluded. Hence, in their view, "the present findings compel us to rethink how we assess and treat BDs."

Dr. Sperry and colleagues say that that their results make the case for comprehensive and ongoing assessment strategies in clinical care of BD patients, as well as in longitudinal research of the illness. "We argue that baseline psychodiagnostics assessments should incorporate measures of the seven key predictors of mood instability identified in this study." Doing so, they said, might facilitate early identification of mood instability risk even before diagnostic status (such as BD I or II) is established.

The team also argues in the paper for implementing patient-reported mood assessment measures, which could potentially reveal significant—and treatable—mood instability between major episodes.

The investigators indicated a range of goals for follow-up studies, which should, among other things, include a more diverse patient population, and establish precisely which assessment tools should be used, and how often and for how long to identify mood instability risk in individual cases. Ultimately, an optimized way of making such determinations "may enable earlier, targeted interventions that mitigate mood destabilization and improve long-term outcomes" in those with BD, they said. ❖

Smartphone Sensors + ChatGPT Successfully Tracked & Predicted Symptoms in Adolescents with Anhedonia

Diagnostic Tools/Early Intervention; Next-Generation Therapies: Depression, Anxiety. Childhood Mental Health

Story Highlights:

A pilot study using passive smartphone mobility data plus text analysis by Chat GTP provided real-time, clinically relevant insights about depressed adolescents with anhedonia offering doctors a way to monitor and tweak therapy "in progress."

Journal:
NPP—Digital Psychiatry and Neuroscience
October 13, 2025

The ubiquity of smartphones and the rapid advance and widespread public adoption of "AI" tools like ChatGPT (especially among the young) have raised hopes among some researchers that such technologies can be harnessed in new ways to improve psychiatric assessment and treatment.

A newly published study by a team led by a two-time BBRF grantee reports results of a pilot study testing one way in which mobile phones and the large language model (LLM)-based ChatGPT tool can together aid in the assessment of behavioral changes that are important for therapeutic improvement in a behavioral therapy for adolescents with depression. Innovations of this kind could open doors to therapy adjustments made in real time based on indicators gleaned directly from patients.

The research, appearing in the journal *NPP—Digital Psychiatry and Neuroscience*, is the product of a project directed by **Christian A. Webb, Ph.D.** a 2018 and 2015 BBRF Young Investigator.

As the researchers note, while smartphones can passively collect rich behavioral data—with users' consent—by tracking patterns of movement and activity, and while LLMs like ChatGPT can analyze text and language with impressive accuracy, it has remained unclear whether these technologies can provide insight into the emotional or motivational aspects of an individual user's behavior.

This question helped to drive the team's pilot study, which examined the two technologies in the context of a talk therapy called behavioral activation (BA), which aims to reduce the classic depression symptom of anhedonia (the loss of interest in pursuing rewards or pleasure) by targeting patterns of avoidance and withdrawal and increasing engagement with rewarding activities.

"A core assumption of BA is that increased 'activation' in daily life leads to symptom change, but few studies have rigorously measured whether this is the case," the researchers noted.

"There is a pressing need for objective, low-burden methods to track activation in daily life," they wrote. "Tools that assess activation between [therapy] sessions could help therapists monitor treatment progress and make timely adjustments, an objective consistent with calls for data-informed psychotherapy."

Their pilot study made use of smartphone-based passive sensing of patients' mobility patterns and LLM-derived ratings of daily text entries they provided during the 3-month therapy period. These data were examined in relation to daily assessments of positive and negative emotions, as well as weekly assessments of anhedonia, depressive symptoms, and a traditional self-report of activation.

Christian Webb, Ph.D.

McLean Hospital, Harvard Medical School
2018, 2015 BBRF Young Investigator

The team recruited 38 adolescents, ages 13–18, from the Boston area to receive BA treatment targeting their anhedonia. Each was offered 12 weekly hour-long individual BA therapy sessions. Before each session, participants rated their anhedonia, depression, and "activation" (in essence, their engagement in daily activities and avoidance patterns), using self-report measures. A subset of 13 participants also contributed passive smartphone data—collected continuously via built-in accelerometer and GPS sensors.

Every other week during treatment all participants completed a "burst" of real-time assessments ("ecological momentary assessments") over 5 consecutive days via 2–3 surveys delivered each day via a smartphone app. Each time they were prompted by the app, participants were asked to rate their positive and negative affect (6 items, to be rated on a 5-point scale, generating data about being "happy" "interested" and excited"; and "sad," "nervous," and angry"). Next, they were asked to provide responses via unrestricted texting, "about what they were doing the night before, with whom they were interacting, and the most enjoyable and stressful events since the previous prompt."

The text responses were analyzed using OpenAI's GPT-4o model, as well as by a human "rater" for one-fourth of responses, to evaluate consistency between human and AI ratings. The team found "substantial agreement" between the two.

As for the "silent" real-time monitoring made possible by the smartphone, this data was used to measure activity levels using the intensity of physical activity during the day; the percentage of time a participant spent at home; the distance and "mobility area" traveled each day relative to the home; and places visited each day.

While studies using passive smartphone data can be designed to be entirely unobtrusive, this project intentionally combined passive and interactive elements to test whether the combination of LLM-based text analysis and passive mobility data could provide real-time, clinically relevant insights.

The study produced interesting results. The most important, perhaps, was that "activation" among the depressed and anhedonic adolescents—as deduced from passive sensors in their smartphones—correlated positively with "activation ratings" generated by GPT analysis of their texts and patients' activation self-report score over the course of therapy.

On the GPS side, greater activation corresponded to more places visited and more time spent away from home, though not distance traveled. In addition, increases in GPT-rated activation were associated with higher daily positive and lower daily negative affect. The team also found that smartphone passive sensing features could be used to forecast weekly improvements in anhedonia and depressive symptoms.

The results also led the researchers to suggest that "LLMs such as GPT can extract psychologically meaningful information from unstructured text." Past work had shown that LLMs can be used to analyze language from in-person psychotherapy sessions to inform clinical decision-making. "Importantly, our study shows that LLM-based assessments can also provide clinically relevant insights based on language generated outside the therapy room, offering scalable and unobtrusive ways to monitor therapeutic processes in patients' daily lives."

"This implies that daily text assessments could help clinicians monitor" the response to behavioral activation therapy "in real time, while mobility patterns could indicate whether treatment is gradually translating into symptom improvement." They concluded that the tools they tested "hold promise for advancing data-informed psychotherapy by tracking therapeutic processes in real time, reducing reliance on self-report and enabling personalized, adaptive interventions." ❖

The team also included: **Diego A. Pizzagalli, Ph.D.**, 2017 BBRF Distinguished Investigator, 2008 Independent Investigator; and **Erika E. Forbes, Ph.D.**, 2014 BBRF Independent Investigator, 2006 Young Investigator.

Deep-Brain Stimulation Guided by Brain Mapping Resulted in Rapid & Acute Reduction in Severe OCD Symptoms in Trial Patient

Next-Generation Therapies: Obsessive-Compulsive Disorder (OCD)

Story Highlights:

Researchers developed and successfully tested in a first patient a novel protocol designed to identify optimal personalized brain stimulation targets for implantation of a deep-brain stimulation device to treat severe, treatment-resistant OCD. DBS in this patient rapidly and sharply reduced symptoms.

Journal:
Translational Psychiatry
October 31, 2025

A research team led by a recent BBRF grantee has developed and tested in a first patient a novel protocol designed to identify optimal personalized brain stimulation targets for severe, treatment-resistant OCD. Once identified, the team used these targets to guide precise implantation of a deep-brain stimulation (DBS) device whose subsequent activation resulted in rapid and acute reduction of the patient's OCD symptoms (62% lower 6 months after the procedure).

The study was led by **Andrew Moses Lee, M.D., Ph.D.**, a 2020 BBRF Young Investigator grantee, and senior team members **Andrew D. Krystal, M.D.**, recipient of BBRF Young Investigator grants in 1997 and 1993, and Edward F. Chang, M.D., all at the University of California, San Francisco. They joined colleagues in reporting in the journal *Translational Psychiatry* results of their unique FDA-approved clinical trial involving a single OCD patient.

A woman in her 20s, she suffered from "extreme, treatment-refractory OCD," with symptoms consisting mostly of harm-based obsessions associated with checking compulsions. "These symptoms were disabling, occupied most of her waking hours, caused her to leave nursing school, and prevented her from driving or leaving home," the team noted. She had not responded to multiple trials of serotonin reuptake inhibitors (SRIs), or to augmentation strategies, outpatient and intensive residential therapy, or transcranial magnetic stimulation (TMS).

TMS, a non-invasive method in which neural stimulation is delivered on an outpatient basis, has been tested in OCD, but is not effective in many patients (the response rate is much higher in patients with depression). Deep-brain stimulation, in contrast, is invasive: it entails brain surgery, remains experimental, and is only considered as a potential treatment option, whether in depression or OCD, in severe cases involving disabling or life-threatening symptoms that have not responded to multiple courses of other therapies.

DBS has been tested previously in a number of OCD patients. The methods used have resulted in responses in about 60% of patients, and average symptom reductions of around 40%. But, the team notes, there has been a "lack of consistency in the response to any individual target" in these DBS trials. This could be due to variations in the underlying circuit abnormalities causing symptoms across these patients, anatomical variation across individuals, or poor engagement of the intended circuit target in specific cases.

"To address these challenges, we developed a protocol that aimed to identify personalized neuromodulation [i.e., brain stimulation] targets," the researchers explained. They adapted methods commonly used in epilepsy to guide surgical treatment. Several years

Andrew Moses Lee, M.D., Ph.D.

University of California, San Francisco
2020 BBRF Young Investigator

Andrew D. Krystal, M.D.

University of California, San Francisco
1997, 1993 BBRF Young Investigator

ago, Dr. Krystal and 2018 BBRF Young Investigator **Katherine W. Scangos, M.D., Ph.D.**, used a similar approach to identify treatment targets prior to implantation of a DBS device for a patient with life-threatening severe refractory depression.

The protocol unfolds in three stages. First, the patient undergoes an invasive procedure to implant a series of electrodes, in this case across a circuit known to be implicated in OCD called the cortico-striato-thalamo-cortical circuit (CSTC). Then, a process called stimulation mapping ensues. Over a period of days, the team delivers varying amounts of stimulation to the implanted electrodes, individually and in various combinations. The aim here is to find the specific sites whose stimulation acutely reduces OCD symptoms. (The patient, if not experiencing symptoms, is given prompts designed to generate symptoms while these tests are under way.) Stimulation of some sites is observed to cause adverse effects; these are avoided in preparing a protocol for therapeutic DBS stimulation. The third phase involves multiple 20-minute stimulation sessions of the top candidate stimulation sites, with actual stimulation and a placebo version delivered randomly (neither the patient nor the doctors delivering the stimulation know when the "real" stimulation is being delivered.) Results of these tests enabled the team to plan where exactly in the brain to implant the DBS device. This surgery was performed 8 weeks after the mapping electrodes were removed. Two weeks after DBS implantation, the device was programmed to deliver optimal clinical improvement, based on the patient's real-time responses.

All of this work enabled the team to discover that at least in this patient, "high frequency activity" of neural activation within the CTSC circuit correlates with OCD symptom severity. Tests in future subjects will begin to enable the team to say whether this correlation is present in all or many OCD patients, or not. In this case, the preliminary mapping and probing enabled the team to identify two highly specific targets for DBS stimulation to reduce symptoms. These targets are both within the brain's right ventral capsule (VC), an area involved in connecting the prefrontal cortex and the ventral striatum, which is implicated in emotion processing and behavior.

Combined stimulation of these targets acutely and rapidly reduced the patient's severe OCD symptoms. Prolonged stimulation of the VC targets via DBS suppressed high frequency activity in the orbitofrontal and cingulate cortices, which were found to be structurally and functionally connected. This result leads the team to describe these sites as "cortical nodes encoding the severity of OCD symptoms."

"This case provides the first proof-of-concept that invasive brain mapping can be used to guide a novel personalized, multi-site neuromodulation approach to treat refractory OCD," the team wrote. The results "open up the possibility of personalized neuromodulatory therapies."

Will the results in this patient generalize to others with severe OCD symptoms? The two stimulation sites identified by preliminary brain mapping are similar but not identical to those targeted in past experiments with DBS. About 40% of OCD patients in those experiments did not respond to DBS. This suggests the importance of performing the preliminary mapping phase of the protocol in other patients, to determine if in some refractory patients alternate stimulation sites might generate a response like the one seen in the just-reported trial. On the other hand, if the stimulation sites used in this study do tend to generalize, it may indicate a circuit dysfunction in severe OCD that is commonplace, and which might be "consistently targeted to achieve therapeutic responses." This in turn would argue against the need to personalize the targeting, an invasive and difficult procedure for the patient that it would be advantageous to avoid, if possible. ❖

The team also included **Joline M. Fan, M.D.**, 2022 BBRF Young Investigator.

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Ralph DiLeone, Ph.D.
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C. Neill Epperson, M.D.
André Fenton, Ph.D.
Robert L. Findling, M.D., MBA
Cecilia Flores, Ph.D.
Stan B. Floresco, Ph.D.
Erika Forbes, Ph.D.
Alan Frazer, Ph.D.
Robert Freedman, M.D.
Aurelio Galli, Ph.D.
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David Goldman, M.D.
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Rita Z. Goldstein, Ph.D.
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René Hen, Ph.D.
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Elliot Hong, M.D.
Laura Marianne Huckins, Ph.D.
Alicia Izquierdo, Ph.D.
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Ellen E. Lee, M.D.
Francis S. Lee, M.D., Ph.D.
Robert H. Lenox, M.D.
David A. Lewis, M.D.
Jeffrey A. Lieberman, M.D.
Gregory A. Light, Ph.D.
Kelvin O. Lim, M.D.
Sarah Hollingsworth Lisanby, M.D.
Conor Liston, M.D., Ph.D.
Daniel Lodge, Ph.D.
Joan L. Luby, M.D.
Irwin Lucki, Ph.D.
Beatriz Luna, Ph.D.
Gary Lynch, Ph.D.
Robert C. Malenka, M.D., Ph.D.
Anil K. Malhotra, M.D.
J. John Mann, M.D.
Stephen Maren, Ph.D.
Keri Martinowich, Ph.D.
Daniel H. Mathalon, Ph.D., M.D.
Helen S. Mayberg, M.D.
Colleen A. McClung, Ph.D.
Ronald McKay, Ph.D.
Kathleen R. Merikangas, Ph.D.
Guo-Li Ming, M.D., Ph.D.
Karoly Mirnics, M.D., Ph.D.
Bita Moghaddam, Ph.D.
Lisa M. Monteggia, Ph.D.
David A. Morilak, Ph.D.
James W. Murrough, M.D., Ph.D.
Charles B. Nemeroff, M.D., Ph.D.
Eric J. Nestler, M.D., Ph.D.
Thomas C. Neylan, Ph.D.
Andrew A. Nierenberg, M.D.
Patricio O'Donnell, M.D., Ph.D.
Dost Ongur, M.D., Ph.D.
Maria A. Oquendo, M.D., Ph.D.
Sachin Patel, M.D., Ph.D.
Martin P. Paulus, M.D.
Godfrey D. Pearlson, M.D., Ph.D.
Peter Penzes, Ph.D.
Mary L. Phillips, M.D., M.D. (CANTAB)
Marina R. Picciotto, Ph.D.
Daniel S. Pine, M.D.
Diego A. Pizzagalli, Ph.D.
Robert M. Post, M.D.
James B. Potash, M.D., M.P.H.
Susan Powell, Ph.D.
Pasko Rakic, M.D., Ph.D.
Kerry J. Ressler, M.D., Ph.D.
Victoria B. Risbrough, Ph.D.
Carolyn Rodriguez, M.D., Ph.D.
Bryan L. Roth, M.D., Ph.D.
Panos Roussos, M.D., MS., Ph.D.
Laura M. Rowland, Ph.D.
Peter H. Rudebeck, Ph.D.
Scott J. Russo, Ph.D.
Gerard Sanacora, M.D., Ph.D.
Akira Sawa, M.D., Ph.D.
Alan F. Schatzberg, M.D.
Nina R. Schooler, Ph.D.
Robert Schwarcz, Ph.D.
Srijan Sen, M.D., Ph.D.
Yvette I. Sheline, M.D.
David A. Silbersweig, M.D.
Vikaas S. Sohal, M.D., Ph.D.
Matthew W. State, M.D., Ph.D.
Murray B. Stein, M.D., M.P.H., F.R.C.F.C.
Joanna E. Steinglass, M.D.
Stephen M. Strakowski, M.D.
Carol A. Tamminga, M.D.
Laurence H. Tecott, M.D., Ph.D.
Flora M. Vaccarino, M.D., Ph.D.
Rita J. Valentino, Ph.D.
Jeremy M. Veenstra-VanderWeele, M.D.
Susan M. Voglmaier, M.D., Ph.D.
Aristotle N. Voineskos, M.D., Ph.D.
Nora D. Volkow, M.D.
Karen Dineen Wagner, M.D., Ph.D.
Daniel R. Weinberger, M.D.
Myrna M. Weissman, Ph.D.
Rachel Yehuda, Ph.D.
Jared W. Young, Ph.D.
L. Trevor Young, M.D., Ph.D., F.R.C.P.C., F.C.A.H.S.
Carlos A. Zarate, Jr., M.D.
Jon-Kar Zubieta, M.D., Ph.D.

2025 New BBRF Scientific Council Members

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

This group of 191 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.

Emery N. Brown, M.D., Ph.D.
Massachusetts Institute of Technology

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

Matthew Hill, Ph.D.
Hotchkiss Brain Institute, University of Calgary
2010 Young Investigator Grantee

Sheena Josselyn, Ph.D.
The Hospital for Sick Children (SickKids)
University of Toronto, Canada
2014 Independent Investigator Grantee
2000, 1998 Young Investigator Grantee

Atsushi Kamiya, M.D., Ph.D.
Johns Hopkins School of Medicine
2009, 2007 Young Investigator Grantee

Conor Liston, M.D., Ph.D.
Weill Cornell Medical College
2013 Young Investigator Grantee

James W. Murrough, M.D., Ph.D.
Icahn School of Medicine at Mount Sinai
2009 Young Investigator Grantee

Sachin Patel, M.D., Ph.D.
Northwestern University Feinberg School of Medicine
2015 Independent Investigator Grantee

Diego A. Pizzagalli, Ph.D.
University of California, Irvine
2017 Distinguished Investigator Grantee
2008 Independent Investigator Grantee

Panos Roussos, M.D., Ph.D.
Friedman Brain Institute
Icahn School of Medicine at Mount Sinai
2013 Young Investigator Grantee

Peter H. Rudebeck, Ph.D.
Icahn School of Medicine at Mount Sinai
2015 Young Investigator Grantee

Srijan Sen, M.D., Ph.D.
University of Michigan

"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

2025 BBRF GRANTEES

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

BY THE NUMBERS SINCE 1987

As of March 2026

AWARDED TO SCIENTISTS

$476 MILLION

GRANTS

6,886

GRANTEES

5,731

UNIVERSITIES & MEDICAL CENTERS

611

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 2025 Distinguished Investigator (DI) Grants

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

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 2025 Distinguished Investigators are exploring new frontiers in understanding a wide range of neuropsychiatric illnesses, including opioid use disorder, depression in pregnant women, schizophrenia, bipolar disorder, and the effects of psychedelics on perception and consciousness.

"At WoodNext Foundation, we believe that bold, high-impact scientific research is essential to advancing our understanding of mental health and improving lives. We are honored to support BBRF's Distinguished Investigator Grants, which empower leading researchers to push the boundaries of knowledge and develop innovative approaches to mental illness."

Nancy Chan, *Executive Director of the WoodNext Foundation*

"Mental illnesses affect millions of individuals and families, yet there is still so much to learn about the underlying biology and potential treatments. By supporting bold, innovative research, our Distinguished Investigator Grants empower leading scientists to pursue ideas that could pave the way for major breakthroughs in preventing, diagnosing, and treating psychiatric illnesses. We are deeply grateful to the WoodNext Foundation for their generous support, which makes it possible to fund these pioneering studies."

Dr. Jeffrey Borenstein

2025 BBRF Distinguished Investigator Grants by Illness

ADDICTION / SUBSTANCE USE DISORDERS

Rita Goldstein, Ph.D.
Diagnostic Tools/Early Intervention

Venetia Zachariou, Ph.D.
Basic Research

AUTISM SPECTRUM DISORDERS

Sagiv Shifman, Ph.D.
Basic Research

BIOLOGY OF THE BRAIN

These projects focus on how the brain works

NEURAL CIRCUIT MODULATION

Eva S. Anton, Ph.D.
Basic Research

MARKERS OF BRAIN INFLAMMATION

Marek Kubicki, M.D., Ph.D.
Diagnostic Tools/Early Intervention

PSYCHEDELICS AND PERCEPTION

Doris Tsao, Ph.D.
Basic Research

BIPOLAR DISORDER

Jared W. Young, Ph.D.
Basic Research
Next-Generation Therapies

DEPRESSION

Flavio Frohlich, Ph.D.
Next-Generation Therapies

OBESSSIVE-COMPULSIVE DISORDER (OCD)

Christopher J. Pittenger, M.D., Ph.D.
Basic Research

SCHIZOPHRENIA

Stanislav S. Zakharenko, M.D., Ph.D.
Basic Research

"We are thrilled by this year's slate of Distinguished Investigator award winners. Their proposals represent highly innovative approaches across the spectrum of neuropsychiatric disorders and leveraging research approaches from basic scientific analysis of animal and cell models to translational, mechanism-based studies in humans. I am especially appreciative of the outstanding work by BBRF's Scientific Council members who evaluated an unusually competitive pool of applications to identify the winning proposals."

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

BBRF Young Investigator Grants in 2025

895 YOUNG INVESTIGATOR APPLICATIONS

165 YOUNG INVESTIGATOR GRANTS AWARDED

$11.4 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. Judith Ford

2025 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

Gabrielle Agin-Liebes, Ph.D.
Next-Generation Therapies

Kevin Braunscheidel, Ph.D.
Basic Research
Next-Generation Therapies

Yusmaris Cariaco, Ph.D.
Basic Research

Ahmet O. Ceceli, Ph.D.
Basic Research

Kurt M. Fraser, Ph.D.
Basic Research

Jacqueline Giovanniello, Ph.D.
Basic Research

Joao Guassi Moreira, Ph.D.
Diagnostic Tools/Early Intervention

Kwang-Hyun Hur, Ph.D.
Next-Generation Therapies

Anel A. Jaramillo, Ph.D.
Next-Generation Therapies

Paul F. Kramer, Ph.D.
Basic Research

Karolina M. Lempert, Ph.D.
Basic Research

Ofir Livne, M.D.
Diagnostic Tools/Early Intervention

Travis T. Mallard, Ph.D.
Basic Research

Edward H. Nieh, Ph.D.
Basic Research

Alexios Panoutsopoulos, Ph.D.
Basic Research

Kristin Perry, Ph.D.
Basic Research

Nicole Petersen, Ph.D.
Basic Research

Sema G. Quadir, M.D.
Basic Research

Siara K. Rouzer, Ph.D.
Basic Research

Jason M. Tucciarone, M.D., Ph.D.
Basic Research

Jonathan W. VanRyzin, Ph.D.
Basic Research

Joseph M. Villarin, M.D., Ph.D.
Basic Research

Leigh C. Walker, Ph.D.
Basic Research

Junshi Wang, Ph.D.
Basic Research

Lu Wang, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Heather B. Ward, M.D.
Next-Generation Therapies

Belgin Yalcin, Ph.D.
Basic Research

Junhua Yang, Ph.D.
Basic Research

Lynn Yap, Ph.D.
Basic Research

ATTENTION-DEFICIT HYPERACTIVITY DISORDER (ADHD)

Camila S.A. Cosmo, M.D.
Next-Generation Therapies

Nicholas D. Fogleman, Ph.D.
Basic Research

Travis T. Mallard, Ph.D.
Basic Research

Xiangling Meng, Ph.D.
Basic Research

Nadezhda (Nadya) N. Modyanova, Ph.D.
Basic Research
Next-Generation Therapies

Ali Mohebi, Ph.D.
Basic Research

Tadaaki Nishioka, Ph.D.
Basic Research

Alexios Panoutsopoulos, Ph.D.
Basic Research

Lisa Pavinato, Ph.D.
Basic Research

Ashley Song, Ph.D.
Basic Research

ANXIETY DISORDERS

Gabrielle Agin-Liebes, Ph.D.
Next-Generation Therapies

Sophie Bagur, Ph.D.
Basic Research

Ritchie Chen, Ph.D.
Basic Research

María del Carmen Camarena Delgado, Ph.D.
Basic Research

Josepheen De Asis-Cruz, M.D., Ph.D.
Basic Research

Raoni C. dos Santos, Ph.D.
Basic Research

Kyle Harrington Flippo, Ph.D.
Basic Research

Rodolfo Flores Garcia, Ph.D.
Basic Research

Pegah Kassraian, Ph.D.
Basic Research

Annelise A. Madison, Ph.D.
Basic Research

Laura Modol, Ph.D.
Basic Research

Jessie Muir, Ph.D.
Next-Generation Therapies

Kally C. O'Reilly Sparks, Ph.D.
Basic Research

Alexios Panoutsopoulos, Ph.D.
Basic Research

Puja K. Parekh, Ph.D.
Basic Research

Payam Piray, Ph.D.
Basic Research

Nicole R. Provenza, Ph.D.
Next-Generation Therapies

Luis E. Rosas-Vidal, M.D., Ph.D.
Basic Research

Fangmiao Sun, Ph.D.
Basic Research

Tomoki Suzuki, M.D., Ph.D.
Basic Research

Kristin L. Szuhany, Ph.D.
Basic Research
Next-Generation Therapies

Sarah M. Tashjian, Ph.D.
Basic Research

Michael Totty, Ph.D.
Basic Research

Sebnem N. Tuncdemir, Ph.D.
Basic Research

Milenna T. van Dijk, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Weizhen Xie, Ph.D.
New Technologies
Next-Generation Therapies

Rongzhen Yan, Ph.D.
Basic Research

Bohan Zhao, Ph.D.
Basic Research

AUTISM SPECTRUM DISORDER (ASD)

Kyle Harrington Flippo, Ph.D.
Basic Research

Xiangling Meng, Ph.D.
Basic Research

Nadezhda (Nadya) N. Modyanova, Ph.D.
Basic Research
Next-Generation Therapies

Hemanth Mohan, Ph.D.
Basic Research

Mohammed A. Mostajo-Radji, Ph.D.
Basic Research

Mauricio M. Oliveira, Ph.D.
Basic Research

Lisa Pavinato, Ph.D.
Basic Research

Giorgia Picci, Ph.D.
Basic Research

Ashley Song, Ph.D.
Basic Research

Fangmiao Sun, Ph.D.
Basic Research

Tomoki Suzuki, M.D., Ph.D.
Basic Research

BIOLOGY OF THE BRAIN

These projects focus on how the brain works

MARKERS OF PRENATAL ENVIRONMENTAL UNPREDICTABILITY

Annie Aitken, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

ANHEDONIA, IMMUNE MARKERS & MENOPAUSAL TRANSITION

Erin M. Bondy, Ph.D.
Basic Research

INTEROCEPTION, THE INSULA, & EMOTIONS

Ritchie Chen, Ph.D.
Basic Research

PREDICTIVE PROCESSING & COGNITIVE DYSFUNCTION

Steven P. Errington, Ph.D.
Basic Research

MODULATION OF SYNAPTIC STRENGTH

Linlin Fan, Ph.D.
New Technologies

PSYCHEDELICS & THE SEROTONIN 2-A RECEPTOR

Ryan H. Gumpper, Ph.D.

Basic Research

PSYCHEDELIC-INDUCED PLASTICITY

Sarah J. Jefferson, M.D., Ph.D.

Basic Research

EPIDEMIOLOGY OF GENETIC RISK

Sonja LaBianca, M.D., Ph.D.

Diagnostic Tools/Early Intervention

INFERRING COGNITIVE STATES FROM SPEECH

Baihan Lin, Ph.D.

Diagnostic Tools/Early Intervention

Basic Research

MUSCLE IMAGING & EMOTIONAL STATES

Timothy A. Machado, Ph.D.

New Technologies

Basic Research

EARLY SOCIAL EXPERIENCE & NEURONAL DEVELOPMENT

Kally C. O'Reilly Sparks, Ph.D.

Basic Research

SOCIAL FACTORS & MATERNAL MENTAL HEALTH

Elizabeth Sahagun, Ph.D.

Basic Research

NEURAL ACTIVITY PATTERNS IN PSYCHIATRIC ILLNESSES

Ethan A. Solomon, M.D., Ph.D.

Diagnostic Tools/Early Interventions

BIPOLAR DISORDER

Clara Albinana, Ph.D.

Basic Research

Natalie Bareis, Ph.D.

Next-Generation Therapies

Natalia E. Fares-Otero, Ph.D.

Basic Research

Yang Ge, Ph.D.

Basic Research

Kwang-Hyun Hur, Ph.D.

Next-Generation Therapies

Giacomo Maddaloni, Ph.D.

Basic Research

Lindsay M. Melhuish Beaupre, Ph.D.

Diagnostic Tools/Early Intervention

Caitlin E. Millett, Ph.D.

Basic Research

Jean-Paul G. Noel, Ph.D.

Basic Research

Anthony D. Ramnauth, Ph.D.

Basic Research

Jennifer E. Siegel-Ramsay, Ph.D.

Basic Research

Shivang Sullere, Ph.D.

Basic Research

Brittany J. Wolff, Ph.D.

Basic Research

CHILDHOOD & ADOLESENCE

Lorenza Dall'Aglio, Ph.D.

Next-Generation Therapies

Nicholas D. Fogleman, Ph.D.

Basic Research

Joao Guassi Moreira, Ph.D.

Diagnostic Tools/Early Intervention

Clotilde Guidetti, M.D.

Next-Generation Therapies

Carina Heller, Ph.D.

Basic Research

Nicole R. Karcher, Ph.D.

Diagnostic Tools/Early Intervention

Basic Research

Christina A.G. Laurenzi, Ph.D.

Next-Generation Therapies

Bochao D. Lin, Ph.D.

Diagnostic Tools/Early Intervention

Travis T. Mallard, Ph.D.

Basic Research

Lluís Miquel Rio, Ph.D.

Basic Research

Laura Modol, Ph.D.

Basic Research

Nadezhda (Nadya) N. Modyanova, Ph.D.

Basic Research

Next-Generation Therapies

Kally C. O'Reilly Sparks, Ph.D.

Basic Research

Giorgia Picci, Ph.D.

Basic Research

Reza Rahimian, Ph.D.

Basic Research

Prithviraj Rajebhosale, Ph.D.

Basic Research

Siara K. Rouzer, Ph.D.
Basic Research

Jennifer E. Siegel-Ramsay, Ph.D.
Basic Research

Ashley Song, Ph.D.
Basic Research

Sarah M. Tashjian, Ph.D.
Basic Research

Milenna T. van Dijk, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Alecia C. Vogel, M.D., 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

Gabrielle Agin-Liebes, Ph.D.
Next-Generation Therapies

Clara Albinana, Ph.D.
Basic Research

Benedetta Bigio, Ph.D.
Basic Research

Gustavo Costa Medeiros, M.D.
Next-Generation Therapies

Iris Dalhuisen, Ph.D.
Next-Generation Therapies

Lorenza Dall'Aglio, Ph.D.
Next-Generation Therapies

Raoni C. dos Santos, Ph.D.
Basic Research

Rand Eid, Ph.D.
Basic Research

Meghan E. Flanigan, Ph.D.
Next-Generation Therapies

Rodolfo Flores Garcia, Ph.D.
Basic Research

Clotilde Guidetti, M.D.
Next-Generation Therapies

Carina Heller, Ph.D.
Basic Research

Kwang-Hyun Hur, Ph.D.
Next-Generation Therapies

Jenessa N. Johnston, Ph.D.
Basic Research
Next-Generation Therapies

Miranda Koloski, Ph.D.
Basic Research

Christina A.G. Laurenzi, Ph.D.
Next-Generation Therapies

Younga H. Lee, Ph.D.
Diagnostic Tools/Early Intervention

Long Li, Ph.D.
Basic Research

Andrea Locarno, Ph.D.
Basic Research

Annelise A. Madison, Ph.D.
Basic Research

Magdalena Martinez Garcia, Ph.D.
Diagnostic Tools/Early Intervention

Lindsay M. Melhuish Beaupre, Ph.D.
Diagnostic Tools/Early Intervention

Christina Metcalf, Ph.D.
Next-Generation Therapies

Akiko Mizuno, Ph.D.
Basic Research

Daniel P. Moriarity, Ph.D.
Next-Generation Therapies

Jessie Muir, Ph.D.
Next-Generation Therapies

Tadaaki Nishioka, Ph.D.
Basic Research

Jean-Paul G. Noel, Ph.D.
Basic Research

Jessica A. Osterhout, Ph.D.
Basic Research

Puja K. Parekh, Ph.D.
Basic Research

Nicole Petersen, Ph.D.
Basic Research

Mohsen Poorganji, Ph.D.
Next-Generation Therapies

Reza Rahimian, Ph.D.
Basic Research

Anthony D. Ramnauth
Basic Research

Zachary P. Rosenthal, M.D., Ph.D.
New Technologies

Mohammad S.E. Sendi, Ph.D.
Next-Generation Therapies

Tsvetan Serchov, Ph.D.
Basic Research

Christopher V. Sikes-Keilp, M.D.
Basic Research

Kunmi Sobowale, M.D.
Diagnostic Tools/Early Intervention

Fangmiao Sun, Ph.D.
Basic Research

Kristin L. Szuhany, Ph.D.
Basic Research
Next-Generation Therapies

Joshua R. Tatz, Ph.D.
Basic Research

Ye E. Tian, Ph.D.
Diagnostic Tools/Early Intervention

Milenna T. van Dijk, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Cassis Varlow, Ph.D.
Next-Generation Therapies

Alecia C. Vogel, M.D., Ph.D.
Basic Research

Lauren N. Woodie, Ph.D.
Basic Research

Rongzhen Yan, Ph.D.
Basic Research

Bohan Zhao, Ph.D.
Basic Research

EATING DISORDERS

Melissa L. Cooper, Ph.D.
Basic Research

Kwang-Hyun Hur, Ph.D.
Next-Generation Therapies

Salvador Valencia Sanchez, Ph.D.
Basic Research

Elizabeth A. Velkoff, Ph.D.
Diagnostic Tools/Early Intervention

Bohan Zhao, Ph.D.
Basic Research

OBSESSIVE-COMPULSIVE DISORDER (OCD)

Rebecca C. Cox, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Meghan J. Kulak, M.D.
Next-Generation Therapies

Elizabeth E. Manning, Ph.D.
Next-Generation Therapies

Nicole R. Provenza, Ph.D.
Next-Generation Therapies

Joseph M. Villarin, M.D., Ph.D.
Basic Research

OTHER DISORDERS

ALZHEIMER'S DISEASE

Benedetta Bigio, Ph.D.
Basic Research

Lorenzo Pasquini, Ph.D.
Next-Generation Therapies

ANTISOCIAL PERSONALITY DISORDER

Long Li, Ph.D.
Basic Research

CONDUCT DISORDER

Travis T. Mallard, Ph.D.
Basic Research

MORAL INJURY & FORENSIC PSYCHIATRIC SERVICES

Ilvy Goossens, Ph.D.
Diagnostic Tools/Early Intervention

POST-TRAUMATIC STRESS DISORDER (PTSD)

Gabrielle Agin-Liebes, Ph.D.
Next-Generation Therapies

Ariana Anderson, Ph.D.
Diagnostic Tools/Early Intervention

Victor Cazares, Ph.D.
Basic Research
Next-Generation Therapies

Jennifer J. Donegan, Ph.D.
Basic Research

Kyle Harrington Flippo, Ph.D.
Basic Research

Rodolfo Flores Garcia, Ph.D.
Basic Research

Long Li, Ph.D.
Basic Research

Mauricio M. Oliveira, Ph.D.
Basic Research

Luis E. Rosas-Vidal, M.D., Ph.D.
Basic Research

Adam Steel, Ph.D.
Basic Research

Weinan Sun, Ph.D.
Basic Research

Joshua R. Tatz, Ph.D.
Basic Research

Michael Totty, Ph.D.
Basic Research

Sebnem N. Tuncdemir, Ph.D.
Basic Research

PRENATAL BRAIN DEVELOPMENT

Annie Aitken, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Yusmaris Cariaco, Ph.D.
Basic Research

Josepheen De Asis- Cruz, M.D., Ph.D.
Basic Research

Claudia Z. Han, Ph.D.
Basic Research

Deepak A. Kaji, M.D., Ph.D.
Basic Research
Next-Generation Therapies

Xiangling Meng, Ph.D.
Basic Research

Mohammed A. Mostajo-Radji, Ph.D.
Basic Research

Alexios Panoutsopoulos, Ph.D.
Basic Research

Belgin Yalcin, Ph.D.
Basic Research

PSYCHOSIS

Sidhant Chopra, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Danique Jeurissen, Ph.D.
Basic Research

Nicole R. Karcher, Ph.D.
Diagnostic Tools/Early Intervention
Basic Research

Benson S. Ku, M.D., Ph.D.
Basic Research

Baihan Lin, Ph.D.
Diagnostic Tools/Early Intervention
Basic Research

Bochao D. Lin, Ph.D.
Diagnostic Tools/Early Intervention
Basic Research

Lauren Luther, Ph.D.
Next-Generation Therapies

Katie F.M. Marwick, M.D., Ph.D.
Diagnostic Tools/Early Intervention

Christina Mo, Ph.D.
Basic Research

Weibo Niu, Ph.D.
Basic Research
Next-Generation Therapies

Jean-Paul G. Noel, Ph.D.
Basic Research

Prithviraj Rajebhosale, Ph.D.
Basic Research

Alfredo L. Sklar, M.D., Ph.D.
Basic Research

Joseph M. Villarin, M.D., Ph.D.
Basic Research

SCHIZOPHRENIA

Jamie A. Abbott, Ph.D.
Basic Research
Next-Generation Therapies

Novin Balafkan, Ph.D.
Next-Generation Therapies
Basic Research

Samuel T. Barlow, Ph.D.
Basic Research

Kynon J. Benjamin, Ph.D.
Basic Research

Alexandra S. Bova, Ph.D.
Basic Research

Philip D. Campbell, M.D., Ph.D.
Basic Research

Runnan Cao, Ph.D.
Basic Research

Sidhant Chopra, Ph.D.
Basic Research
Diagnostic Tools/Early Intervention

Dibyadeep Datta, Ph.D.
Basic Research

Steven P. Errington, Ph.D.
Basic Research

Pawan Sirwan Faris, Ph.D.
Basic Research

Marc Forrest, Ph.D.
Basic Research

Yang Ge, Ph.D.
Basic Research

Claudia Z. Han, Ph.D.
Basic Research

Benxia Hu, Ph.D.
Basic Research

Kwang-Hyun Hur, Ph.D.
Next-Generation Therapies

Alison R. Hwong, M.D., Ph.D.
Diagnostic Tools/Early Intervention

Danique Jeurissen, Ph.D.
Basic Research

Deepak A. Kaji, M.D., Ph.D.
Basic Research
Next-Generation Therapies

Nicole R. Karcher, Ph.D.
Diagnostic Tools/Early Intervention
Basic Research

Pegah Kassraian, Ph.D.
Basic Research

Bas Lendemeijer, Ph.D.
Basic Research

Baihan Lin, Ph.D.
Diagnostic Tools/Early Intervention
Basic Research

Christina Mo, Ph.D.
Basic Research

Hemanth Mohan, Ph.D.
Basic Research

Ali Mohebi, Ph.D.
Basic Research

Mohammed A. Mostajo-Radji, Ph.D.
Basic Research

Weibo Niu, Ph.D.
Basic Research
Next-Generation Therapies

Jean-Paul G. Noel, Ph.D.
Basic Research

Nicole Petersen, Ph.D.
Basic Research

Prithviraj Rajebhosale, Ph.D.
Basic Research

Anthony D. Ramnauth, Ph.D.
Basic Research

Paul K. Reardon, M.D., Ph.D.
Basic Research

Tal Sharf, Ph.D.
Basic Research
New Technologies

Alfredo L. Sklar, M.D., Ph.D.
Basic Research

Weinan Sun, Ph.D.
Basic Research

Tomoki Suzuki, M.D., Ph.D.
Basic Research

Joseph M. Villarin, M.D., Ph.D.
Basic Research

Heather B. Ward, M.D.
Next-Generation Therapies

Hui Yang, Ph.D.
Basic Research

Marco Zierhut, M.D.
Next-Generation Therapies

SUICIDE PREVENTION

Christina A.G. Laurenzi, Ph.D.
Next-Generation Therapies

Lluís Miquel Rio, Ph.D.
Basic Research

Reza Rahimian, Ph.D.
Basic Research

2025 Young Investigators: Institutional Affiliations at the Time of the Grant

(in alphabetical order)

Aarhus University, Denmark

Adelphi University

Baylor College of Medicine (2)

Boys Town National Research Hospital

The Broad Institute of MIT

Butler Hospital

Centre for Addiction and Mental Health, University of Toronto, Canada

Centre National de la Recherche Scientifique, France

Charité - University Medicine Berlin, Germany

Children's National Medical Center

Chinese Academy of Sciences, People's Republic of China

Columbia University (3)

Cornell University

Creedmoor Psychiatric Center

Dell Medical School, University of Texas at Austin

Drexel University

Emory Clinic

Emory University

ESPCI Paris, France

Feinstein Institute for Medical Research/Northwell Health

Fundacio Institut Mar d'Investigacions Mediques, Spain

George Washington University

Harvard Medical School

Harvard University (2)

Haukeland University Hospital, Norway

Icahn School of Medicine at Mount Sinai (7)

Institut D'investigacions Biomèdiques August Pi I Sunyer, Spain

Institute of Biomedicine & Biotechnology of Cantabria, Spain

Johns Hopkins University School of Medicine (2)

Maastricht University, The Netherlands

Massachusetts General Hospital (5)

Massachusetts Institute of Technology

Mayo Clinic, Jacksonville

McGill University, Canada (2)

McLean Hospital (2)

McMaster University, Canada

Medical University of South Carolina

Montana State University

National Institute of Mental Health (NIMH/NIH) (2)

New York University (4)

New York University Medical Center

New York University School of Medicine (2)

Northwestern University (4)

Ocean State Research Institute, Inc.

Orygen, Australia

Princeton University

Psychiatric Center Ballerup, Mental Health Services of the Capital Region of Denmark

Research Foundation for Mental Hygiene, Inc./NKI (3)

Research Foundation for Mental Hygiene, Inc./ NYSPI (2)

Research Foundation for the State University of New York (SUNY)/University at Buffalo

The Rockefeller University

Scripps Research

Stanford University (4)

Stellenbosch University, South Africa

Stockholm University, Sweden

Temple University

Texas A&M Health Science Center

Texas A&M University

Università della Svizzera Italiana, Italy

University of Alabama at Birmingham

University of Cadiz, Spain

University of California, Davis (2)

University of California, Los Angeles (5)

University of California, San Diego (2)

University of California, San Francisco (2)

University of California, Santa Barbara

University of California, Santa Cruz (2)

University of Colorado Anschutz Medical Campus

University of Colorado, Boulder

University of Connecticut Health Cente

University of Edinburgh, UK

University of Illinois at Urbana-Champaign

University of Iowa (2)

University of Iowa College of Medicine

University of Kentucky

University of Maryland

University of Maryland, Baltimore (2)

University of Melbourne, Australia (4)

University of Michigan (2)

University of Minnesota (3)

University of Newcastle, UK (2)

University of Nijmegen, The Netherlands

University of North Carolina at Chapel Hill (5)

University of Oregon

University of Ottawa, Canada

University of Pavia, Italy

University of Pennsylvania

University of Pennsylvania School of Medicine (3)

University of Pittsburgh (2)

University of South Carolina

University of Southern California

University of Texas at Austin

University of Texas at Dallas

University of Texas at El Paso

University of Texas Health Science Center at Houston (2)

University of Utah

University of Virginia

University of Wisconsin (2)

Vanderbilt University Medical Center

Washington University, St. Louis (3)

Washington University School of Medicine (2)

Williams College

Yale University (3)

Yale University School of Medicine

2025 BBRF EVENTS

Scientific Council Dinner

Presenting the Klerman & Freedman Awards

July 25, 2025

BBRF's Scientific Council Dinner awarded the annual Klerman & Freedman Prizes and Honorable Mentions to six outstanding BBRF Young Investigator Grantees for their exceptional clinical and basic research. They were selected by committees of the Foundation's Scientific Council.

Geoffrey Simon, BBRF Board Chairman

The researchers' important work is furthering the quest to identify the biological roots of mental illness to enable the development of new diagnostic tools, more effective and targeted treatments, and to pave the way toward prevention.

The evening concluded with an interesting and timely discussion on the state of mental health research between Dr. Jeffrey Borenstein and BBRF Scientific Council Member Dr. Joshua Gordon, the immediate past Director of the National Institute of Mental Health.

Dr. Jeffrey Borenstein and Dr. Joshua Gordon

ANNUAL KLERMAN PRIZE FOR EXCEPTIONAL CLINICAL RESEARCH

Joseph Taylor, M.D., Ph.D.
Mass General Brigham
Harvard Medical School

HONORABLE MENTIONS

Ryan T. Ash, M.D., Ph.D.
Stanford University School of Medicine
University of California, San Francisco

Nathaniel G. Harnett, Ph.D.
McLean Hospital
Harvard Medical School

ANNUAL FREEDMAN PRIZE FOR EXCEPTIONAL BASIC RESEARCH

Long Li, Ph.D.
Institute of Biophysics, Chinese Academy of Sciences

HONORABLE MENTIONS

Hermany Munguba, Ph.D.
University College London

Zachary Pennington, Ph.D.
Icahn School of Medicine at Mount Sinai
University of British Columbia

Scientist Spotlight with: **Joseph J. Taylor, M.D., Ph.D.**

2025 BBRF Klerman Prize for Exceptional Clinical Research
2022 BBRF Young Investigator Grantee

Dr. Taylor has been a core faculty member of the Center for Brain Circuit Therapeutics at Brigham and Women's Hospital and Harvard Medical School since the Center launched in 2020. He is appointed as an Associate Psychiatrist at Brigham and Women's Hospital, and as an Instructor in Psychiatry at Harvard Medical School.

His research focuses on deriving and testing brain stimulation targets for psychiatric illness. He derives targets with network mapping, a method that leverages the human connectome (a wiring diagram of the human brain) to examine the connectivity patterns of brain lesion locations or brain stimulation coordinates that causally modify neuropsychiatric symptoms. Dr. Taylor tests these targets in clinical trials using invasive and non-invasive brain circuit interventions.

WHAT IS THE CURRENT STATE OF YOUR BBRF FUNDED RESEARCH?

To date we have completed all grant elements within the allotted timeframe. We are finalizing our analyses and preparing a manuscript.

WHAT DID YOUR BBRF YI GRANT MEAN TO YOU?

The BBRF Young Investigator Grant allowed me to zero in on a critical scientific question. Without it, I would have been unable to run the study or collect pilot data for my first NIH R01 application.

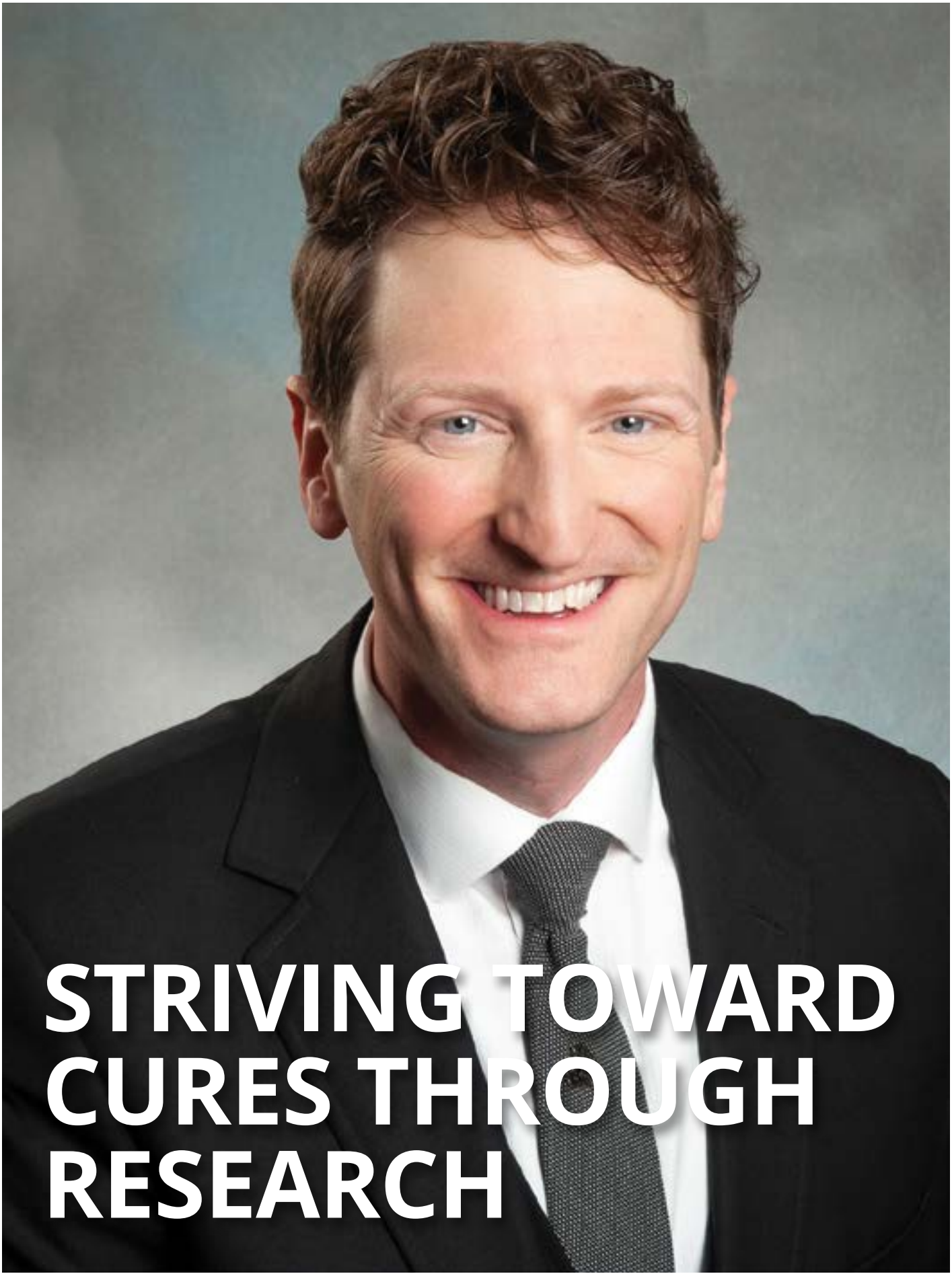
HOW DO YOUR INTERACTIONS WITH PEOPLE LIVING WITH MENTAL ILLNESS AFFECT YOUR RESEARCH AND VICE-VERSA?

Few moments compare to the privilege of helping someone at risk of losing hope.

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

I want my work to help people feel like themselves again.

"The Klerman Prize is a celebration of community—of the family members, friends, colleagues, mentors, mentees, administrators, patients, study participants, and funders who play a role in good science. I am deeply humbled, and I can't wait to pay it forward."



STRIVING TOWARD CURES THROUGH RESEARCH

STRIVING TOWARD CURES THROUGH RESEARCH

Scientist Spotlight with: **Long Lee, Ph.D.**

2025 BBRF Freedman Prize for Exceptional Basic Research
2021 BBRF Young Investigator Grantee

Dr. Li's lab focuses on developing novel animal models for neuropsychiatric disorders, such as depression and PTSD, to uncover new molecular targets and therapeutic avenues. Specifically, the lab investigates neuronal molecular biomarkers modulated by innovative drugs for treating depression and anxiety, as well as seek to develop proactive coping strategies for stress. Their goal is to pinpoint which neurons and molecules can be targeted to alleviate disease symptoms. By elucidating the cellular mechanisms of therapeutic drugs in the brain, the lab aims to enhance drug specificity and minimize adverse effects. It is hoped that this foundational understanding will ultimately guide the design of optimized treatments with improved efficacy and tolerability.

WHAT IS THE CURRENT STATE OF YOUR BBRF FUNDED RESEARCH?

My 2021 BBRF Foundation YI project has been completed. In addition to finding a potential new target for depression treatment and producing several publications, it has also left our lab with many novel scientific ideas and questions to explore.

"As a young investigator, I am incredibly honored to receive the Freedman Prize, and deeply motivated by the Foundation's commitment to supporting basic science that lays the groundwork for better treatments. This recognition reinforces our mission to uncover the neural and molecular underpinnings of depression and PTSD, with the ultimate goal of translating these insights into more precise and effective therapies."

WHAT DID YOUR BBRF YI GRANT MEAN TO YOU?

The BBRF Young Investigator Grant represents far more than funding to me: First, personally, it gives me a lot of confidence in research; second, it's both an honor and a responsibility to translate this opportunity into meaningful advances for mental health.

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

I have seen and talked to many families suffering from mental illnesses. That's exactly why I chose my career as a neuroscientist. Interacting with individuals living with mental illness has profoundly shaped both my scientific trajectory and personal perspective. Their resilience fuels my commitment to research, while their lived experiences anchor my work in real-world urgency—reminding me that behind every data point is a human story. Conversely, I hope our lab's efforts offer more than just future solutions; even small moments of listening and validation can bridge the gap between science and dignity. This reciprocity is why mental health research isn't just a profession, but a shared journey.

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

In the best possible scenario, by uncovering novel therapeutic targets, my research could redefine how we understand and treat depression and PTSD. For patients, this could mean faster, more effective relief with fewer side effects. For families, it could alleviate the heartbreak of watching loved ones struggle without answers, replacing uncertainty with hope. Ultimately, my discoveries might not just treat these disorders but prevent them—transforming mental healthcare from reactive to proactive and giving people back the lives they deserve.

2025 INTERNATIONAL MENTAL HEALTH RESEARCH SYMPOSIUM
OCTOBER 24, 2025

BBRF held its annual International Mental Health Symposium at the Kaufman Music Center in New York City, which was simultaneously live-streamed. Five BBRF Outstanding Achievement Prizewinners gave presentations on topics that included new treatment and remediation approaches for cognitive impairment in schizophrenia; a non-invasive brain stimulation approach to potentially enhance emotion recognition in schizophrenia; genome research shedding light on biological mechanisms in bipolar disorder; the late-onset trajectory and predictability of adult ADHD; and a reflection on the subjective mental qualities of brain and behavior disorders. The symposium also featured a presentation from Nur Yanayirah, Founder of MotherHope Indonesia, the winner of the 2025 Pardes Humanitarian Prize in Mental Health.

Dr. Carol Tamminga, a BBRF Scientific Council member, served as the Symposium moderator.

The symposium is available to watch on the BBRF YouTube channel:
https://bbrfoundation.org/event/international-mental-health-research-symposium

Photo by Chad David Kraus

Joseph LeDoux, Ph.D., Antigona Martinez, Ph.D., Daniel C. Javitt, M.D., Ph.D., Jeffrey Borenstein, M.D., Nur Yanayirah, Ole A. Andreassen M.D., Ph.D., Luis Augusto Paim Rohde, M.D., Ph.D., and Geoffrey Simon, BBRF Board Chairman.

PRESENTATIONS

Listening To Schizophrenia: How Modern Neuroscience Explains the Subjective Experience of Schizophrenia and Points to New Treatment and Remediation Approaches

Daniel C. Javitt, M.D., Ph.D.

Columbia University Medical Center
Nathan S. Kline Institute for Psychiatric Research

BBRF LIEBER PRIZEWINNER FOR OUTSTANDING ACHIEVEMENT IN SCHIZOPHRENIA RESEARCH

Targeting Brain Circuits to Improve Emotion Recognition in Schizophrenia

Antigona Martinez, Ph.D.

Nathan S. Kline Institute for Psychiatric Research
Columbia University

BBRF MALTZ PRIZEWINNER FOR INNOVATIVE AND PROMISING SCHIZOPHRENIA RESEARCH

Genetic Analyses Yield Biological Insights into Bipolar Disorder with Potential Clinical Relevance

Ole A. Andreassen M.D., Ph.D.

University of Oslo and Oslo University Hospital
Centre for Precision Psychiatry

BBRF COLVIN PRIZEWINNER FOR OUTSTANDING ACHIEVEMENT IN MOOD DISORDERS RESEARCH

What Can a Research Center in Brazil Tell Us About ADHD?

Luis Augusto Paim Rohde, M.D., Ph.D.

Hospital de Clínicas de Porto Alegre
Federal University of Rio Grande do Sul, Brazil

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

What Happened to the "Mental" in "Mental Disorders"?

Joseph LeDoux, Ph.D.

New York University
NYU Langone Medical School

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

Empowering Maternal Voices Through Peer Support and Advocacy

Nur Yanayirah

Founder MotherHope Indonesia

PRIZEWINNER, BBRF PARDES HUMANITARIAN PRIZE IN MENTAL HEALTH

The Pardes Humanitarian Prize in Mental Health

The 2025 Pardes Humanitarian Prize in Mental Health was awarded on October 24th to MotherHope Indonesia, a pioneering grassroots foundation dedicated to maternal mental health awareness and care in Indonesia.

Dr. Jeffrey Borenstein, President & CEO of BBRF, and Nur Yanayirah, Founder of MotherHope Indonesia

Tamar and Milton Maltz

The 2025 Honorary Pardes Humanitarian Prize in Mental Health was awarded to Tamar & Milton Maltz, whose visionary philanthropic leadership has advanced groundbreaking mental health research and advocacy.

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.

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

2024
Franca Ma-ih Sulem Yong
Honorary Tribute: Graham Boeckh Foundation

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 24, 2025

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

Daniel C. Javitt, M.D., Ph.D.

Columbia University Medical Center

BBRF Scientific Council Member
1995 BBRF Independent Investigator
1990 BBRF Young Investigator

RUANE PRIZE

Outstanding Achievement in Child & Adolescent Psychiatric Research

Luis Augusto Paim Rohde, M.D., Ph.D.

Hospital de Clínicas de Porto Alegre

MALTZ PRIZE

Innovative & Promising Schizophrenia Research

Antigona Martinez, Ph.D.

Columbia University Medical Center

GOLDMAN-RAKIC PRIZE

Outstanding Achievement in Cognitive Neuroscience

Joseph LeDoux, Ph.D.

New York University

COLVIN PRIZE

Outstanding Achievement in Mood Disorders Research

Ole A. Andreassen M.D., Ph.D.

University of Oslo and Oslo University Hospital

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

Healthy Minds

The Emmy-nominated television series *Healthy Minds with Dr. Jeffrey Borenstein* aims to remove the stigma of mental illness and demonstrate that with help, there is hope. The series features inspiring personal stories from people who have experienced mental health issues, as well as the latest information from experts on new approaches to the diagnosis, treatment, and prevention of mental illness.

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

Season 11 will be available to watch in the Fall at:
https://www.pbs.org/show/healthy-minds-with-dr-jeffrey-borenstein/

SEASON 10 EPISODES:

New Psychiatric Medications (Part One)
Ketamine and S-Ketamine target a different chemical system than many traditional medications for depression with rapid benefits and reduced suicide risk when part of a clinic-based overall treatment plan; a synthetic version of the hormone lost in childbirth may help patients with post-partum depression. *Guest: John Krystal, M.D., Chair, Department of Psychiatry, Yale School of Medicine.*

New Psychiatric Medications (Part Two)
New approaches to psychosis and schizophrenia treat symptoms without the side effects of traditional dopamine blockers; the potential for psychedelics to normalize brain function in depression within a psychotherapy framework; possible psychiatric use of GLP-1 medication to manage side effects and symptoms. *Guest: John Krystal, M.D., Chair, Department of Psychiatry, Yale School of Medicine.*

Alzheimer's Disease: Diagnosis & Treatment
New advances in blood tests for biomarkers mean earlier diagnosis and defined categories of Alzheimer's disease potentially enabling doctors to delay onset of symptoms, with lower costs to patients and higher accuracy. *Guest: Howard Fillit, M.D., Co-Founder and Chief Science Officer, Alzheimer's Drug Discovery Foundation.*

Motivated Behavior: Training Your Brain
The biology of behavior offers tools for a holistic approach to increase good habits and decrease bad habits including how talk therapy, exposure therapy, meditation and spirituality and more can impact the brain's pre-frontal cortex to regulate emotion and decision making. *Guest: Nii Addy, Ph.D., Albert E. Kent Associate Professor of Psychiatry, Yale School of Medicine.*

Borderline Personality Disorder
Borderline Personality Disorder patients now have access to several effective therapies including dialectical behavioral therapy, mentalization based therapy and psychiatric management with goals of self-reliance. *Guest: Lois W. Choi-Kain, M.D., M.Ed., Director, Gunderson Personality Disorders Institute, Associate Professor of Psychiatry, Harvard Medical School.*

Binge Drinking and Alcohol Misuse

How excessive alcohol use—especially begun during adolescence—impacts somatostatin levels in the brain causing anxiety, depression, and future cognitive decline, as well as permanent changes to mood and the brain's pre-frontal cortex. *Guest: Nicole Crowley, Ph.D., Director, Penn State Neuroscience Institute University Park, Associate Professor of Biology and Biomedical Engineering.*

Hoarding Disorder

How excessive acquisition and difficulty discarding possessions leads to hoarding; the genetic and emotional components of the disorder; benefits of cognitive behavior therapy and virtual reality therapy; and how to help family members who don't recognize their problem. *Guest: Carolyn Rodriguez, M.D., Ph.D., Professor of Psychiatry and Behavioral Sciences, Stanford University School of Medicine.*

Eating Disorders and Self-Control

Innovative research uses mathematical models, brain imaging, and behavior assessments to understand and treat the underlying issues of bulimia nervosa and binge eating. *Guest: Laura A. Berner, Ph.D., Associate Professor of Psychiatry, Principal Investigator, Center for Computational Psychiatry, Center of Excellence in Eating and Weight Disorders, Icahn School of Medicine at Mount Sinai.*

Mental Illness and The Criminal Justice System

Exploring the unique issues when someone with a psychiatric or neurodevelopmental condition is brought into the criminal justice system as a result of their symptoms or behaviors, including the best way to proceed in that situation, preventative measures, and the value of an independent forensic mental health evaluation for diagnosis and testimony. *Guest: Criminal attorney Elizabeth Kelley.*

Autism (Part One)

The key factors for diagnostic assessment of autism: developmental history, degree of symptoms, eye contact, and motor behaviors; studies of genetics and infant eye gaze may lead to earlier diagnosis and intervention. *Guest: John N. Constantino, M.D., Liz and Frank Blake Chair of Children's Behavioral and Mental Health, and Chief of Behavioral and Mental Health at Children's Healthcare of Atlanta.*

Autism (Part Two)

The importance of access to early intervention and the right education plan for children with autism; considering the issues unique to the transition from childhood through adolescence and adulthood with autism. *Guest: John N. Constantino, M.D., Liz and Frank Blake Chair of Children's Behavioral & Mental Health, and Chief of Behavioral and Mental Health at Children's Healthcare of Atlanta.*

Growing Brain Cells in the Lab

Research pioneers turn skin cells from patients with psychiatric conditions into stem cells and then brain cells in cultures to learn how disorders arise to potentially develop therapeutics. *Guest: Sergiu P. Pasca, M.D., Professor of Psychiatry and Behavioral Sciences and Bonnie Uytengsu and Family Founding Director, Stanford Brain Organogenesis Program, Stanford University.*

Treatment of Psychosis in Teens and Young Adults

The importance of education about symptoms and early intervention to reduce the duration of untreated psychosis, and the value of programs for young people that use a team approach and peer support for patients and their families; substance-induced psychotic disorder and suicide prevention. *Guest: Robert O. Cotes, M.D., Professor of Psychiatry, Emory University School of Medicine.*

"Over the past decade, Healthy Minds has worked to break down the stigma around mental illness by sharing personal experiences and the latest scientific advances. As we mark our 10th anniversary season, we remain committed to providing viewers with trusted information, promoting understanding, and showing that with help, there is hope."

Dr. Jeffrey Borenstein

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 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 2025:

Fulfilling the Promise of Noninvasive Brain Stimulation as a Precision Medicine Tool
Tuesday, January 14, 2025
Christopher T. Sege, Ph.D.
University of South Carolina

The Crisis of Access to Early Diagnosis of Autism and Emerging Solutions
Tuesday, February 11, 2025
Ami Klin, Ph.D.
Emory University School of Medicine & Emory Center for Translational Social Neuroscience

Emerging Treatments for Social Disconnection in Psychiatric Illness
Tuesday, March 11, 2025
Anya Bershad, M.D., Ph.D.
UCLA-Semel Institute for Neuroscience and Human Behavior

Developing a Potential New Treatment for Chronic PTSD: Ketamine Combined with Written Exposure Therapy
Tuesday, April 8, 2025
Adriana Feder, M.D.
Icahn School of Medicine at Mount Sinai

Is "Stressed" Really "Desserts" Spelled Backwards?: How More Objective Assessment of Eating Behavior Can Refine Our Understanding of Stress Eating
Tuesday, May 13, 2025
Kristin N. Javaras, D.Phil., Ph.D.
McLean Hospital
Harvard Medical School

Does Cannabis Use During Pregnancy Affect Gene Regulation In Placental Tissue?
Tuesday, June 10, 2025
Emma C. Johnson, Ph.D.
Washington University School of Medicine in St. Louis

Transforming Treatment Outcomes for People with OCD
Tuesday, July 8, 2025
Helen Blair Simpson, M.D., Ph.D.
Columbia University / New York State Psychiatric Institute

Improving Psychotherapy for Mood Disorders
Tuesday, August 12, 2025
Marc J. Weintraub, Ph.D.
UCLA Semel Institute for Neuroscience and Human Behavior

Tracking Mood Instability in Bipolar Disorder: Advances in Neuroimaging and Digital Monitoring
Tuesday, September 9, 2025
Danella Hafeman, M.D., Ph.D.
University of Pittsburgh School of Medicine

Investigating the Biological Factors Underlying Suicide Risk
Tuesday, October 8, 2025
Steven Lamontagne, Ph.D.
National Institute of Mental Health (NIMH)

Evaluating the Potential of a Sleep Intervention Among Youth at High-Risk for Borderline Personality Disorder
Tuesday, November 11, 2025
Erin A. Kaufman, Ph.D.
University of Utah

What Can Light Show Us About Brain Activity?: Examining Early Neural and Behavioral Predictors of Childhood ADHD
Tuesday, December 9, 2025
Heather M. Joseph, M.D.
University of Pittsburgh

2025 BBRF EDUCATIONAL WEBINAR

Incorporating Universal Screening and School-Based Mental Health Initiatives in the Classroom

A Q&A with Dr. John Constantino

John N. Constantino, M.D.

Liz and Frank Blake Chair for Children's Behavioral and Mental Health
Chief, Center for Behavioral and Mental Health
Children's Healthcare of Atlanta

Vice Chair, Department of Pediatrics
Professor of Psychiatry & Behavioral Sciences, Pediatrics, and Genetics
Emory University

This special webinar was broadcast via Zoom on November 12, 2025.

Teachers, school counselors, and other educational professionals are on the front line of interacting with children with mental health issues and can often be among some of the first people to see that a child is struggling. Enhancing the potential for early intervention is important and because educational professionals have relationships with students and their families, they are able to make appropriate referrals for evaluation.

Dr. Jeffrey Borenstein, BBRF President & CEO, and Dr. John Constantino discussed a public health approach for teachers and provided educators with a deeper understanding of the importance of incorporating universal screening and school-based mental health initiatives in the classroom.

The program is available on the BBRF website: https://bbrfoundation.org/event/incorporating-universal-screening-and-school-based-mental-health-initiatives-classroom

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THANK YOU TO OUR DONORS

Your support this year enabled us to fund more than $12 million to scientists searching for better treatments, cures, and methods of prevention for mental illness.

Since 1987, in partnership with you, our donors, BBRF has awarded more than $476 million to fund more than 6,800 grants to more than 5,700 leading scientists around the world.

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

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 2025, the Endowment provided $211,209 for the funding of six PTSD research projects.

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

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The Research Partners Program offers donors the opportunity to choose among the best and brightest scientists and the most promising, cutting-edge proposals in mental illness research, as reviewed and selected by BBRF's Scientific Council.

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"Every event brings us that much closer to a day free of mental illness. Thank you for partnering with BBRF." – Jeffrey Borenstein, M.D.

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The Thurkins' wonderful neurodivergent kids
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David W. Park
Grayson M. Parker
Josiah E. Parker III
James (Jim) E. Passe
Logan Pauk
Betty J. Paykel
Dr. Frederick A. Peery
Geraldine (Bender) Peery
Patricia A. Perrino
David J. Persik
Gregory S. Persik
Robert (Robbie) S. Pesta, Jr.
Judith Pestronk
Seymour Pestronk
Parker E. Phillips II
William Brodie Phillips
Reed Pickus
Sybil Pierce
Keith P. Ping
Aaron R. Pixley
Zaccaria (Zac) W. Pogliano
Elizabeth (Betty) M. Poirier
Jonathan (Jon) D. Poplawsky, Ph.D.
Julian F. Porter
Adam C. Potthoff
Rose Pressman
Joshua Prine
Sean P. Pryor
Peppino (Pepé) Puleo
Joel M. Purrington
Dylan Quast
Bert R. Queen
Peter D. Quinn
Marcia Beth Rackley
Arthur J. Radin, C.P.A.
Edna M. Radzikowski
James P. Rafferty, Sr.
Sidharth Ramakrishnan
Delicia Raveenthrarajan
Cassandra (Cassie) N. Ray
Henrietta Raymond
Evan G. Rea
William (Bill) Reader

Memorial Tributes (continued)

John Reason
Steven (Stevie) R. Reid II
Rob and Michelle Reiner
Michael Reschke
Chris Reuter
Eamonn P. Reynolds
William L. Ricketts
Jonathon J. Robbins
Lauren Robinson-Kieves
David A. Rock
Brian Rorick
Thomas Rorick
Ross J. Rosenberg
Jesse S. Rosenthal, M.D.
Sharon Rositer-McGhee
Alan G. Ross
Christina (Chrissy) Rossi
Dr. Andrey A. Roth
Matthew S. Rothman
Sophie Rottenberg
Chris Rudder
Gerald Rudnet
John G. Ryan
Patricia E. Rzasa-Blum
Sam
Carol Sanchez
Lina Sant
Carla Santamaria
Jeffrey Saunders Binz
David L. Schmidlap
Lee R. Schoolmeesters
Jeffrey (JJ) S. Schulman, Jr.
Nora E. Schuster
Urban (Andy) A. Seay III
Ezra L. Seegull
Charles F. Sejud, C.P.A.
Kenneth F. Selig
Anthony W. Semien, Jr.
Timothy B. Sennott
Carrie L. Serene
Barbara A. Serre-Murphy
Andrew Severin
Gary S. Sevitsky
Ratilal C. Shah
Megan L. Sharp
Helen Sher
Bruce Sherman
Harriet Shetler
Sylvia Shick
Shanti Shukla
Ryan B. Sieber
Marcia Simon-Kaplan
Suvicha Sinkantarakorn
Edmund Smith
Edward J. Smith
Jordan J. Smith
Silas W. Smith
William H. Smith II
Donald E. Snyder
Norman Snyder
Joseph (Drew) A. Sobotka
James (Jim) L. Sorensen, Ph.D.
Gena Spaulding
Madeline C. Spina-Borsuk
Jeffrey T. Sramek
Ian J. Stancato
Christian D. Stanford
Frances B. Stanik
John (Jack) A. Stapleton
Gregory L. Starling
Tyler R. Starling
Ruth Stein
Edward G. Steinmetz, Jr.
Margaret (Maggie) M. Still, D.O.
Benjamin H. Stobbe
Kevin T. Stoddard
Jean Lawson Stone
Gene Strobel
Mary J. Strub-Caulkins
Tommy Sunday, Jr.
Richard (Dick) J. Sweeney
Genevieve (Gene) E. Swiderski
Andrew Taddy
Dino B. Taraschi
H. Vonn Taylor
Charles S. Testa
William J. Thompson
Elizabeth Timinsky's brother, Paul
Rosemary Todd
Zachary (Zach) Tolchin
Troy Torres, Jr.
Maria A. Tosti
Leanne Townsend
Arlene Tracy
Victoria (Tori) Trang Cook
Richard M. Travis
Richard Trommer
Paul A. Truman
Donald Trybula
Robert (Bob) T. Tucker
William D. Tucker
Elena Tudosan
Adele C. Tursone
Joseph Tursone
Stephanie L. Van Gundy
Brett A. Van Vort, M.D.
Eugene (Gene) D. Van Vort
Mary (Betsy) E. Vanable
Charles Varkoly
Robert (Bob) L. Veenstra
John M. Vergo
Harriet Vicente
Joaquin B. Villarreal
Gregory Von Burg
Paul Von Burg
Kenneth O. Vorisek
Wm. F. Wagner, Jr.
Dylan J. Wallace
Capt. Anders (Andy) G. Wallin II
Tordis Wallin
Thomas Walsh
Matthew A. Wasserott
Daniel S. Weiss
Thomas J. Wendt
Robert M. Wetzel
Allan Wexler
Michael G. Wieman
Traudl Wildgruber
Nolan R. Williams, M.D.
Lynn Williams-Figg
Douglas Carson Willix
Brian Wilson
Richard K. Wilson
Timothy (Timo) Wilson
Betty J. Winberg
Barbara A. Winkler-Monsanto, M.D.
Arthur O. Wold
Mary Clare Wolf
Kenneth A. Wood
Lindsey Woodward
Peter Wootton
G. Richard Worrell
Keith C. Worrell
Ross Worrell
Surekha Yadawad
Andrew (Andy) Yelenosky
Catherine (Kitty) L. Yelenosky
Neal Zafran
Leah Zanoni
James (Jim) N. Zartman
Thomas T. Zeller
Howard L. Zenker
Linda Zito
Stan Ziubrzynski
Frank R. Zozula

Financial Summary*

Consolidated Statement of Financial Position

		DECEMBER 31, 2025	DECEMBER 31, 2024
ASSETS			
Current Assets			
Cash and cash equivalents		$26,970,756	$38,084,529
Investments, at fair value, current portion		29,432,053	11,991,311
Contributions receivable		—	96,425
Pledges receivable, current portion		4,792	4,895
Prepaid expenses and other assets		205,368	302,557
	Total Current Assets	**56,612,969**	**50,479,717**
Pledges receivable, net, less current portion		9,738	9,738
Assets held in charitable remainder trust		2,332,352	2,300,206
Fixed assets, net		5,207	8,085
Investments, at fair value, less current portion		29,208,249	8,947,449
Right-of-Use Asset		431,511	716,139
	Total Assets	**$88,600,026**	**$62,461,334**
LIABILITIES AND NET ASSETS			
Current Liabilities			
Accounts payable and accrued expenses		$121,815	$52,931
Grants payable		15,474,938	15,014,698
Operating lease liability, current portion		368,116	342,373
Accrued compensation		85,393	71,023
Annuities payable		943,769	970,084
Charitable gift annuities payable		11,889	12,780
	Total Current Liabilities	**17,005,920**	**16,463,889**
Operating lease liability, net, less current portion		160,144	528,260
	Total Liabilities	**17,166,064**	**16,992,149**
Net Assets			
Without donor restrictions		39,287,605	34,274,112
With donor restrictions		32,146,357	11,195,073
	Total Net Assets	**71,433,962**	**45,469,185**
	Total Liabilities and Net Assets	**$88,600,026**	**$62,461,334**

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

Consolidated Statement of Activities

		YEAR ENDED DECEMBER 31, 2025	YEAR ENDED DECEMBER 31, 2024
SUPPORT AND REVENUE			
Contributions		$15,482.874	$15,589,932
Special events, net		173,284	42,878
Contributed non-financial assets		1,999,827	2,054,955
Bequests		23,973,659	3,472,060
Net realized and unrealized gains on investments		2,201,522	3,698,514
Net appreciation of assets held in charitable remainder trust		32,146	414,370
Dividend and interest income		1,793,960	1,462,573
	Total Support and Revenue	**45,657,272**	**26,735,282**
EXPENSES			
Program Services			
Research grants and awards		11,620,567	10,772,382
Scientific advancement		2,319,523	2,397,371
Program support		2,456,798	2,513,497
	Total Program Services	**16,396,888**	**15,633,250**
Supporting Services			
Fundraising**		992,665	989,718
Administration**		2,302,942	1,810,315
	Total Supporting Services	**3,295,607**	**2,800,033**
	Total Expenses	**19,692,495**	**18,433,283**
Change in Net Assets		25,964,777	8,301,999
Net Assets, beginning of year		45,469,185	37,167,186
	Net Assets, end of year	**$71,433,962**	**$45,469,185**

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

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

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