MAGAZINE

Brain&Behavior

NOVEMBER 2017

Pathways to the Future

SPECIAL EDITION

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Brain & Behavior Research Foundation

This special edition of the *Brain & Behavior Magazine* celebrates 30 years of funding cutting edge science. Since 1987, more than 60,000 people have joined us in our mission to fund neuropsychiatric research in order to improve the lives of people living with mental illness. We have provided $380 million in research grants to more than 4,500 scientists globally. We are the largest non-governmental funder of mental illness research grants in the world. Our initial "seed money" results in subsequent federal and private funding for our Grantees with a return of anywhere between 10 and 19 times the original amount invested by the Foundation. By conservative estimates, this multiplier effect has resulted in more than $3.8 billion of additional support for those scientists.

While there is universal acknowledgement of how far brain and behavior research has come since the Foundation's inception and how much progress has been made, we still have so much to learn about the brain's workings. We are excited about recent technological advances that have made possible experiments that would have seemed like science fiction 30 years ago.

As we celebrate our anniversary, we are excited to share the thoughts and insights of 81 of our Outstanding Achievement Prizewinners over the past 30 years. Their comments provide a rare glimpse into the future-visionary statements of men and women who are leaders in their respective specialties. We are also sharing the comments from five of our Scietific Council Members, who are luminaries in the field, from our July Klerman & Freedman Awards dinner. Their assessments of where we are and where we are headed are comprehensive in scope. At the end of this issue, you will see a listing of grantees from 1987 through today.

The Foundation is proud of the accomplishments of the scientists we fund and we are excited to focus on their promising paths of discovery. And we will keep propelling forward. Our Scientific Council will continue to identify the most creative, promising research proposals for funding, helping to accelerate the pace of research and will focus on new approaches with the greatest potential for breakthroughs.

The significant impact of the Foundation is only possible through the collaboration between scientists and our generous donors, who understand that investing in brain and behavior research will continue to bring better treatment and ultimately cures and methods of prevention. We are deeply appreciative of this collaboration. With your sustained commitment, we will increase funding of our grants and continue to lead the field with breakthroughs that improve the lives of those living with mental illness. Thank you for your support.

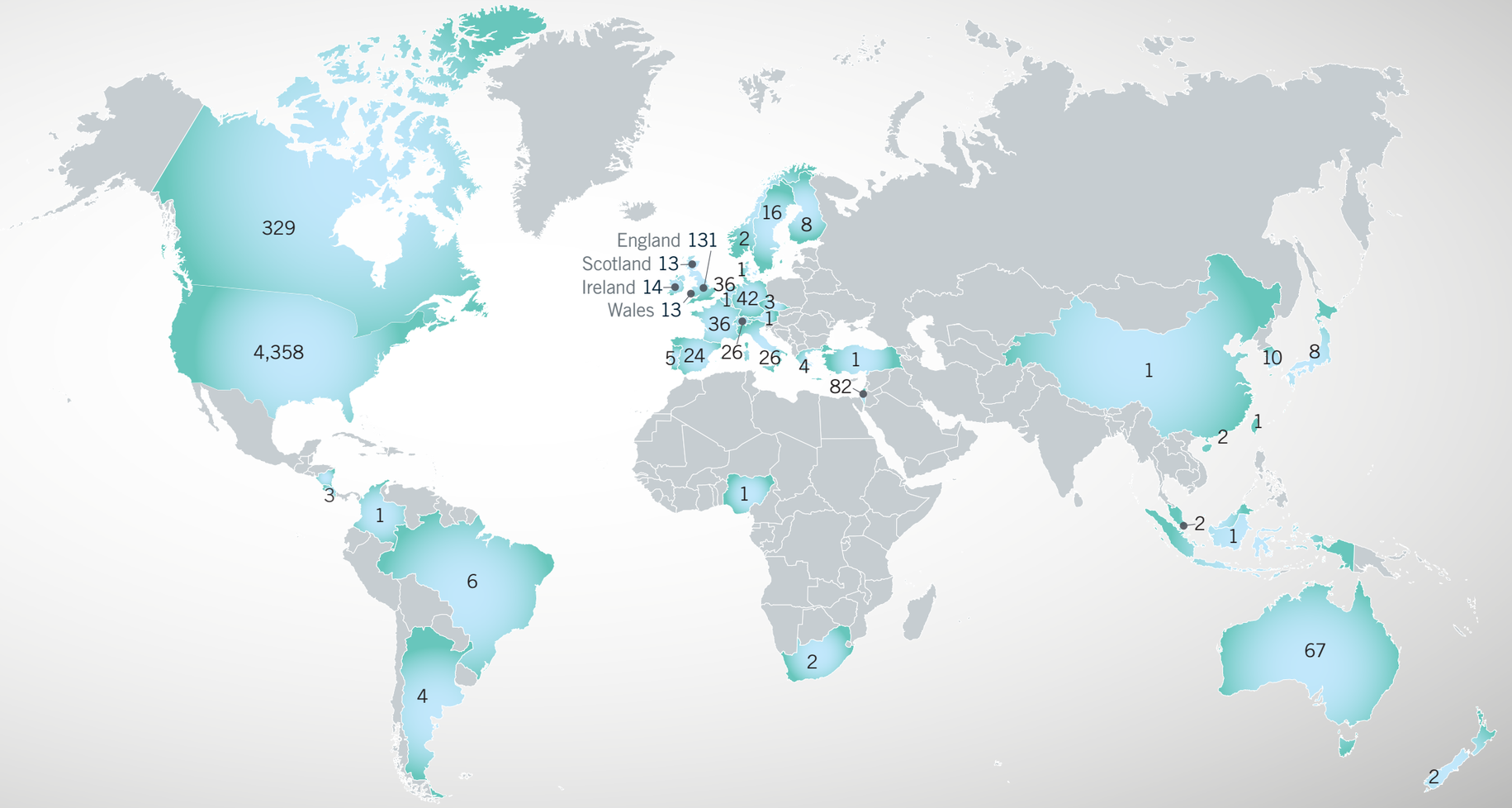
Sincerely,

Jeffrey Borenstein
President & CEO

"The Brain & Behavior Research Foundation has had a profound impact on the scientific content and work in the field. In other words, one can say that in the generation of the positive advances, new ideas, tools, concepts, the Foundation has been a substantial complement to NIMH and other NIH institutions fostering new thinking and research on psychiatric disorders. This is happening not just throughout this nation. This is worldwide."

Herbert Pardes, M.D.
President of the Foundation Scientific Council
Executive Vice Chairman of the Board of Trustees,
NewYork-Presbyterian Hospital

Our Global Footprint



TOTAL AMOUNT AWARDED SINCE 1987

$380M

TOTAL NUMBER OF COUNTRIES

35

TOTAL GRANTS AWARDED

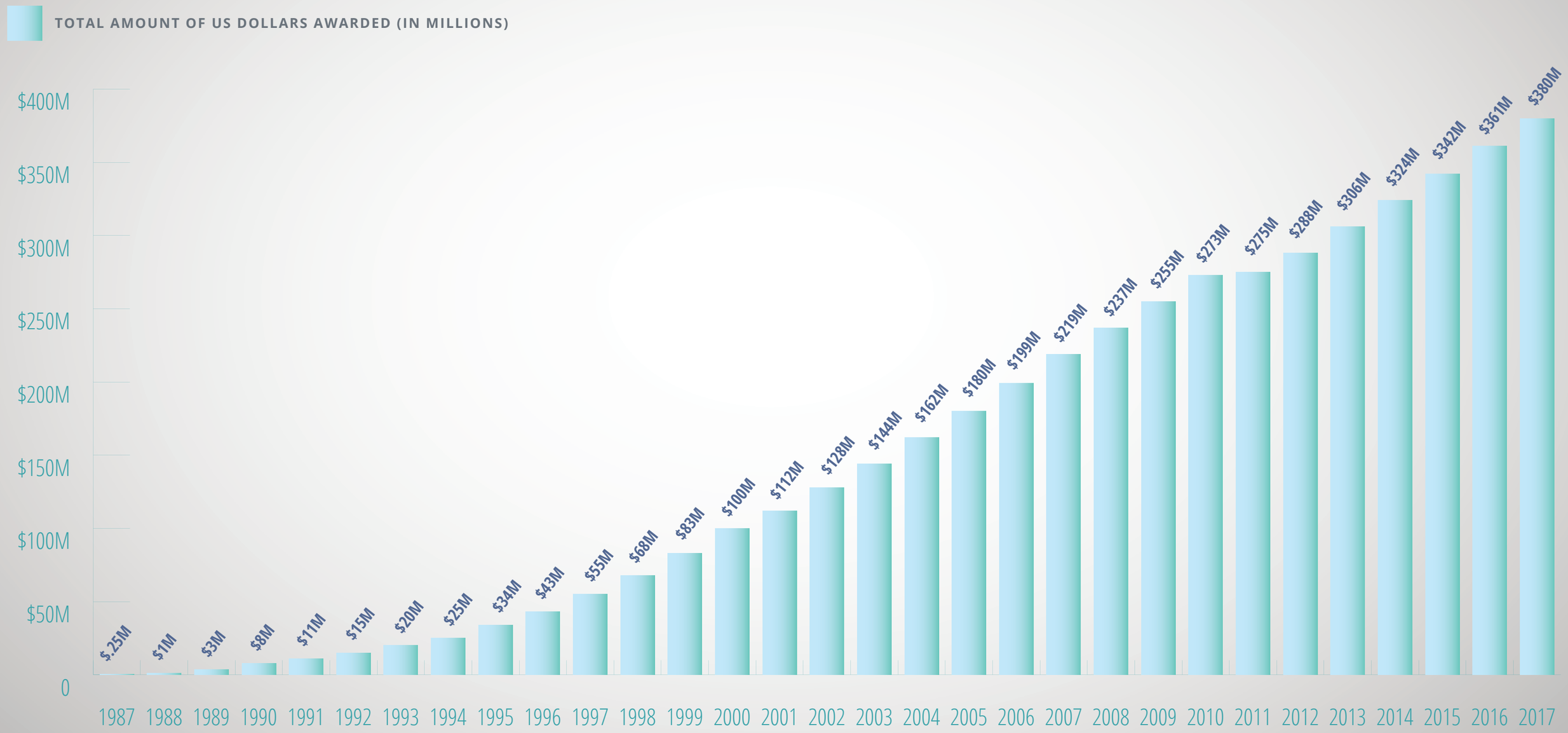
5,500+ In Total

4,282 Young Investigator Grants
828 Independent Investigator Grants
409 Distinguished Investigator Grants

GLOBAL INSTITUTIONS

U.S. 332 | **FOREIGN 218** | **TOTAL 550**

Total Amount Awarded Since 1987



Pathways to the Future

Insights from Five Foundation Scientific Council Members and Luminaries in the Field of Psychiatry

A Note From Dr. Herbert Pardes, Executive Vice Chairman of the Board, NewYork-Presbyterian Hospital and President of The Foundation's Scientific Council

On July 28, 2017, the Foundation hosted its annual Klerman & Freedman Awards dinner honoring the service of the Scientific Council and celebrating 30 years of grant making. The event represented a wonderful celebration of the Brain & Behavior Research Foundation and everyone involved with us.

On the following pages you will see the speeches given at the Klerman & Freedman Awards Dinner by five Foundation Scientific Council Members, all who are luminaries in the field of psychiatry. Their comments address where we have started and where we are heading as we move toward the future.

From my remarks that evening:

> *Prior to this event, Steve Lieber [Chairman of the Board] spoke to me regarding the series of statements made by 81 leading researchers regarding the state of research on psychiatric illness. He asked if I would look at the statements made by these leaders, integrate them and generate a discussion. I enjoyed reading the statements and was greatly impressed. Aside from the content, what impressed me was the optimism and enthusiasm expressed regarding where we are in psychiatric research.*

Daniel Weinberger, M.D.
Director and CEO
Lieber Institute for Brain Development

1993 Lieber Prizewinner
2000, 1990 Distinguished Investigator Grantee

It's been 25 years since I was honored with the Lieber Prize [for Schizophrenia Research], and I've given some thought to where this field was then and where it is now. I think in 1992 we were at an inflection point, one at which we were able to show that the brain was involved in schizophrenia and that with functional imaging techniques we could identify systems in the brain that were associated with the kinds of behaviors that we were studying. It seems remarkable to us today, but prior to that time there was still uncertainty as to whether the brain was in fact going to be important for schizophrenia. Today it's a given.

What's changed is we've gone from being able to look in the brain and understand how the brain might be involved in psychiatric disorders and schizophrenia in particular to being able to map circuits that mediate some of the features that we think are important in how people behave when they have these conditions. Importantly, now we have [predisposing] genes. That is what enables me to say that we've learned more about the causes and mechanisms of schizophrenia in the last 25 years than we knew in all of previous history. Genes represent mechanisms of disease, and that's something we've never had before in research on schizophrenia and other psychiatric disorders. We've had a very rich phenomenology of understanding how these illnesses look, how they behave, and to some degree how they feel, but we really didn't know what they were. I'm reminded it was 24 years ago that I attended a meeting at the National Academy of Sciences and Harold Varmus had just become NIH director. In his inimitable way, Varmus challenged all of us by saying that "If you're not studying genes, you're going to be dinosaurs in an age of mammals."

A lot of genetic discoveries have emerged in the last few years that change the schizophrenia landscape profoundly, just as they've changed the landscape in every common medical disorder from heart disease to diabetes to stroke to cancer. These are tools to understand mechanism of illness, and they're strategies to trying to identify new ways to think about disease, to diagnose it, and ultimately to treat it more effectively. We've learned that there are no genes "for" mental illness, just like there are no genes "for" heart disease or stroke. What we have are genes that increase the biological risk that those things will happen.

A gene for high cholesterol is a risk factor for stroke and heart disease, but it doesn't guarantee heart disease. Genes that are associated with risk for mental illness that change a person's likelihood of manifesting them do not mean that anyone who has them is fated to become ill. We've learned is that there's no master gene or master way to get to schizophrenia. There are many, many roads that can take someone on this trajectory. There are probably thousands of them. In maybe one to two percent of the people that we diagnose with schizophrenia, there are genes that have a strong impact on the probability that they will be ill. Those genes are very important to study because they enlighten to us to what mechanisms at the very basic level of brain cells are implicated in the illnesses. But these are rare. They don't explain most cases.

Most cases are explained by common gene variations that accumulate either in particular combinations or with particular burden in people who are ill. There are probably thousands and thousands of these variations, and this challenges us to think about how we're going to translate genes into a story to understand mechanism of illness.

There's some skepticism that there are too many genes; that it's unmanageable. How do you know which genes matter and which don't? When you look at all these genes, it becomes very clear that the human genome did not evolve to validate our arbitrary clinical diagnostic criteria. These genes are about the development and function of a brain. They're not necessarily about any one diagnosis that we give people. [But what do these genes tell us about schizophrenia? Are the clues they provide related at all to our prior knowledge of schizophrenia?] Albert Einstein once said that nature is subtle but not malicious. It would be malicious if these genes had nothing to do with what's been studied in schizophrenia for 20 or 30 years. It turns out that the dopamine system is implicated in many of the genes. The glutamate system which has been a subject of considerable interest has been implicated by a number of genes. GABA [an inhibitory neurotransmitter] has been implicated by a number of genes. We're learning that the clues we knew of before we had the ability to understand how genetics translates into mechanisms of risk and illness were not without value.

I want to make the point that by having someone's genetic profile, we have the possibly of predicting that person's risk liability. This allows us to think that in the future we might be able to prevent the emergence of illness in people at particularly high risk. Prevention is the 'holy grail' in clinical medicine. Treatment and cure are very desirable, but they come after the fact of illness. Today we don't really have good strategies for prevention, but I think we are going to be able to develop these as time goes on.

I want to make another point about what our knowledge of genetics allows us to do. Since genes are inherited, and most of the genes that influence risk for schizophrenia are there from birth, it raises the question of their role at the dawn of life. One of the other things we've learned over the last 30 years about schizophrenia is that there are clearly developmental components to its origins.

At the Lieber Institute for Brain Development we've recently discovered, however, that some of the genes that are related to risk for schizophrenia are not directly about the developing fetal brain. They're actually about the health and development of the placenta, which is an interesting discovery because it suggests that there may be ways to improve prenatal health by targeting the placenta. That's a potentially much easier way to affect the health of the developing fetus. This suggests there may be the opportunity of identifying particularly high-risk individuals who could potentially be the target of interventions for prevention.

I wanted to end on what I think is the biggest change in the 25 years since my Lieber Prize that I think will ultimately have the biggest impact on schizophrenia research. That is that there is a new generation of people, of researchers focused on basic science, interested in studying mental illness. Twenty-five years ago many such people were going into cancer, into Alzheimer's disease, into neurodegenerative disorders where the data about cause and about molecular mechanism was so much stronger. Psychiatry, schizophrenia research, was seen as sort of a backwater in neuroscience. It's not like that anymore. It's right in the mainstream of neuroscience. The opportunities to make significant discoveries and advance the search for prevention strategies and cures is literally light years ahead of where it was 25 years ago. We have now a generation of the brightest and the best going into research on mental illness. As I say to young scientists, this is an incredible time to be in this field.

William E. Bunney, M.D.
Distinguished Professor,
Psychiatry & Human Behavior
University of California,
Irvine School of Medicine

2001 Falcone Prizewinner
1997 Distinguished Investigator Grantee

I will focus my comments on major depressive disorder. Future research is involved in identifying risk genes. Fifteen have been identified, but they only account for a small percentage of the risk. Another critical component of future research is studying the environmental factors associated with vulnerability for depression and the onset of the depressive illness. A number of studies have shown that an abusive childhood can be associated with depression and suicide. A relevant recent paper published in the Journal of Science by Eric Nestler and his research team showed that early-life stress resulted in lifelong stress susceptibility to the later onset of stress-precipitated depression-like behavior in mice.

I now will make a few brief comments on the prevalence of major depressive disorder, new treatments, the role of 24-hour rhythms in major depressive disorder and how all three of them may lead to a completely new treatment strategy, the use of biomarkers to identify individuals at high risk for suicide, and the use of revolutionary technology to study depression.

The prevalence of major depressive disorder in the U.S. is somewhere around seven percent, while schizophrenia is about one percent and manic depressive illness is two percent. The most recent data from the World Health Organization ranks depression number one of all medical diseases in terms of lifetime disability. Conventional antidepressant medications require two to eight weeks for clinical efficacy.

Within the last decade low-dose ketamine [an anesthetic that is now being experimented with in depressed people] has been shown to dramatically work within 24 hours in treatment-resistant depressed patients who didn't respond to usual medication. Low-dose ketamine is being administered in many countries and actually in specific ketamine depression treatment clinics in the U.S. However, there are two problems with ketamine. One concerns side effects; the other is the short duration of its effectiveness–about a week. These need to be addressed in future research.

New ketamine-like compounds and those which modulate the excitatory neurotransmitter glutamate are under active development. Ketamine has been given intravenously and new studies involve intranasal ketamine. A study of intranasal S-ketamine [a ketamine variant] is nearing completion. There have been a large number studies on the rapid mode of action of ketamine including Ron Duman's work at Yale, Carlos Zarate's NIMH research, and many other important studies.

It is now proposed that genes called "clock" genes may be critical in depression. A subgroup of depressed patients were found to have abnormal 24-hour rhythms of temperature, hormonal secretion, sleep and mood, which are all controlled by clock genes.

A study I've been part of has showed normal clock gene rhythms and controls and abnormal rhythms in depressed patients. Now we come back to ketamine. My wife and I recently published two papers, one in the last month in Biological Psychiatry, showing that ketamine significantly affected eight of the core clock genes. We hypothesized that ketamine may act in part by resetting abnormal clock genes associated with depression. Our future research will study this possibility.

Next I want to address the particularly serious issue of suicide of which an estimated 90 percent of the cases are associated with depression. The World Health Organization estimates that there are 800,000 suicides each year worldwide, one every 40 seconds. In the U.S., the incidence is 42,000 per year. There have been more U.S. deaths by suicide than by homicide every year over the last 50 years.

A significant number of patients who die by suicide see a health care professional within a month of suicide. Therefore, there is an opportunity to identify individuals at high risk for suicide. One could then provide specific and additional treatments. A future research challenge is to continue to find reliable biomarkers in blood that can predict suicide risk. A potential clinical marker for suicide is psychological pain. A colleague of ours studied 200 suicide notes and reported that one of the most frequent comments in the notes was, "I can't stand the mental, not physical pain, the mental pain any longer. I want to die to end the pain."

We developed a scale to measure psychological pain and identified a cutoff score of 31 on the scale, above which patients had an increased risk for suicide. We recently published a study in which we administered the psychological pain scale to VA patients and followed them for seven months. None of the patients scoring below 31 on our scale made suicide attempts. Eight patients scoring above 31 made serious suicide attempts and if they hadn't been found, they would have died; and one did die by suicide. Future research can study molecular biomarkers in blood and clinical markers that could predict high risk for suicide and potentially save lives.

Finally, I want to comment on additional future research opportunities and the use of revolutionary new technology to study depression. We know that there's a genetic component contributing to major depressive disorder. New technology named CRISPR-Cas9 and now a more accurate CRISPR-Cas13a, allows rapid, inexpensive and accurate gene editing. This could be used to alter risk genes identified by genome-wide association studies and sequencing of the human genome.

In the future, there will be an increased focus on personalized medicine to treat diseases including depression. This is in part involves the sequencing of an individual's genome, which is becoming significantly less expensive. Long-read sequencing [a new technique] combined with a technique called optical mapping is a truly new advance, providing more accurate sequencing of the human genome. There are just a few of the technologies that will be critical to further our understanding of depression, develop new treatment strategies, and possibly, prevent depressive illness.

Helen Mayberg, M.D.
Professor of Psychiatry, Neurology, and Radiology
Dorothy C. Fuqua Chair in Psychiatric Neuroimaging and Therapeutics
Emory University School of Medicine

2007 Falcone Prizewinner for Outstanding Achievement in Affective Disorders Research (Colvin Prize)
2002 Distinguished Investigator Grantee
1995 Independent Investigator Grantee
1991 Young Investigator Grantee

In 1987 I was a neurologist in a radiology department and started studying depression and neurologic disease. I started working in the early days of receptor imaging. At Columbia, we couldn't even get a CT scan in the middle of the night. So, when I think about 30 years in the evolution of neuroscience, I think we all need to realize that we're in a golden age.

We can do optogenetics because [Scientific Council member and former grantee] Karl Deisseroth helped us to see the way, and we now have the brain and circuits and timing in ways that I don't think I could have imagined when I began my career. We should keep this in mind when we get impatient on where we are. And we should celebrate that the people in this room have enabled great strides forward with their donations, because they believe it's an important cause.

You [donors] have suffered because of someone close to you. Our progress hasn't been fast enough. We do the best we can with the tools we have. Imaging has allowed us to find the important areas of the brain and then attack them with every new tool that we've got. We have a lot of treatments for mood disorders. They [often] work. The problem is we give you something and it doesn't work, and so then we try something else, and if you're lucky you have insurance and maybe it doesn't run out. As the work has evolved, we have [been able to recognize] classes of depressions. Sure, we have genes and we have all the other things that we'll eventually figure out in detail, but right now, an individual patient can get a brain scan, and we can register their brain type and actually know not only if they will get better with a given drug or therapy, but we'll know what treatments are not likely to work.

That changes the game right now. It means we can reverse-engineer what's important about those circuits. This is changing how we think about things right now with the tools we have right now. We have to really exploit them in major ways. We have a circuit and we know what needs to change, and we can stick an electrode in and we can change the circuit. We can effect change, and the only way we are able to do it is because of all the research that has gone on over a period of decades.

We are benefiting from a revolution of tools. We have the Brain Initiative; President Obama brilliantly imagined that we build tools to understand the brain better. We will ask, not just how do you look at a circuit in real time; we'll ask, how do you look at all of the circuit, all of the brain. Companies and scientists are building devices to let us do it safely in people.

It's seven years since I won the Falcone Prize. [Using a method called deep brain stimulation,] we placed an electrode in very sick, depressed peoples' brains, and they did better. People got excited and people replicated it, and we've replicated it. We have people 10 years out of that treatment who are well, people who were in the hospital, who were suicidal. They're well with an electrode in their brain because we've changed the rhythm and we hold it there. We need to figure out what [precisely] we did, because that's fundamentally going to tell us what depression is.

On the other hand, if you move too fast to get it to everybody, everybody gets impatient and companies fail. We've got to be patient just like we have to be patient with research on genes. We have to be patient with a failed drug and realize that the cause [of the failure] is important, the road is hard, and that the science is there and that with systematic study we can learn and succeed.

We can do better. Our thinking about what depression is fundamentally about is changing, and I think that imaging, the new tools, the new animal models, the integration of those with clinical practice–we'll move back and forth between our animal models and patients–this is the future, it is where we are headed. When we get a lead in humans, we can scale it back to the animals and we get their help–then return with what we've learned to our patients. This is the future.

Joseph T. Coyle, M.D.
Eben S. Draper Chair of Psychiatry and Neuroscience
Harvard Medical School

2004 Lieber Prizewinner
2004, 1995 Distinguished Investigator Grantee

At a couple of points in my career I really had to move into new areas, and at NIH or NIMH, when you move into a new area, you're not going to get funded unless you can demonstrate you've already done the work. That's where NARSAD came in two times in my career.

As chairman of the Department of Psychiatry at Harvard Medical School, I would say NARSAD has been the mother's milk for junior faculty members who wanted to do research in psychiatry, and it was these funds that have allowed them to demonstrate that they could do the research and write [larger] grant proposals [e.g., for the NIH] that would be competitive. Donors, I don't know if you appreciate how much you have meant to neuroscience and to young neuroscientists.

I have been asked to discuss recent advances in child psychiatry, and I can say that until recently, talking about child psychiatric research was an oxymoron. When I became the head of Child Psychiatry at Johns Hopkins, my goal was to introduce neuroscience into child psychiatry. Yale had a neuroscience research program in child psychiatry, as did Stanford. That was it. You'd go to a child psychiatry meeting and say something about the brain, and people would look at you, like what are you talking about?

That's not the case now, and I think NARSAD has played an important role. But from my perspective the distinction between child and adult psychiatry is somewhat spurious, as it focuses on the time of symptom emergence and not on the time that the pathology really starts attacking the brain. Recent genetic and molecular studies further undermine the distinction and suggest that the most serious psychiatric disorders have developmental antecedents.

Let me touch upon recent advances in our understanding of autism, the prototypic child psychiatric disorder since the symptomatic onset is in the second year of life. Autism was originally described 74 years ago, and at that time it was thought to be fairly rare. It was also thought to be caused by what was known as refrigerator mothers. For that reason, many families until recently who had children with autism were repelled by psychiatry because they felt like it stigmatized them.

Epidemiologic studies in the last two decades demonstrate that autism is not rare but rather affects about one percent of the population and predominantly males. Thirty years ago, pioneering twin studies on autism demonstrated that if one identical twin had autism, the chance that the other would have it is about 90 percent, indicating a high degree of heritability.

Now let's scroll forward 30 years. Just a couple of months ago, a genome-wide association study (GWAS) which involved 16,000 subjects with autism and 140,000 controls was published. I should point out a study of this size could not be carried out by any one clinic, hospital or center; rather it required the collaboration of many research groups from across the world to accumulate this number of genetically characterized genomes, and I want to emphasize this because it speaks to the growing spirit of cooperation that is necessary to accumulate the large number of cases required to achieve statistical significance.

Several genes conferring risk for autism were identified. They encode proteins located in the synapses [i.e., the gaps connecting] glutamate neurons. Gutamate is the main excitatory neurotransmitter in the brain, and it involves about 70 percent of the synapses in the cortex. More importantly, it is the neurotransmitter that drives synaptic plasticity and the developmental processes associated with it.

In another study, Glessner and their colleagues looked for mutations that actually changed the structure of the protein, and they found five patients with mutations of the AMPA receptor. That's a glutamate receptor, and it's one of the glutamate receptors that is responsible for plasticity, which is key for learning and memory.

The pathology of the autism involves glutamate synapses, but in

around 40 percent of autism, the problem is not too few synapses like we see in schizophrenia but rather too many. During fetal development actually there is an excess number of synapses that form, and then after birth those superfluous and ineffective synapses are eliminated.

We call this pruning. It's kind of like going around your garden and getting rid of the parts of the plant you don't want, and we think that this pruning increases fidelity of communication and reduces noise. If you have too many synapses firing in your brain, you can see why a child with autism would just be overwhelmed by the stimuli in their environment. This pruning process normally occurs after birth.

Joe Piven at the University of North Carolina was one of the first to demonstrate increased cortical volume with magnetic resonance imaging in about 40 percent of individuals with autism. Joe and his colleagues recently published a very large prospective study involving over 100 infants, newborns who were at high risk for autism because they had a sibling with autism. That increases their risk about 20 times what it would be in the population, and they were imaged repeatedly after birth.

What they found is that there's a hyper-expansion of the cortical surface between six and 12 months of age after birth, and this results in overgrowth of the cortex between 12 and 24 months of age in infants that ultimately went on and developed autism. The time during which these structural changes were taking place was when the autistic symptoms appeared in these infants. They also did a functional imaging study and were able to discern at six months children who were going to go on and development autism. Early detection is the holy grail in autism as it should be for all mental disorders because it's very clear now that the earlier the cognitive behavioral intervention is introduced, the better the outcome. So, the clock has turned back from say maybe six, seven years old to two years old, and now potentially to six months old.

I just want to go to the other extreme of development, and that is early neglect and abuse early in development. Childhood neglect and abuse is one of the most robust predictors of psychopathology in adulthood, and it increases the risk for anxiety disorders. It increases the risk for mood disorder, depression, and also increases the risk for schizophrenia. Many people don't know this–the importance of risk genes you're carrying when you bump into this environmental adversity.

Eric Nestler's group looked at a mouse model of depression. In order to recreate human abuse and neglect in childhood they provided the mother mice with an insufficient amount of nesting materials so they couldn't make good nests. Then they took the babies away periodically and kept them away from their mother for a couple hours. This was from the age of 10 to 20 days after birth–roughly the human equivalent of from 2 to about 12 years old. And they showed that when they did this, these mice were much more liable to develop severe depression when they were stressed as adults, much more than mice that had not had that experience.

But more importantly, they drilled down and they found a transcription factor [a regulator of gene expression] in the ventral medial tegmental area of the brain, in the brain's reward circuit. It looks like they found the proximal pathway leading to a persistent vulnerability to depression as a consequence of stress later in life, and this provides us targets, targets to potentially intervene for those who are at high risk, and I think as we pursue this, it'll be clear that there are risk genes that modify this--increase the risk or decrease the risk.

These vignettes indicate that research in the developmental aspects of mental disorders is flourishing. In part, this reflects the powerful influence of molecular approaches. You can sequence a genome for $1,000. Twenty years ago, it was $200 million. The cooperation among genetic psychiatric labs across the world has transformed our ability to identify these risk genes. Notably, many of the risk genes identified in schizophrenia, bipolar disorder and attention deficit hyperactivity disorder are genes that affect brain development.

The final thing I'd like to say is that the future really depends on the young people, and I think the increasing rapidity of advances we're seeing has to do with young people coming in with new ways of thinking about things and new senses of cooperation–instead of the older way of "this is my work and it's not your work." I think NARSAD can get us to the next level in developing these new ways of thinking about things.

Myrna Weissman, Ph.D.
Diane Goldman Kemper Family
Professor of Epidemiology in Psychiatry
Columbia University College of Physicians & Surgeons

1994 Selo Prizewinner (Mood Disorders Research)
2005, 2000, 1991 Distinguished Investigator Grantee

Psychotherapy as you know it, through Woody Allen or maybe your own past experience, is no longer the norm. Since this is an event in honor of [my late former husband] Gerald Klerman, let me indulge myself to tell you about psychotherapy. It's my assignment but it's also to tell you about Gerry. Forty years ago Gerry, a psychopharmacologist, had just finished a multi-centered study in schizophrenia at NIMH.

He worked in parallel with Dr. Aaron Beck. They were friends but they were quite independent, and they started a revolution. Psychopharmacologic agents were the new thing. They were being tested. Methods for assessing the efficacy–ratings scales–had been developed. The FDA had guidelines, but there was only one problem. The public likes psychotherapy, and for that there was not one clinical trial when they started.

Against this background, Gerry developed interpersonal therapy (IPT). It was based on the fact that depression developed in the context of life stress. He realized that there were genetic causes. It certainly ran in families but that the triggers were life stress such as grief, transitions, disputes, or loneliness. Beck, working with neuropsychologists, developed cognitive behavioral therapy (CBT), which was based on the premise that depressed patients had faulty thinking, and if you could change the thinking, you could reduce the depression.

Independently but again as friends they developed manuals, training programs, fidelity measures and clinical trials, and they also extended these trials beyond depression to anxiety to eating disorders and to a number of other disorders. Today there are over 300 trials of IPT and CBT, and there are at least 10 other evidence-based psychotherapies available.

Let me give you a glimpse of IPT. I was there as Gerry's assistant and continue this work as my hobby but also something I'm very interested in. What I'm going to say is true for CBT as well. First, psychotherapy is not a cult. We need many different types of therapy. It's not one type fits all, but there must be evidence for its efficacy. We would not want to have just Prozac for depression or Crestor for high cholesterol. The same is true for psychotherapy. One treatment may not work for one patient, and we may not know why, but it's pretty good if we could switch to another.

So, what's going on today in IPT? Let me just tell you briefly what some of the advances are. There are studies on depressed pregnant women to improve child nutrition. The idea is if you can reduce the depression and also not use medications during pregnancy then you might help the child eat better and thrive better as an infant. This is going on right now. There are primary-care brief versions of IPT for distress in order to have early intervention, because most depressions are first treated in primary care or at least those suffering first come to primary care doctors.

There are adaptations of IPT for adolescents, for college students, for the elderly, for PTSD. IPT has been translated into nine languages and CBT probably into more. There is an international society–people from 30 countries belong–and there's Internet training. Grand Challenges of Canada recently gave a million dollars for training and implementing IPT for Syrian refugees in Lebanon. Haiti after its great earthquake some years back also used IPT, and I wonder whether we might have offered mental health first aid as this IPT, CBT or something like that after Katrina. Maybe we would not have had such an aftermath.

Our group has finished two clinical trials successfully in Uganda, and it was so successful that it has been taken up by a diplomat working with economists and providing it to people in the major cities in Uganda because they found it was cost-effective. Psychotherapy is not something offered instead of medication. It should be seen as just another tool. My current work with the WHO on a manual for depressed patients coming to primary care in Muslim countries was quite an eye-opener because it showed that the problems were similar across cultures. It was quite easy to adapt this New Haven-born treatment to Pakistan and other countries. We need more of this first aid, and I think the press has not fully appreciated the revolution, and I think the people have not fully appreciated the revolution. Forty years ago, Gerry wrote a paper, *Should there be an FDA for psychotherapy?* The question is still unanswered, and the answer is still yes.

Let me end by just giving a glimpse of what NARSAD has been in my life and the life of the young investigators. My work right now is primarily studying biomarkers in the treatment of depression and also in looking across generations of depressed people in families to see what might be the mechanisms. Right now, two young investigators [in my lab] are being funded to look at structural and functional mechanisms.

NARSAD has also funded a study to bring mental health pilot work to black churches because there is such a reluctance to go to mental health professionals among African Americans. This resulted in a K Award, and is now being introduced in the Zuckerman Center at Columbia University as a community service. NARSAD is a scaffold. It fills gaps and it invests in the new talent, the new ideas and the next innovation, and I personally am grateful for all of the support that I have received and that others have.

The Past, Present, and Future

Visionary Statements on Brain Research by Foundation Outstanding Achievement Prizewinners

Outstanding Achievement Prizes

These top prizes in psychiatric and neuroscience research recognize leading scientists for their innovation and productivity, and for achieving breakthrough discoveries that are bringing us closer to our goal of conquering mental illness.

These prizes not only recognize and award extraordinary leadership in key fields of psychiatric research; they provide models of accomplishment for younger scientists involved in brain and behavior studies.

The Outstanding Achievement Prizewinners are dedicated teachers and scientists who represent models of accomplishment for younger scientists in brain and behavior research.

Lieber Prize for Schizophrenia Research

Established in 1987 by Constance and Stephen Lieber to bring public recognition to the outstanding discoveries being made in schizophrenia research.

Colvin Prize for Mood Disorders Research

Established in 1993, this prize was formerly known under the successive titles of the Selo Prize, Falcone Prize, and Bipolar Mood Disorders Prize. The prize was renamed in 2012 in honor of the late Oliver D. Colvin, Jr., a great benefactor of the Foundation who left the largest single contribution in the Foundation's history.

Ruane Prize for Childhood & Adolescent Psychiatric Research

This prize was initiated in 2000 by philanthropists Joy and William Ruane to recognize important advances in understanding and treatment of early-onset brain and behavior disorders.

Goldman-Rakic Prize for Cognitive Neuroscience

This prize was created by Constance and Stephen Lieber in memory of Patricia Goldman-Rakic, Ph.D., a distinguished neuroscientist renowned for discoveries about the brain's frontal lobe, after her tragic death in an automobile accident in 2003.

Maltz Prize for Innovative & Promising Schizophrenia Research

Established in 2004, the prize was formerly known as the Baer Prize and was renamed in 2016 in honor of Board Members Milton and Tamar Maltz. The Maltz Prize is given to an investigator who has undertaken innovative and promising research in schizophrenia. Winners of this prize are selected by the Lieber Prize recipient(s) of the same year.

The Lieber Prize

for Outstanding Achievement in Schizophrenia Research

Francine M. Benes, M.D., Ph.D.
Harvard Medical School
2002 Lieber Prizewinner

Schizophrenia is a uniquely complex disorder affecting human cognition and emotion in the absence of any diagnostic histopathology. Sophisticated new technologies, however, are detecting microscopic and molecular alterations in regions of the brain that are likely related to the mediation of these abnormalities. The "high tech" capabilities of MRI technology to define relevant brain networks and "risk" genes for schizophrenia have been essential for understanding the pathophysiology of schizophrenia. Parallel PM studies are contributing to our understanding of this disorder by unmasking discrete alterations in the wiring of complex microcircuitrs that can be explored under controlled conditions using empiric models developed by basic neuroscientists. It has become clear that postmortem studies provide an essential bridge to studies in live human subjects with experiments conducted in vitro and in vivo using neurobiologic models. The latter make it possible to explore the validity and relevance of findings in schizophrenia subjects. Postmortem studies of schizophrenia are a sine qua non for defining specific microcircuitry abnormalities in schizophrenia, as they attain anatomical resolutions and molecular sensitivities that are a thousand times and a million times greater, respectively, than those attainable in live human subjects. While postmortem studies of schizophrenia are bringing us closer to understanding how microcircuitry abnormalities may be related to "risk" genes and abnormal brain networks, they also have the potential to translate brain findings into the development of innovative new treatments by providing essential information from a studies that span the entire technological continuum of translational neuroscience.

David L. Braff, M.D.
University of California, San Diego School of Medicine
2014 Lieber Prizewinner

Schizophrenia is a profoundly complex disorder of the human brain that results in devastating levels of disability. We have made progress in understanding schizophrenia in the context of the "gene-X-environment" (GXE) paradigm. In terms of genomics, large–scale projects have identified many common and de novo risk loci. One problem with this extensive work is that the effect sizes of the many risk loci remain quite small. These genetic risk loci do seem to tag long suspected aberrant CNS processes in schizophrenia vulnerability, including glutamate dysfunction, neuronal pruning dysregulation and inflammatory impacts. Also, while new medications are being developed, cognitive and sensory training interventions offer an exciting "non–drug" path to schizophrenia treatment. What will the future hold? There are three discovery pathways available: 1. A "game changing" serendipitous finding (e.g., the discovery of Thorazine as a treatment) 2. A dramatic "Kuhnian" insight such as Darwin and Mendel's concepts 3. Largely incremental advances (the most likely path). For example, using biomarker endophenotypic deficits of key, e.g., quantitative domains (e.g., in neurocognition) offer powerful discovery pathways. As I say to my students, "you are the luckiest people in science: although we have learned a lot about schizophrenia, there is so much left to do." The path to understanding and developing more effective personalized treatments based on genotype and biomarkers is inevitably going to be long but will also be exciting, and hopefully will eventually relieve the profound suffering of our patients and their families.

Benjamin S. Bunney, M.D.
Yale University
1987 Lieber Prizewinner

Since 1987, when the first Lieber prize was awarded, the development of new techniques has fueled an exponential growth in schizophrenia research and allowed investigation into many new areas of potential etiology and pathogenesis. In 1987 the human genome had not yet been mapped and techniques such as PCR and CRISPR-Cas9 had not been invented. Although the first gene transfer was performed in 1980, gene manipulation allowing for knock-in and knock-out animal models did not exist. The "dopamine hypothesis" of schizophrenia still provided the impetus for a lot of the schizophrenia related research. Now, thanks to the work of thousands of researchers, funded by NIMH and the Brain and Behavior Research Foundation, we are gaining confidence that schizophrenia is a CNS developmental disorder. It is posited that mistakes in the piecing together, shaping and malfunctioning of brain structures occur, which are caused by two, as yet unidentified, converging elements–genetic predisposition and environmental events. Neither has been identified but reams of data are accumulating regarding the possible genes involved, the proteins for which they are responsible and environmental factors, both pre- and post-natal. Relatively young fields such as neuroengineering are yielding powerful and ever improving techniques, optogenetics being an example, to help us understand the function of specific neurons within neuronal circuits. Given the growing plethora of possibly relevant research findings, it will take new techniques and continued research to determine their salience for biomarkers as well as etiology and pathogenesis. For example, techniques for mining Big Data that are becoming more and more powerful as they are combined with developments in artificial intelligence may be helpful in this regard. Thanks to the efforts of many researchers, we have come a long way since 1987 in our understanding of schizophrenia. But we won't reap the benefits of our new knowledge without continued funding to support the army of researchers now dedicated to obtaining better treatments and, ultimately, prevention.

Marc G. Caron, Ph.D.
Duke University Medical Center
2013 Lieber Prizewinner

Schizophrenia is an inherited and complex brain disorder likely resulting from a landscape of genetic mutations. Although each mutation explains only a minute portion of disease burden, many of these mutants point to functional imbalances in neuronal brain circuits as being responsible for both positive

and negative symptoms of the disease. Current antipsychotic therapies target the brain dopamine systems via blockade of the G protein coupled receptor (GPCR) D2 dopamine receptor (D2R) to lessen positive symptoms, but fail to correct the cognitive and executive function deficits associated with negative symptoms. Thus, better therapies are needed with broader efficacy. To this end, we have leveraged the newly appreciated concept that GPCRs can signal not only through conventional G protein activation but also through the kinases and ß-arrestins components of the so-called desensitization pathway, to mediate distinct cellular and physiological responses. We have recently provided proof-of-concept in animal models that a novel ß-arrestin-biased D2R ligand can, like antipsychotics, block the hyperdopaminergic tone presumably responsible for the positive symptoms, but unlike typical antipsychotics, can simultaneously enhance DA function in mesolimbic neurons. Current work is testing the hypothesis that this ß-arrestin-biased D2R ligand will ameliorate cognitive and executive functions in preclinical models. A therapeutic agent that can deliver multifaceted restoration of dopamine brain functions should transform the treatment of psychotic disorders.

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

University of Maryland
2000 Lieber Prizewinner

Advance in knowledge and concepts are rapidly changing opportunities in science related to schizophrenia. Of profound importance is the far more systematic addressing of the heterogeneity of schizophrenia. There is a recognition that there are many different symptoms associated with the diagnosis and that people with this diagnosis vary as to which symptoms are present and when they developed. Science is redirected from the diagnostic level to the actual pathology, with implications that cross current diagnostic boundaries. Brain mechanisms and their causes will vary across cases, and so too will therapeutic discovery. New discovery in schizophrenia will be informative for other disorders where some of the patients have the same pathology. Perhaps the most promising area of science with clinical impact relates to prevention. Recent work has confirmed that young people with mild but clinically relevant symptoms are at increased risk for developing a psychotic disorder. Initial clinical trials suggest that treatment can reduce symptoms and progression to full psychosis. This sets the stage for detecting risk even earlier, before the onset of symptoms. It is hoped that current studies will yield information relevant to identifying persons at risk in advance of symptoms and that knowledge on the genetic and environmental risk factors will provide a basis for primary prevention approaches. The study paradigm thus moves from treatment to primary prevention with the aim of reducing the incidence of schizophrenia and related psychotic disorders.

Joseph T. Coyle, M.D.

Harvard University
2004 Lieber Prizewinner

Since commencing my involvement in neuroscience research 50 years ago as a medical student in Sol Snyder's laboratory, schizophrenia has been the "holy grail" for me. However, my experience directing a schizophrenia outpatient clinic convinced me early on that the dopamine hypothesis was not totally explanatory. In a an experimental detour that changed the trajectory of my research career from focusing on catecholamines to glutamate, Robbie Schwarcz, my first fellow, and I drew from the observations of John Olney that systemic glutamate killed neurons in the infant rat brain. We also took advantage of the potent glutamate receptor agonist, kainic acid, characterized by Japanese neuropharmacologists and showed that when injected into the rat striatum it recreated the pathology of Huntington disease. Searching to better understand how excessive activation of glutamate receptors might account for human neurodegenerative disorders such as Huntington disease and Alzheimer disease, my laboratory isolated a neuropeptide implicated in negative modulation of glutamatergic neurotransmission. Our postmortem study demonstrated that its catabolism was reduced in cortex in schizophrenia. This led to the hypothesis that the fundamental deficit in schizophrenia was hypofunction of NMDA receptors. This proposal dovetailed with the results of human and animal studies with the NMDA receptor antagonist, ketamine. NMDA receptor hypofunction in cortex best accounted for the cognitive impairments and motivational deficits that accounted for persistent disability and was the proximate cause of psychosis. The last 15 years have been particularly productive as we have pursued this hypothesis in my laboratory and with collaborators in our NIMH Conte Center, taking advantage of molecular techniques. To recreate NMDA receptor hypofunction, we silenced the gene, serine racemase, that is responsible for synthesizing the NMDA receptor co-agonist, D-serine, and showed that it reproduced the cortical atrophy, synaptic pathology, cognitive deficits and the neurochemical pathology of schizophrenia. Furthermore, these deficits could be largely reversed by treatment in adulthood by restoring D-serine brain levels or treating with a mGluR3 positive allosteric modulator. The results were validated by the genetic findings that a dozen risk genes for schizophrenia including serine racemase are within two degrees of separation from the NMDA receptor. As we look to the future, we must recognize that having the risk gene does not guarantee a rapid pathway to treatment (witness Huntington disease). Nevertheless, we may be reaching a critical mass of knowledge of brain disorders so that like in the case of cancer we may have achieved a tipping point that leads to an accelerating discovery of more effective treatments. I want to emphasize that NARSAD played a critical role at each stage of my journey.

Robert Freedman, M.D.

University of Colorado, Denver
2015 Lieber Prizewinner

The epidemiology of schizophrenia points to prenatal brain development as a special period of risk, in which genes that are associated with schizophrenia are working to construct the brain. At this point, the brain problems that later give rise to schizophrenia are already being formed. Infection of the mother can compound the problems. To prevent schizophrenia in these children, we need to investigate how to prevent these brain abnormalities from ever forming. My own research has identified a promising treatment, increase of the mother's intake of the nutrient phosphatidylcholine, which ameliorates the effects of some genetic risk and infections. Children whose mothers received this treatment are now reaching 4 years old and are less likely to show attention and social problems seen in children who develop schizophrenia as adults. I foresee the field developing this treatment and others to produce children who are born resilient instead of prone to mental illnesses.

Michael F. Green, Ph.D.

University of California, Los Angeles
2016 Lieber Prizewinner

My impression is that the next phase of schizophrenia research will revolve around the idea of recovery. The goal will be to enhance the ability of people with schizophrenia to integrate fully into the community. We do not expect miracles–after all, these are individuals who have had functional challenges for most of their lives. However, we can envision a time when people with schizophrenia can maintain personal connections to friends and family, find someone to love, attend college, and a hold a job successfully. To accomplish this, we need to know two things. First, we need a detailed understanding of what is holding back people with schizophrenia from more complete integration. For example, we know at a general level that these include problems in cognition (i.e., processing social and non-social information in their daily lives) and in motivation (i.e., a desire to engage community life and other people). Second, we need effective treatments for these problems. Such treatments will likely involve some combination of novel drugs, innovative training methods, and perhaps new approaches such as neurostimulation. It is now abundantly clear that the functional disability associated with schizophrenia is persistent, substantial, and multifaceted. Similarly, the scientific approach to address this problem will need to be persistent, substantial, and multifaceted.

Paul Greengard, Ph.D.

The Rockefeller University
1996 Lieber Prizewinner

Until recently, remarkably little was known about the causes of schizophrenia, or even about the regions of the brain involved in this disease. In recent decades, we have learned that there are a large number of diverse types of nerve cells in the brain and that the different cell types have extremely varied compositions of the proteins that they express. Using this background, schizophrenia researchers are now able to identify the regions, and the cell types within those regions, that are involved in schizophrenia. By manipulating the amount and function of specific proteins in specific brain regions, scientists are able, for the first time, to identify signaling pathways that lie at the basis of schizophrenia. Genetic studies in which a given protein can be increased or decreased in amount in animal models of schizophrenia enable us to test hypotheses concerning the role of such proteins in causing or combating the disease process. Identification of such proteins also permits the development of pharmaceutical compounds aimed at either increasing or decreasing the activity of such proteins. Through an iterative process, we can anticipate highly effective drugs with minimal side effects within the next few years.

Eve C. Johnstone, M.D.

University of Edinburgh, Scotland
2007 Lieber Prizewinner

I qualified in medicine in 1967 and I worked in academic psychiatry, principally in schizophrenia research from 1972 until 2010 when I retired. Nowadays I am only peripherally involved in research and not at all in clinical practice. From my own point of view my research career worked out very well–we made some findings that I think are really worthwhile. I was lucky to be in the right places at the right time and I had some wonderful colleagues. The work I did essentially depended upon three main methods of investigation-imaging, treatment trials and clinical/outcome studies. Non-invasive imaging was just coming in at an ideal time for me and it has lasted as a useful investigative technique for more than 40 years. Nowadays it is probably most useful in combination with other techniques such as genetic work. Clinical trials are less fashionable now probably because we need new treatments and new theories to drive the development of such treatments. Clinical/outcome studies are very labor-intensive and you need a really worthwhile question to justify the level of investment. Other techniques are now providing new possibilities and I am particularly interested in stem cell work using blood and skin samples from people whose psychoses are associated with minor genetic anomalies. This work is particularly valuable in relation to extended families with individual members with and without the anomalies and with and without the psychoses. We have such families from our large clinical/outcome studies and while I could not do the lab work, I am fascinated by the findings and happy to see my case material of ongoing value. I hope that the testing of batteries of drugs against the dishes of relevant stem cells may ultimately give use the effective new treatments that we need.

Kenneth S. Kendler, M.D.

Virginia Commonwealth University
1995 Lieber Prizewinner

I received the Lieber Prize in 1995, during another era of research in the genetics of schizophrenia. To everyone's satisfaction, twin and adoption studies had demonstrated that genetic factors made a major contribution to the etiology of schizophrenia. Linkage studies were starting to get going, but no major successes had yet occurred (or were going to occur). Now, in 2017, the field has been transformed in ways that would have been impossible to predict 22 years ago. While genome-wide association studies took time to prove their utility, they have now been spectacularly successful, although the effect size of the loci identified are all small. We are beginning to clarify other

parts of the genetic architecture. Sequencing is still in early days but the broad outlines are becoming evident. The impact of large genome anomalies–copy number variants–are also increasingly understood. The greatest question ahead of us now is to find the biological stories contained in all these statistical signals. Can we translate these findings into increased understanding of biological mechanisms of illness? And, if so, can we used these biological insights to develop new ways to prevent or treat these disorders? I am cautiously optimistic. The problem is a very complex one. But our tools are improving and the research community is growing, in size and sophistication.

Joel E. Kleinman, M.D., Ph.D.

National Institute of Mental Health
2011 Lieber Prizewinner

The identification of genetic variants that increase risk for schizophrenia is one of the major advances for research on this syndrome. In so far as these variants are species-specific, postmortem human brain tissue has been critical for identifying the molecular mechanisms associated with these genes. The identification of specific molecular mechanisms has the potential to help us elucidate how environmental factors interact with genes to cause schizophrenia. Moreover, this approach will hopefully give us new targets for improved treatments for schizophrenia, especially for cognitive deficits. Postmortem human brain studies have been one of the areas of my expertise for 40 years allowing me the opportunity to contribute to this line of research in schizophrenia.

Jeffrey A. Lieberman, M.D.

Columbia University
2006 Lieber Prizewinner

We may be approaching a tipping point in our historic efforts to elucidate the causes of schizophrenia and alleviate its symptoms and prevent disability. Three areas of research are leading the way. Genetic studies are revealing the architecture of schizophrenia's heritability and causation. Findings of genetic mutations (inherited and sporadic) that have large effects and high penetrance can point to therapeutic agents targeting gene products outside of the scope of psychotropic drugs and that would never have been considered. At the same time biomarkers, using imaging and electrophysiological procedures in particular, are being used to identify people who are at imminent risk of developing schizophrenia and signaling the need for intervention. These also are highlighting the neural circuitry of schizophrenia and potential therapeutic approaches using neuromodulation. In this context, collaborative multi-specialty care provided to individuals at the initial stages of their illness are now capable of enabling recovery and limiting the damage caused by schizophrenia or even preventing its onset. All in all, we can do more to help people with schizophrenia than ever before. It's now a matter of providing these services in a timely fashion and enabling their access. Moreover, with continued research, improved treatments and even eventual cures can be expected to follow.

Stephen R. Marder, M.D.

University of California Los Angeles
2016 Lieber Prizewinner

I anticipate advances in the treatment of schizophrenia that will occur in the next decade and beyond. The most important advances that can affect people who are currently living with schizophrenia may emerge from research focusing on non-pharmacological treatments. In this area, strategies for improving cognition, negative symptoms, positive symptoms, and social cognition have been shown to be effective, with effect sizes indicating that the amount of improvement is meaningful. Unfortunately, these findings have not affected clinical care. Advances are likely to emerge from studies that implement these interventions and deliver them to the most appropriate patients at the right time. New pharmacological strategies or neuromodulation may facilitate the effects of these interventions, but they will not be the primary treatment. I am most excited about treatment research that will be disease-modifying. That is, pharmacological or non-pharmacological interventions that target the underlying pathology in the brain–perhaps an inflammatory process or a defect in synaptic plasticity–that would be delivered before the illness emerges or at the early stages of the illness and would change the course of the illness. This is a very ambitious goal and one that will require developments in genetics, basic neuroscience, and clinical science. Fortunately, BBRF has played a major role in supporting the development of scientists who are involved in all of these research areas.

Patrick McGorry, M.D., Ph.D., FRCP, FRANCZP

Orygen & University of Melbourne, Australia
2015 Lieber Prizewinner

The future of schizophrenia research is dependent upon a paradigm shift to a transdiagnostic approach to aetiology, neurobiology and treatment. Schizophrenia researchers have led the psychiatric world in moving from a late– or end–stage focus to early diagnosis and a focus on the earliest stages of illness. This has shown that the course of illness is plastic and can be greatly improved if timely evidence–based care is provided. In doing so we have learned that the need for care precedes diagnostic "clarity" according to a diagnostic system that was constructed over a century ago, based on late macrophenotypes. The early microphenotypes are fluid, can lead in many directions and comorbidity is the rule rather than the exception at all stages of illness. So phenotypic diagnostic clarity may ultimately be a mirage, or at least a matter of degree in a more dimensional sense. Our psychosocial and biological treatments generally lack specificity for the current diagnostic categories, especially early in the course. Later on, the ubiquity of comorbidity underpins what is often derided as "polypharmacy." A transdiagnostic clinical staging model could be validated both by clinical trial data, and also by key biomarkers which, in reflecting potential mechanisms of disease (genetic research will guide this search), are likely to extend across diagnostic boundaries and hence redraw boundaries. This implies the critical need to study and follow early–stage samples across the diagnostic spectrum accessed via primary care and youth mental health platforms. I expect genetic as well as biomarker research to increasingly support the transdiagnostic approach and the search for mechanisms, rather than, as once hoped, the validation of flawed and historical conceptions from the age of steam. A crucial feature of future successful research, and indeed a civilized society, is dramatically increased global investment to guarantee evidence–based care for all people with schizophrenia and mental illness across the spectrum.

Herbert Y. Meltzer, M.D.

Northwestern University, Feinberg School of Medicine
1992 Lieber Prizewinner

The most important advance in mood disorder research in the near term will be the further development of mood–altering and suicide–preventing drugs for major depression and bipolar disorder, based upon rapastinel (GLyx 13) and ketamine–related formulations. We can expect more effective and safer drugs than these breakthrough agents, based upon further research into their mechanisms of action. These will be orally available agents which can be used as first–line and maintenance treatments, rather than reserved for treatment failures and to avert suicide. Rapastinel, as my lab has shown, has potential to also address the cognitive impairment present in mood disorders, which would address a major unmet need in their treatment. Indeed, I expect future research to clarify how stress contributes to both mood symptoms and cognitive impairment in the major mood disorders. We can also expect advances in pharmacogenetics to extend current knowledge about biomarkers for suicide, e.g., a mutation in the cholesterol synthesizing gene, ACP1, which we and others have shown to be the top biomarker for suicide risk in bipolar disorder and schizophrenia. Pharmacogenetic research will also lead to diagnostic tests to facilitate diagnosis and choice of medication in mood disorders.

Sir Robin M. Murray, M.D., F.R.S.

King's College, London
2003 Lieber Prizewinner

For a UK psychiatrist, one troubling development in the USA is the increasing use of cannabis (marijuana). Marijuana is like alcohol; the majority of people who use it sensibly come to no harm, but heavy users increase their risk of adverse effects. It is now clear that heavy use of high potency varieties of cannabis increases risk of psychosis; in London, one quarter of the patients we see with schizophrenia would not have developed it but for their heavy use of marijuana. Cannabis is more potent than it used to be. In the 1960s, marijuana contained only about 3–4% of tetrahydrocannabinol (THC), but now high potency varieties often contain 16–20%. Furthermore, in some states one can legally buy preparations with 40% or 60% THC, and of course synthetic cannabinoids such as K2 and spice are available on the internet. In the UK the average psychiatrist, and indeed the average young person, is aware of the risks, and consumption of cannabis has declined over the last 10 years. As cannabis use becomes more common in the USA, it is vital to study the mechanisms underlying cannabis-associated psychosis, and to monitor the effects of legalization on mental health.

Michael J. Owen, M.D., Ph.D.

Cardiff University, UK
2012 Lieber Prizewinner

We now stand at a point of great opportunity arising from the confluence of three streams of scientific endeavor. First, recent genomic studies have identified a substantial number of risk alleles for psychiatric disorders including schizophrenia, bipolar disorder and autism, and we can expect further advances over the next 5 years. One of the key discoveries has been that genetic risk does not obey current diagnostic boundaries, with many risk variants instead increasing susceptibility across a range of disorders. Pathways of risk are beginning to emerge from the genomic findings from within disorder, and cross-disorder studies. Although these are currently rather broad and do not provide for clear mechanistic understanding, the findings point to the importance of particular synaptic proteins, immunological pathways and epigenetic regulators. Second, advances in stem cell biology and genome engineering now allow human disease risk to be modeled with high construct validity in neuronal cells. Third, advances in neuroscience allow us to probe disease mechanisms across development and at neuronal and systems levels in animal as well as cellular models, and in humans. The challenge now is to integrate advances across these three areas to identify disease mechanisms and new drug targets and to develop new diagnostic strata that map more closely onto underlying mechanisms and define patient subgroups for treatment studies.

Philip Seeman, M.D., Ph.D.

University of Toronto, Canada
1990 Lieber Prizewinner

There are many causes of schizophrenia, including spontaneous mutations in one's DNA, birth injuries, brain accidents, prolonged isolation, and long-term misuse of addicting drugs. Despite the different causes, the signs and symptoms of schizophrenia are similar, with positive symptoms such as delusions and hallucinations, and negative signs, such as social withdrawal, and an unrealistic recognition of reality. While the positive signs are alleviated by antipsychotic medication, the negative signs are less responsive to medication. All the antipsychotic drugs act by a similar mechanism of interfering with the transmission of dopamine within the brain, either by blocking the type 2 dopamine receptor or by competing with the natural dopamine in the brain. The dopamine type 2 receptor, known as the D2 receptor, can exist in either a state of high affinity for dopamine or a state of low affinity for dopamine, analogous to hemoglobin that has red and blue states of existence. Although animal models of schizophrenia do not properly reflect this uniquely human disease of schizophrenia, all animal models show an elevation in the number of high-affinity states of the brain's dopamine type 2 receptor. For the future measurement of D2High states in humans, the current

work is to develop markers to label D2High states for diagnosis and treatment.

Solomon H. Snyder, M.D.

Johns Hopkins University
2001 Lieber Prizewinner

Much of the research that has given insights into brain mechanisms of schizophrenia has involved studies elucidating the mechanisms of action of antipsychotic and psychotomimetic drugs. Thus, blockade of dopamine D2 receptors by classic antipsychotic/neuroleptic agents led to the "dopamine" hypothesis, namely that an excess of dopamine neurotransmission may be pathophysiologic. The close resemblance of ketamine psychosis to schizophrenia led to interest in the mechanisms of ketamine action, specifically its blockade of glutamate-NMDA receptors. This implied that agents stimulating such receptors, especially their "glycine" site, may be therapeutic. More recently, genome research has linked over a hundred genes to the propensity for schizophrenia. Unfortunately, each of these genes appears to contribute only a small portion of the risk for schizophrenia, restricting the utility of such findings. Nonetheless, the strong association of specific genes to the disease hints at specific aberrations that may be pathogenic. The fact that many of the schizophrenia-associated genes are associated with synaptic function bolsters the relevance of this line of research. Particularly tantalizing is the possibility that a concatenation of several predisposing genes triggers psychosis. In summary, insights from drug actions are giving way to a focus on defined genetic mechanisms that may tell us much about the origins, progression and therapy of schizophrenia.

Patrick F. Sullivan, M.D., FRANZCP

University of North Carolina & Karolinska Institutet, Sweden
2014 Lieber Prizewinner

Schizophrenia research has always been hard. This is one of the toughest and most complicated disorders in all of medicine. However, it's beginning to change. We finally have a minimally adequate set of scientific tools that we can use to ask the right questions of the brain, the most complicated machine known to us. We are beginning to get a full idea of the genetic changes that underlie schizophrenia. Due to unprecedented international collaboration, we are working hard to deliver "actionable" findings that reveal the fundamental biology, inform clinical practice, and deliver new therapeutic targets. We think that we have identified the cell types that confer risk for schizophrenia. The genetic findings agree reasonably well with known and predicted drug targets. For at least one of our patients, we have been able to fully explain precisely why he became ill. Prediction of the future is always hazardous but, given that we finally have a minimally adequate toolkit, it is possible that we are entering a golden age of research into the fundamental basis of schizophrenia.

Ming T. Tsuang, M.D., Ph.D., D.Sc.

University of California, San Diego
2010 Lieber Prizewinner

It is imperative to the future of schizophrenia research that we focus on ways in which we can identify psychosis before onset and intervene early – with the aim being early recovery/good outcome and/or complete prevention. The search for biomarkers which predict early psychotic symptoms at the prodromal stage is a worthy and important direction to follow. These biomarkers may be genetic, neuroanatomical, neurochemical, neurophysiological, or psychosocial. In the future they will be ascertained through genetic studies, neurophysiology research, outcome studies and neuroimaging, for example. As we have recently learned, one of the strongest genetic associations at a population level involves genes which have an immune function. This is based on work done on very large samples such as the Psychiatric Genetics Consortium which identified genes on chromosome 6 having a strong association with schizophrenia. Dysregulation of immune system functions has been a feature of recent work on schizophrenia etiology. By comparing the RNA from immune cells among clinical high risk patients vs. controls, in the future, RNA may be used as a biomarker assay for psychosis risk, while immunotherapy may become a viable approach for clinical treatment. Recent prospective longitudinal neuroimaging studies on prodromal subjects have shown accelerated gray matter loss and third ventricle expansion around the time of onset of psychosis. Steeper gray matter loss seems to be unique to those Clinical High Risk (CHR) individuals with higher levels of sub-psychotic pre-delusional symptoms. This may reflect pathophysiological processes driving emergence of psychosis. Identification and intervention of this brain matter loss is a future goal for investigators. Furthermore, we must stay focused on developing coping skills to address the everyday challenges of schizophrenia through psychosocial research. Should we canvass recent discoveries it appears that schizophrenia research has an exciting future as our technology, knowledge and methods advance. Disciplines recently thought to be nowhere near the ambit of neuropsychiatry research – such as the microbiome – are now being interrogated, while genetics, neuroimaging and psychosocial research continue to make great strides in identifying, intervening, and preventing schizophrenia.

Daniel R. Weinberger, M.D.

The Lieber Institute, Johns Hopkins University School of Medicine
1993 Lieber Prizewinner

Schizophrenia research has taken a giant leap forward from the time that I was honored with the Lieber Prize in 1993. We have learned more in the past two decades about the causes and mechanisms underlying major psychiatric disorders than in all of prior history. BBRF has played a major role in this sea change in general and in my scientific work in particular. When I started my research career at the NIMH, the overarching goal was to uncover objective scientific evidence that the brain was the main actor responsible for mental illness. Refrains from an earlier era attributing psychiatric illness to family dynamics still echoed in the hallways of the NIH. My early work involved applying neuroimaging technology to explore how structural and functional variations in the brain were associated with schizophrenia. We showed in those studies that the brain is definitively involved. We also surmised from associated observations that these brain changes reflected events in brain development from much earlier in life. But, we did not know how these changes arose and how they translated into the illness. We had evidence of relevant biological phenomena but no information about causation. The recent discovery of genes related to risk for schizophrenia has changed the research landscape profoundly and enduringly. Genes represent causative mechanisms and they open a new era in diagnosis, treatment and prevention. We are far from achieving the full promise that information about basic mechanisms offers, but we are seeing a seismic shift in how we study schizophrenia and related conditions. It is now evident that in most cases, there are no singular causal 'schizophrenia genes' per se, but rather numerous, maybe up to thousands of genetic variants in the broad population that contribute small increments of risk to this complex psychiatric syndrome. Identifying genetic variants that confer schizophrenia risk and elucidating their impact on the underlying neurobiology of the disorder are critical, albeit formidable, tasks, given the vast unknowns and complexity of normal and diseased brain function. The earlier circumstantial and epidemiological evidence linking the origins of schizophrenia to prenatal life have been validated by molecular studies in post mortem human brain, which have shown that genes associated with schizophrenia risk affect early neurodevelopmental processes such as neuronal differentiation and maturation. An emergent inference then is that early brain development mediates schizophrenia genetic risk, with fundamental implications for pathogenesis. These discoveries and insights provide a rich opportunity to find new treatments and new approaches to prevention. No treatment used in psychiatry today, whether talk or medical, was discovered based on an understanding of causative mechanisms and pathogenesis. Having objective clues to causative mechanisms has to be a better approach. I anticipate that within the next decade, we will see new treatments emerge based on this new research data. I anticipate that we will identify combinations of genetic and environmental signatures of increased risk which will be used to target high-risk individuals for preventive monitoring and improve long term outcome. And, because of these breakthroughs and the support of organizations like BBRF, psychiatry research will emerge as a top choice career for the best and the brightest of the next generation of research scientists. ❐

The Colvin Prize

for Outstanding Achievement in Mood Disorders Research

(formerly known as the Selo, Falcone & Bipolar Mood Disorder Prize)

Hagop S. Akiskal, M.D.
University of California, San Diego
2001 Falcone Prizewinner

Mood disorders today are being investigated in specialized mood clinics where patients have thorough evaluations with semi-structured interviews. Clinical, interpersonal, and biological data are gathered in a systematic fashion. Interpersonal and cognitive-behavioral therapy, as well as marital counseling, is used adjunctively with other patient-friendly psychotherapies. Modified psychodynamic psychotherapy is still alive. Many clinics focus on women, and this is particularly true for seasonal depressions. Subthreshold mood disorders have acquired particular significance from an epidemiological and preventive perspective. Moderate to severe depressions, associated with middle age and later life are increasingly receiving stimulation-type treatments. Both psychosocial family and pharmacotherapeutic approaches are being used in children. This literature is more of an art than a science. A plethora of new specific and non-specific pharmacological agents have entered the market. From a preventive perspective, it is important for primary care physicians and internists to have patients screened in their offices on specialized questionnaires with special attention to identify suicidality, and its prevention is a clinical art in need of becoming a science; furthermore, the boundary between depressive, bipolar, and ADHD is in need of changing from art to clinical science. Suicide prevention both at a population level and in clinical practice represents a new frontier, yet ECT remains a necessary tool. The stereotype that women try and men [succeed in] kill[ing] themselves is a dangerous stereotype. The integration of different psychosocial approaches and therapeutic and biological technologies, recognized by NIMH and NARSAD, will delineate future vistas. The greatest challenge in understanding human nature resides in the genetic factors in artistic and scientific creativity. Thus, molecular genetics represents the most promising frontier. As Sir Martin Roth has said, "Psychiatry will remain the most human of the sciences and the most scientific of the humanities."

Robert H. Belmaker, M.D.
Ben-Gurion University, Israel
2000 Falcone Prizewinner

My area of research for over 40 years has been the mechanism of action of medicines that ameliorate the symptoms of bipolar disorder. For many years I was convinced that unraveling the mechanism of these compounds would lead to a single common mechanism and that the understanding of that single common mechanism would allow us to understand the single central biochemical abnormality predisposing to bipolar mood swings. I probably would have been willing to take a significant financial bet when I finished psychiatric residency that I would have that answer by the time I retired. However, after about 20 or 30 years of research it began to seem much more likely that the various classes of medicines that help bipolar disorder have very different mechanisms of action and their common point of convergence has not been found, even though many of them have similar clinical indications. Moreover, my study and that of others of the mechanism of action of therapies of bipolar disorder has not converged on genetic and biochemical studies of the causes of bipolar disorder, which also seemed to be vary varied in many areas of the genome and with disparate pathophysiological relationships to areas as far flung as inflammation and cannabis abuse. Imaging of the brain, which I once thought would never be likely to lead to results on the molecular level that I believed necessary to understand bipolar disorder, now has reached a level of precision that is truly inspiring. My prediction is that the combination of neuroimaging with advanced computer neuropsychological testing under laboratory conditions of model stresses, will lead to the discovery of those circuits that function inadequately in those predisposed to bipolar disorder. I predict that we will thereby learn how to strengthen these circuits not by pharmacological means or by electrophysiological brain stimulation, but by exercises using computer based neuropsychological programs during the well and even preventive phase of bipolar disorder.

Michael Berk, Ph.D., MBBCh, MMed, FF(Psych)SA, FRANCZP
Deakin University
2015 Colvin Prizewinner

Mood disorders and depression in particular are a deceptively simple construct, as currently defined. However there are many paths to depression, spanning a normal adaptive physiological reaction to serious brain disorder, driven by factors as diverse as personality, lifestyle and genetics. This heterogeneity makes algorithm based diagnosis and management complex and unreliable. The ability to personalize treatment, either by diagnostic stratification or biomarkers remains a critical goal, although success to date has been very limited and has not led to widely clinically adopted measures. Incorporating predisposing, precipitating and perpetuating factors into clinical formulation and hence management remains best practice. A second area of future development is the study of the role of non-monoaminergic pathways in pathophysiology. These span inflammation, neurogenesis, apoptosis, redox signaling and mitochondrial function, and these are increasingly being explored via systems paradigms and –omics technologies. Importantly, these have the capacity to suggest novel therapeutic approaches. Lastly, it's worth noting that the major successes in areas such as cancer and cardiovascular disease have been in prevention, not cure. Psychiatry has lagged in this domain, not least because of the complexity of the risk pathways. Nevertheless, known risks can be targeted, including lifestyle factors such as diet, smoking and physical activity, social factors such as community and social engagement as well as institutional responses to factors such as sexual and physical abuse. It's clear that many of these factors can only be realistically targeted via public health and policy approaches, noting that many of these risks are common across non-communicable disorders, reinforcing the necessity for cross-disciplinary public health approaches.

Wade Berrettini, M.D., Ph.D.
University of Pennsylvania, Perelman School of Medicine
1996 Selo Prizewinner

The development of the first monoamine oxidase inhibitors and tricyclic antidepressants occurred roughly 55–60 years ago. For the next 50 years, there were no antidepressants successfully developed which did not act directly on one or more monoamine molecules, including their receptors, transporters and enzymes. Despite the clinical successes of these medications in mood and anxiety disorders, primarily, many patients with recurrent unipolar disorders were not helped by these useful monoamine-based agents. Remarkably, in the past 5 years, a new antidepressant, not acting directly on monoamines, has been identified, buprenorphine. Buprenorphine is FDA-approved for the treatment of pain and opioid addiction. Its efficacy in pain and opioid addiction is due to its activity as a mu opioid receptor partial agonist. The antidepressant activity of buprenorphine is due to its kappa opioid receptor antagonism. The antidepressant benefit may be evident at 1/20 the dose used to treat opioid addiction (typically 16–24 mg daily). There is evidence that buprenorphine may benefit depressive patients when used alone or in combination with monoamine-based antidepressants. Is buprenorphine a harbinger of successful development of alternative classes of antidepressants? I hope so.

Boris Birmaher, M.D.
University of Pittsburgh School of Medicine
2013 Colvin Prizewinner

Untreated, bipolar disorder (BP) has devastating consequences for the psychosocial development of the child. Fortunately, new developments are shedding light on who is at risk to develop BP and the factors associated with better longitudinal course and outcome. The Pittsburgh Bipolar Offspring Study (BIOS) found that youth with persistent anxiety/depression, mood lability and subclinical manic symptoms, and particularly those whose parents had early onset BP, have a 50% likelihood of developing this illness. Although important, these results apply to the group as a whole and not for an individual subject. Thus, similar to the risk calculators created to predict individualized risk of developing cancer or myocardial infarction, BIOS created a risk calculator to predict the risk of developing BP for a specific child. This calculator will be instrumental for future biological studies and for the development of preventative treatments that can delay the onset of this illness until the child has developed the cognitive and social skills to cope with the disorder. Once youths have developed BP it is important to be able to predict their long–term functioning. Contrary to the common idea that BP usually is associated with poor prognosis, the Course and Outcome of Bipolar Youth (COBY) study showed that a substantial subgroup of youths with BP with certain characteristics are persistently euthymic [i.e., not depressed]. These findings give hope to families and youth and are informative for the long–term treatment of this illness.

William E. Bunney, Jr., M.D.
University of California, Irvine
2001 Falcone Prizewinner

In the last decade and a half, significant progress has been made in the understanding and treatment of mood disorders. Most current antidepressants require 2–10 weeks for significant improvement while low doses of ketamine produce a rapid response within 24 hrs. However, some patients experience a brief episode of psychotomimetic symptoms. Researchers are attempting to identify ketamine-like compounds without these side effects. A subset of depressed patients has abnormal 24–hour rhythms affecting sleep, temperature, mood and hormonal secretion, which are controlled by clock genes. Depressed patients compared to controls have dramatically altered clock genes in the brain. Ketamine interacts with clock genes and it has been hypothesized that ketamine may reset the abnormal clock genes. One-third of patients who commit suicide visit a healthcare professional in the month prior to suicide. New molecular and clinical markers could be used to identify individuals at high risk for suicide. Future research will accelerate the investigation of risk genes and genes that can protect against depression. A revolutionary technique that involves precise editing of genes is called CRISPR-cas13a which allows for diagnostics and disease monitoring. Finally, a 3D technology, CLARITY, will facilitate the study and identification of neuropathways in the human brain.

Joseph Calabrese, M.D.
Case Western Reserve School of Medicine, Cleveland, Ohio
2004 Selo Prizewinner

As a result of good mentors and generous donors, I have had the good fortune of being able to contribute to the development of mood stabilizers, including lithium, the atypical antipsychotics, the selective serotonin-re-uptake inhibitors, the anticonvulsants, and most recently, the D3- preferring D2/D3 partial agonists. Our scientific focus has always been the development of mood stabilizers for use in the treatment of bipolar disorder, and in particular, those that targeted the depressed phase of the illness–the phase where patients lived their symptomatic lives, and unfortunately, in many instances, ended their lives. I owe all of this to my wonderful mentors at the National Institute of Mental Health and my donors. Whereas we have enjoyed the benefits of serendipity over past years, we are now entering a new generation of clinical research, research that thoughtfully targets pathophysiology based upon precise science.

J. Raymond DePaulo, Jr., M.D.
Johns Hopkins University School of Medicine
1996 Selo Prizewinner

Mood disorders are common, costly, and disabling. Major depression and classic mania (i.e., bipolar disorder) can only be recognized clinically. Severe forms of them can be well treated. However, patient frequently fail to get effective treatment and we don't know why our treatments work or fail for our patients. We know many genes in the causal pathway to these conditions. We have other molecular clues from animal models. We have clues to treatment success from brain imaging, but we don't know how the genes and other molecular players reshape brain function to produce depression or mania, or to explain how they produce episodic patterns of illness. We need much more research: molecular and brain imaging surely, but we need to support large treatment and outcomes research to find predictors of outcomes, especially treatment response. We must interrogate molecules, synapses and circuits and we must develop better methods to engage and follow our patients so that they can partner more effectively in treatment. We need BBRF to grow and prosper; we also need the NIH budget to grow and to prioritize research in mood disorders. Finally we need to support collaborative communities (e.g., National Network of Depression Centers) where academic centers working with patients and families will conduct research to carry us from discovery to recovery.

Ronald S. Duman, Ph.D.
Yale University
2002 Falcone Prizewinner

Depression is a common, devastating illness and although current pharmacotherapies help some patients, the high rates of partial- or non-response and a delayed therapeutic onset leave many patients inadequately treated. However, new insights into the neurobiology of stress and human mood disorders have shed light on the mechanisms underlying the vulnerability of individuals to depression and have pointed to novel antidepressants. Environmental events such as chronic or traumatic stress, as well as other risk factors contribute to depression through converging molecular and cellular mechanisms that disrupt synaptic structure and neuronal activity, resulting in dysfunction of the circuit connectivity for mood regulation and cognition. Although current antidepressants such as serotonin reuptake inhibitors produce subtle changes that take effect in weeks or months, new agents such as ketamine result in rapid improvement in mood ratings within hours of dosing in patients resistant to typical antidepressants. Moreover, these new agents reverse the synaptic connectivity deficits caused by stress within a similar time scale and underlie the rapid antidepressant behavioral responses. Current studies are focused on characterization of the molecular and cellular signaling pathways that mediate the rapid antidepressant actions of these agents and identification of novel targets for further development of safer agents with fewer side effects.

Elliot S. Gershon, M.D.
University of Chicago
1996 Selo Prizewinner

The major progress in psychiatric genetic research on mood and other disorders has been findings of genetic variants associated with bipolar disorder, and in the past year with major depressive disorder. Furthermore, there are significant overlaps of these findings, and of the polygenic predisposition, between schizophrenia and bipolar disorder. These genetic findings, based on tens of thousands of patient and healthy volunteers, are significantly enhancing our molecular understandings of the major mood and other psychiatric disorders. However, they have not yet led to improvements in pharmacologic or other treatments. If I have to pinpoint one direction in which I expect major progress in the next few years, it would be progress in molecular bases for diagnosis, course of illness, and in development of pharmaceutical or other treatments targeted to the individual patient's genetic constitution.

Mark S. George, M.D.
Medical University of South Carolina
2008 Falcone Prizewinner

Organizations like NARSAD are successful if they can support innovative research that disrupts the current modes of thinking about an illness. This radical research then fosters innovations that lead to new understanding and hopefully new treatments. When they are truly successful, a field may have a paradigm shift. NARSAD has been enormously successful in funding high-risk mood disorders studies decades ago that helped build a foundation for a total overthrow of old thinking with new theories and concepts. In 2000, depression was thought of as a chemical imbalance, which was largely acute (several months to a year), without lifelong sequalae. Because of the confusion with normal sadness, grief and worry, depressions were stigmatized. We now think all of these concepts are wrong. Depression is now discussed as a brain disorder involving disrupted circuits in the brain, much like Parkinson's Disease. Many acute episodes recur, and substantial numbers of patients have lifelong illnesses akin to diabetes. Stigma drops as people adopt this new line of thinking. There is a focus on early aggressive treatment with long-term follow-up and lifelong management. My own work proposing noninvasive brain stimulation as a treatment arose out of brain imaging studies of depression and sadness, and began to define these dysfunctional circuits. Because I was operating out of a different paradigm, my ideas and those of the other brain stimulation depression pioneers were not well received, particularly by a conservative NIH. Luckily, early NARSAD grants helped me continue my science. There is now tremendous excitement as the new paradigm emerges. Now, we have five different transcranial magnetic stimulation (TMS) companies with FDA approval, and universal insurance approval of TMS for depression. We can use sophisticated brain imaging to see which circuits are not acting correctly in depression, and then document their return to more normal activity after a successful course of TMS. We have even now shown that a therapeutic course of TMS over 4-6 weeks actually regrows the brain in some of the mood regulating regions that were not working. Some researchers are proposing individualized medicine approaches where psychiatrists position TMS directly over the brain region, for that person, that is not working. We now understand how talking therapies like cognitive behavioral therapy (CBT) work on changing circuits, and are combining external brain stimulation with devices like TMS with sophisticated behavioral "exercises," rapidly changing circuits and

enabling patients to have self-learned skills for the next problem that life throws at them. And the focus now is not just on getting patients undepressed, but keeping them remitted for years in order to rebuild their lives. We have not yet come up with a single "cure" for depression, but we are developing multiple different treatments, some aggressive and invasive, others relatively simple. With this expanded range of options, we are making a real difference in public health and outcomes for a set of diseases that troubles mankind and creates more disease burden than any other illness. Within the next decade, this NARSAD inspired revolution realistically may enable "cures" for most patients. All it takes is a bit more research.

Robert M. A. Hirschfeld, M.D.

Weill Cornell Medical College
2003 Falcone Prizewinner

Many years ago Stan Kenton, the great band leader and jazz legend in the mid-20th century, was asked by a reporter where jazz was going. Without hesitating, he replied, "we are going to Cleveland on Friday." My response to where the future of mood disorder research is going is similar—I know where I am going this weekend, but don't have a clue about the future of mood disorder research. That said, I fervently hope that personally targeted more effective treatments with benign side effect profiles will be developed. I spend considerable time as a clinician trying to ameliorate the naturally occurring mood swings and managing the distressing side effects of the treatments I prescribe.

Kay Redfield Jamison, Ph.D.

The Johns Hopkins University
2000 Falcone Prizewinner

I think the field is going full-ahead in directions that have great promise, and raise a few concerns. Mostly, great promise. The most generative fields, almost certainly, remain genetic and neuroimaging research, which will lead to earlier and more accurate diagnosis of mood disorders, help sort out the important relationship between mania (far too little studied) and recurrent depression, and generate more specific and less problematic treatments. We will better understand the enormously important relationship between pathological and normal mood states, as well as the relationship between temperament, cognitive styles (including those associated with imaginative thought), and moods. We will gain a much deeper understanding of ourselves in comparison with other species in terms of mood, energy, behavior, and cognition. As a result of increased knowledge and a more sophisticated ability to manipulate the human genome, we will be confronted with profound ethical issues in clinical practice and social policy.

Husseini K. Manji, M.D.

George Washington University
1999 Falcone Prizewinner

It is a pleasure to comment here on the progress and future of studying and treating mood disorders. Although considerably more research is undoubtedly needed, I believe that the tremendous research advances made in recent years — only a handful of which are reviewed below — hold considerable promise for making a real difference in the lives of individuals suffering from these devastating disorders. From the standpoint of novel therapeutics, one of the most exciting areas of research is the concept of synaptic plasticity, loosely defined as the processes that regulate the strength of a signal transmitted through a synapse. We now know that a major mechanism underlying synaptic plasticity is the trafficking of NMDA and AMPA receptor subunits. Taken together with clinical findings that low-dose ketamine (an NMDA antagonist) has considerable antidepressant efficacy and acts within hours or days, the evidence suggests that it may indeed be possible to treat severe treatment-resistant depression rapidly. This breakthrough concept goes against accepted therapeutic "dogma" in our field. Another exciting area of research suggests that neuroimmune cascades may play a role in mood disorders. Extensive data have shown that proinflammatory cytokines are elevated in mood disorders, but for many years they were thought to be linked only to the medical comorbidities associated with mood disorders (e.g., cardiovascular disease, type 2 diabetes). However, using agents that target these neuroactive cytokines, astute researchers found preliminary evidence that cytokines play a fundamental role in mood disorders. Formal clinical trials are under way. Much of the disability caused by mood disorders is due to the fact that they are highly recurrent. Thus, an ability to attempt to intercept disease progression, and to move away from a "diagnose & treat" model to a "predict & preempt" would undoubtedly have great benefit. In this regard, although mood disorders are not classic neurodegenerative disorders, in many patients, these disorders are associated with regional atrophic brain changes. Studies have shown that lithium exerts neurotrophic/neuroprotective effects which may underlie its ability to attenuate recurrences. Thus, there is considerable excitement about the ability to develop novel treatments to recapitulate many of lithium's beneficial effects with fewer side-effects. Finally, our ubiquitous mobile devices, equipped with small, unobtrusive sensors, make it possible to capture streaming data on aspects of patients' physiology, behavior, and symptoms in real time (i.e., rather than only at clinic visits). Although issues of validation, privacy, etc., need to be fully addressed, this may lead to "early warning signals" of changes in clinical state (e.g., worsening depression, suicidality, switch into mania), and hopefully preempting severe exacerbations. As the evidence reviewed above suggests, many reasons exist to be optimistic that science and technology will continue to grow our understanding of the pathogenesis of mood disorders and help us create improved treatments.

Helen S. Mayberg, M.D.

Emory University
2007 Falcone Prizewinner

We no longer debate if depression is a brain disorder—that is a given. But the evolution, viewed over the last 30 years, gives one pause: from mind to chemistry to brain circuits to complex dynamical system, now explored with mind-boggling new tools and all emerging over a relatively short period of time. In addition to the ongoing technical advances that will take neuroscience in important and unimaginable new directions, is the need to leverage available strategies to develop real world solutions that offer precision approaches to treatment of individual patients now, while also providing an adaptable platform to integrate the innovations of the future. We are seeing the emergence of the first precision medicine approaches to the treatment of depression, building on the experience and insights of the cancer and infectious disease communities to develop biomarkers that match individual patients to the treatment that will get them well, while avoiding those that are unlikely to provide benefit. Whether it is the choice of psychotherapy, medication or brain stimulation, understanding the complexity of brain circuits and the influence of genes, developmental insults and ongoing life stress and experience will be required to make fundamental progress on understanding depression risk, pathogenesis and treatment mechanisms, and to have clinically meaningful impact on patients and their families by further preventing relapse and facilitating resilience.

Francis J. McMahon, M.D.

National Institute of Mental Health Intramural Research Program
2016 Colvin Prizewinner

Mood disorder research has made great progress in recent years. Studies once focused on descriptions of the illnesses and their correlates have grown into studies that are beginning to define the causes of mood disorders and the neurobiological pathways through which these causes act. Through genetic studies, we can now, for the first time, begin to understand why mood disorders are inherited, how inherited risk acts over the lifetime of an individual, and how we might intervene to improve outcomes. In the future, we must move toward studies that elucidate the real causes of mood disorders. Once we understand causes, we will be in a much stronger position to seek treatments and cures.

David J. Miklowitz, Ph.D.

UCLA School of Medicine
2011 Colvin Prizewinner

The study of bipolar disorder is, in my opinion, becoming more balanced. Most investigators recognize that genetic, behavioral, and psychological or environmental factors (e.g., childhood adversity) all have a role in illness onset and prognosis. We are increasingly recognizing that optimal treatment regimens include targeted psychotherapy as well as medications. The role of the immune system in bipolar disorder is becoming more evident. The unique changes in neural circuitry associated with bipolar disorder are beginning to emerge from meta-analyses. Progress has been made in the search for genes as well: studies of multi-generational families with bipolar disorder have identified "neuroimaging phenotypes" that are genetically-transmitted candidate traits for the illness. The pharmacological options for bipolar disorder are becoming broader; we are no longer relying only on lithium, valproate or antidepressants. We have clinical decision-making algorithms that include mood stabilizers, antipsychotics, antidepressants and anxiolytics, given in different combinations at different illness phases; and more experimental treatments such as ketamine, transcranial magnetic stimulation, or deep brain stimulation. A big area of progress has been the identification of childhood behavioral antecedents. Although there are still debates about what is and is not a child with bipolar disorder, we agree that many people have their first onset in childhood or adolescence, even though the diagnosis may not be clear until much later. Having a parent with bipolar disorder is a key risk factor. In the near future we may have enough evidence to choose medications for specific patients in part on the basis of their familial responses to these agents. Finally, our lab and other labs continue to test psychosocial interventions that may alter the course of bipolar disorder in children or adults. Psychoeducation–acquainting patients and families with what we know about the illness and its self-management–is now considered to be a key element of good clinical care. Specific therapy approaches (e.g., interpersonal or cognitive-behavioral therapy, family-focused therapy, structured group treatment) are being studied from the perspective of transportability into community care settings. A major objective within the next decade is to develop much clearer guidelines for what forms of therapy works best for what types of patients.

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

University of Miami
1997 Selo Prizewinner

It hardly seems possible that 20 years have passed since NARSAD awarded me the Selo Prize for Outstanding Achievement in Mood Disorders Research. Of the many professional organizations I have been fortunate to be associated with, NARSAD, now BBRF, is one of my absolute favorites. This is not only because of the kind recognition they have provided to my research in the form of the award noted above, a Distinguished Investigator award, and an appointment to the Scientific Council, but the extraordinary and unique role it plays in the development of young investigators in our field. Our small and medium sized grants are virtually a prerequisite to obtain NIH funding in this very competitive era. In terms of mood disorders research, we have made some remarkable strides in further elucidating the neurobiological underpinnings of depression and bipolar disorder with increasing evidence for a preeminent role of inflammation, gene-

environment interactions and epigenetics, to name a few of the recent findings. This has led to clear progress in understanding who in the population is at risk for these devastating disorders. We have been less successful in developing novel treatments for those who do not respond to conventional treatments such as antidepressants, mood stabilizers or electroconvulsive therapy. Clearly we are on the verge of realizing true personalized medicine in psychiatry being able to match individual patients with the most optimal treatment for them. This is truly the Holy Grail for practitioners.

Andrew A. Nierenberg, M.D.

Harvard Medical School
2013 Colvin Prizewinner

The future of therapeutics for mood disorders holds the promise to integrate precision with personalized medicine. Precision medicine will arise from pluripotent cells derived from individuals with mood disorders, along with neuroimaging and other clinically informative biomarkers. Pluripotent cells will provide a transcriptome (after exposure to medications or to-be-developed reagents) which will allow for a molecular diagnosis (similar to a tumor biopsy). Neuroimaging will provide a circuit-based phenotype of dysregulations in functional networks. Other to-be-defined biomarkers will include smart phone-based dynamic real time assessments, genetics, and potential blood-based biomarkers. These multi-dimensional assessments will be combined using a causal dynamic network computational model which will guide short and long term treatment as well as therapeutics for prevention and early intervention. Clinicians will use the information from precision medicine innovations to determine personalized treatment of mood disorders by collaborating with people with mood disorders and their families. They will share decision making about benefits and risks and measure outcomes together iteratively. The outcomes data from precision and personalized treatment of mood disorders will be collected and available for everyone through open source, including patients and their families. Crowdsourcing analyses will lead to new innovations which can be fed back into the system to even further improve outcomes.

A. John Rush, M.D.

National University of Singapore & Duke Medical School
2000 Falcone Prizewinner

Depressive disorders affect a large proportion of patients and account for substantial disability. Recent research, however, has made some substantial advances that fall into four main domains: the types of treatments we offer; how we deliver those treatments; to whom we offer specific treatments, and our understanding of the basic neurobiology underpinning these depressions. There have been remarkable advances in our ability to use brain stimulation methods–especially those that do not entail anesthesia or the induction of seizures to help patients recover from depressive episodes and prevent them from slipping into new episodes. These brain stimulation interventions are becoming widely used to help those for whom medications or therapy have not been sufficiently effective. The rapid reversal of severe depression, and suicidal ideation, has launched efforts to develop acutely active medications that could reverse depression within a day. The psychotherapies have also advanced with evidence of efficacy in mindfulness-based treatments and the development of specific treatments to combat suicidal ideation and risk. We have also learned how to better deliver the treatments that we have, using simple clinically available scales, upon which advances and biomarkers will be placed in the future. The adoption of so-called measurement-based care to tailor the delivery of treatment to individuals–whether medications, psychotherapies, or brain simulation methods–has been shown to increase the effectiveness of medications without increasing their side effect burden. In addition these simple clinical measures increase the detection of individuals earlier in the course of illness and more effectively engage patients as participants in their care. A third major advance is our developing ability to select individual patients for a particular treatment; this should be strongly encouraged or avoided depending on clinical, biological, neuro-functional, genetic and other studies. To illustrate, recent studies suggest that inflammatory processes can be measured in the blood and may suggest the preferential selection or avoidance of particular antidepressant medications. Finally, the widespread evaluation of multiple indicators of brain function–including functional imaging, metabolomics, proteomics, genetics, and tests of brain function–are shedding light on not only etiologically distinct subtypes of depression, but they are also helping to inform treatment selection decisions and to provide important prognostic information so that care delivery can be better tailored to different individuals with depression.

Harold A. Sackeim, Ph.D.

Columbia University
2004 Falcone Prizewinner

Brain stimulation is a rapidly emerging field of medicine, offering a fundamental alternative to pharmacology in the development of neurotherapeutics. Brain stimulation technologies also offer unparalleled opportunities to advance understanding of basic neural mechanisms and the pathophysiology of disease states. The application of these technologies to the treatment of mood disorders has been especially fruitful. Electroconvulsive therapy (ECT), the "grandfather" of neuromodulation technologies, has undergone dramatic improvement that have shed it of also most all its adverse cognitive side effects, while retaining its profound therapeutic properties and placing it on a firm scientific basis. Transcranial Magnetic Stimulation (TMS) and Vagus Nerve Stimulation (VNS) have already made important contributions to therapeutics of treatment-resistant depression (TRD). Similarly, there is marked interest in the use of deep brain stimulation (DBS) and various forms of noninvasive transcranial electrical stimulation (tES) in treatment-resistant depression. I expect the explosive growth of the field of brain stimulation to continue, and one can expect new technologies to expand our capacity to stimulate neural tissue focally and thus directly alter circuit and network function. For example, I believe we will soon have the capacity to release or activate specific molecules on a local basis in the human brain by making them sensitive to brain stimulation interventions. I expect that these advances in biotechnology will result in improved therapeutics for a variety of neuropsychiatric conditions. However, mood disorders have been especially responsive to this class of interventions, and we anticipate substantial progress in coming years as we learn how to best apply existing technologies and explore novel, emerging technologies.

Alan F. Schatzberg, M.D.

Stanford University
2005 Falcone Prizewinner

The development in the late 1980s of widely effective antidepressants such as the selective serotonin reuptake inhibitors (SSRIs) had a major impact on the treatment of patients with major depression. These agents are broadly effective and generally well tolerated; however, many patients do not respond, leaving an ever-growing number of refractory patients. Unfortunately, the numerous, failed investigational antidepressant trials have resulted in several large pharmaceutical companied discontinuing their antidepressant development programs. Still, a few interesting antidepressant strategies have begun to yield promising results. However, several of them do raise potential social issues because they involve agents of potential abuse. For example, ketamine given intermittently has been demonstrated to have transient antidepressant effects and is being developed as an intranasal formulation. The drug can be abused and there are many ketamine abusers in this country and in China. A mu partial agonist, buprenorphine, that is used to treat opiate addiction, has recently been reported at low oral doses to have anti-suicidal properties. This drug is also subject to abuse. To mitigate the risk of abuse, one company is developing a combination of buprenorphine with samidorphan (a mu antagonist) to lessen the risk of tolerance and dependence. The combination has been shown to be effective at low doses of both molecules in two refractory depression studies. Last, the hallucinogen psilocybin has been reported to have potent and enduring antidepressant properties in double-blind studies of cancer patients with pronounced depression and anxiety. Development of these various agents will raise questions regarding the risk-benefit ratio of the specific compound for the patient as well as for our society writ large.

Thomas G. Schulze, M. D.

Medical Center of the University of Munich
2016 Colvin Prizewinner

When I started my career two decades ago, mood disorder genetics was characterized by studies in small samples producing disparate and very often non-replicable results, leading to widespread frustration within the scientific community. The advent of genomics in the first decade of the new millennium and the fact that researchers all over the world have embraced the idea of large-scale collaborative efforts, however, have propelled powerful genome-wide association studies (GWAS). Ever increasing sample sizes, now totaling several tens or hundreds of thousands individuals have yielded a large number of vulnerability loci for bipolar and unipolar depression. Taking into account ongoing studies, we can expect to see close to 100 confirmed GWAS hits for mood disorders within shortly, thus narrowing the gap to recent successes in schizophrenia. These GWAS are being complemented by exome or whole-genome sequencing studies. All these findings will help develop a better understanding of pathways and mechanisms underlying mood disorder. They will help spur developments of novel pharmacological targets and lead to innovative drug repurposing trials. Planning for the next two decades, we should not be complacent, though. We need to leave the comfort zone of studying diagnostic entities. We have to study the biology of (disease) course, as well as outcome and recovery. We have to dramatically increase samples sizes for pharmacogenetics studies. We have to put the other "omics" into the equation. And, we have to extend our research to include populations from all around the world to get a global picture of genetic liability factors. Above all, these endeavors have to adhere to the idea of friendly data sharing, creating a long-lasting bond between clinicians and basic scientists that will eventually deliver on our promise of offering hope and cure to millions.

Eduard Vieta, M.D., Ph.D.

University of Barcelona, Spain
2012 Colvin Prizewinner

If we want to reach the cure for bipolar disorder, I deeply believe that the way to go is to focus on the understanding of how emotions, mood and energy are regulated in the brain. Research on bipolar disorder needs to go in two main directions: one, integrating the advances in the physiology and pathophysiology of brain processes, and two, addressing the unmet needs of people with this condition through strategies of early detection and intervention. I am increasingly convinced that even "early intervention," as it is conceptualized today, is too late, and that most of the morbidity of this condition needs to be addressed long before illness onset. Working through large consortia and looking into the analysis of big data and machine learning for genetics, proteomics, transcriptomics and metabolomics, as well as the connectome, may only be fruitful if it goes in hand with accurate, deep phenotyping, beyond the boundaries of traditional classifications. Innovative mechanisms of action, new treatment targets, and novel methods (including chemical, physical and psychotherapeutic interventions) need to be developed over the next two decades, and only great amounts of private and public funding and industry-government collaborations can make it possible.

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

University of Texas Medical Branch
2012 Colvin Prizewinner

There have been significant advances in the treatment of mood disorders in youth during the past decade. My area of research interest focuses on identification of effective pharmacological treatments for depression and bipolar disorder in children and

adolescents. The next step in treatment research for youth is to identify which treatment is effective for a specific child or adolescent. Neurobiological and psychosocial factors which may contribute to treatment response require further study. Since there is a familial component to mood disorders, it is important to determine whether a child with a mood disorder will have symptom improvement with the same medication that was effective for treating a parent with a mood disorder.

Myrna M. Weissman, Ph.D.
New York State Psychiatric Institute
1994 Selo Prizewinner

I was invited to comment on mood disorders because of winning the Brain and Behavior Selo Award in 1994 for "outstanding achievement in Mood Disorder Research." The award included my late husband Gerald Klerman, M.D. who had died in 1992. The Award in 1994 covered our clinical and epidemiology research showing that depression was a disorder that first begins in youth but reoccurs through the lifespan. We showed that it was more common in women; that rates had increased in cohorts born since World War II; that it was highly familial in biological relatives; and that it bred true. The award also included our development of Interpersonal Psychotherapy (IPT) for depression. In 1994 there were about 8 clinical trials. Our findings, if presented today for the Selo Award, would not win the prize. That is good news. The findings have been replicated, mostly accepted, and the field has now moved onto extending and deepening the direction. In 2017 studies of families at high risk for depression are now incorporating neuroimaging, electrophysiology and genetics to unravel the biological mechanisms underlying depression. Machine learning coupled with Magnetic Resonance Imaging (MRI) has begun to find dimensions of depression that cut across and underneath our conventional diagnostic group. This search for precision in medicine is leading to diagnostic classification based on biomarkers, neural circuits and cognitive processing. The results from a clinical trial, Establishing Moderators and Bio-signatures of Antidepressant Response in Clinical care (EMBARC), designed to systematically explore promising clinical and biological markers of antidepressant treatment outcome, will be ready soon. To maximize genetic understanding of a complex disorder such as depression, scientists worldwide have joined their data in a Genome Wide Association (GWA) analysis. Promising new genetic findings from the GWA should be released this year. Computational science and biomedical engineering are promising new partners for understanding brain processing and translating findings into office-based diagnostic tests or for monitoring clinical outcome and detection of early signs of relapses. Evidence-based psychotherapy developments have not been static. IPT now has nearly 100 clinical trials and a simpler version for health workers for worldwide distribution was launched by the World Health Organization in 2016. There is now a solid base of psychotherapies with evidence from controlled clinical trials. Cognitive Behavioral Therapy (CBT) has had the most clinical trials and even wider dissemination, and recent studies to test the biological mechanism of change. CBT investigators had led efforts to develop and test electronic versions to reduce cost and increase availability. Science builds on strong past discoveries and may deepen or even refute them. NARSAD has been the scaffold for many young scientists who have contributed to these discoveries. A review of the NARSAD Young Investigator 2017 winners will give you a preview of what discoveries to expect in the future. The NARSAD scaffold of research support is a critical piece early in their work. ❒

The Ruane Prize

for Outstanding Achievement in Childhood & Adolescent Psychiatric Research

David Brent, M.D.
University of Pittsburgh School of Medicine
2006 Ruane Prizewinner

The suicide rate in the United States has increased dramatically in the past decade. Four new developments hold promise for improving our ability to predict and prevent suicide. First, data mining of electronic health records using machine learning and natural language processing can be used to identify individuals at risk for suicide, and notify primary care and emergency department clinicians of their patients' suicidal risk. Second, digital phenotyping using passive cell phone data, speech quality, and facial expressions can assess suicidal risk, monitor clinical status, and provide feedback to both patients and clinicians. Third, smartphone applications, triggered by digital phenotyping, can be used to provide timely interventions for suicidal patients. Finally, brief, computerized adaptive tests, which personalize risk assessment by selecting questions from a larger item bank based on patients' answers to initial screening questions, can identify individuals at imminent risk for suicide in primary care, emergency department, or hospital settings. These four approaches can be used to combat the staggering increase in the national suicide rate by extending the reach of evidence-based approaches and getting the right assessments and right interventions to the right people at the right time.

Word cloud provided by F. Xavier Castellanos, M.D.

F. Xavier Castellanos, M.D.
NYU Langone Medical Center, Department of Child and Adolescent Psychiatry
2015 Ruane Prizewinner
(Comment pertains to image above)

Science is a profoundly social process, and so is science advocacy. Confronted with irrefutable evidence that autism spectrum disorder is vastly more common than formerly believed, advocates have focused on increasing the priority of autism research for funders as well as investigators. This word cloud illustrates the most commonly encountered terms in the more than 600 grants currently funded by NIH which include both 'autism' and 'child' as keywords. It encapsulates current research directions, including the awareness that many children with autism also have Attention-Deficit/Hyperactivity Disorder (ADHD); the conviction that brain imaging remains an important means of discovering mechanisms; the importance of focusing on outcomes and risk factors, including environmental exposures. Also highlighted are constructs such as cognitive control, and of course, genomics. While much more research into neurodevelopmental disorders such as autism and ADHD is still needed, the pace of discovery is unquestionably accelerating.

BJ Casey, Ph.D.
Yale University
2015 Ruane Prizewinner

A fundamental issue in psychiatric medicine is the need for empirical evidence indicating when, during development, a treatment, or intervention will be most effective for a patient. Dramatic behavioral and brain changes occur across development, especially during adolescence, when there is a peak in diagnosis of many psychiatric disorders. The most common disorders during this time are anxiety, stress and mood disorders. Current research findings suggest the importance of treating the biological state of the developing versus developed brain by optimizing treatments that match the developmental readiness of the brain.

E. Jane Costello, Ph.D.
Duke University Medical Center
2009 Ruane Prizewinner

The most important progress made in child and adolescent psychiatry in the past decade is the gradual melding of the specialty with adult psychiatry. As we have learned more about the brain, we appreciate the continuity of growth and development, from conception to adulthood and in some cases (e.g., the development of "wisdom") well into middle and old age. Second, treatment methods, whether psychopharmacologic or psychotherapeutic, have no clear age-based cut-off points. Third, the more we have learned about the genetics and epigenetics of mental illness, the greater the importance of avoidable trauma in the development of mental disorders. Fourth, longitudinal, developmental studies are revealing that psychiatric disorders are common: about 80% of youth experience one or more by age 18, and share the equifinality–the same effect or result–from many risk exposures. The same factors, such as poverty, inequality, and poor parental education resulting in malnutrition and obesity, have comorbid adult outcomes with psychiatric disorders, such as diabetes and cardiovascular disease. These advances have, however, been held up by unexpected problems in some areas where a decade ago great progress was predicted, most notably in genomics. Rather than the "Decade of the Brain," we are learning more about how the environment, from the microbiome to the climate, affects human development. Future progress will require us to develop new ways to explore this interface, and to prevent the accumulation of dangerous exposures over the life span.

Rachel G. Klein, Ph.D.
New York University
2004 Ruane Prizewinner

Historically, child psychiatry has been a stepchild of psychiatry. For decades, research focused on adults, with little attention paid to childhood antecedents. This neglect has been dramatically reversed in the past decade, because research has shown conclusively that most serious psychiatric disorders have their origins in childhood. We have come to understand that the study of all aspects of development, especially brain development, will be essential to further our understanding of mental disorders. Such efforts, though relatively recent, have already dismissed the popular notion that childhood mental disorders are arbitrary labels. The validity of childhood psychiatric disorders has been established by research demonstrating their continuity into adulthood, and especially by showing brain abnormalities in some childhood disorders. Moreover, studies have established genetic influences in multiple childhood conditions. Although progress has been significant, much remains unknown. As an example, in spite of establishing genetic transmission, specific mechanisms of gene expression are not yet understood. The future of child psychiatry rests on efforts to study brain development to enable the identification of early dysfunction. This knowledge will inform early treatment and, ultimately, prevention of the disorders as well as their progression into adulthood. Indeed, we now know, based on systematic research, that early intervention fosters better outcomes in schizophrenia, whose onset is usually in mid to late adolescence. Such scientific efforts require the collaboration of multiple disciplines, involving clinicians, neurobiologists, molecular biologists, and others. The promise of neurodevelopmental research for child psychiatry is too important to miss the opportunity to pursue it.

Terrie E. Moffitt, Ph.D. & Avshalom Caspi, Ph.D.
Duke University
2010 Ruane Prizewinners

In both child and adult psychiatry, evidence is building that a person's tendency toward mental disorder can be assessed along a single dimension. A high score on this dimension indicates younger onset of disorder, longer persistence of disorder over time, more comorbid disorders during a lifetime, and greater severity of symptoms. This single dimension is termed "p", because it resembles a dimension already familiar to behavioral scientists and clinicians: the g factor of general intelligence. Just as the g dimension reflects low-to-high intellectual ability, the p dimension represents low-to-high psychopathology severity, with thought disorder at the extreme. The dimension of "p" influences present/absent status on hundreds of psychiatric symptoms, which our diagnostic systems typically aggregate into dozens of diagnoses, which in turn make up the two domains of externalizing versus internalizing disorder, which finally aggregate into one dimension from low to high: "p". Studies show that the higher a person scores on p, the worse that person fares on measures of their family history of psychiatric illness, brain function, childhood developmental history, temperament and personality, polygenic risk scores from genome wide association studies, and life impairment. This single p dimension may help to account for psychiatry's lack of specificity: multiple different diagnoses share the same risk factors, and often respond to the same therapies. Research is needed to test if "p" can predict treatment resistance.

Daniel S. Pine, M.D.
National Institute of Mental Health
2011 Ruane Prizewinner

In many ways, the progress in child psychiatry over t
he past 20 years has been profound, and this progress outlines the future in research in this area. In particular, most mental health conditions are now recognized as disorders of brain development. This recognition reflects progress that has been made through studies in child psychiatry. Moreover, tremendous advances also have occurred in treatment, where many medications and psychotherapies have been shown to substantially impact pediatric mental illnesses. Finally, cascades of risk factors have been identified. These include genetic and environmental risks, which have been shown to shape brain development and associated risks for mental illnesses. In light of these advances, hope for future discoveries is amazingly high. On the other hand, the more that we learn about mental illness and brain development, the more complex we have understood the problems to be in child psychiatry. As a result, progress over the next decade is likely to accrue gradually, as the individual pieces of a complex puzzle are identified and slowly assembled.

Judith L. Rapoport, M.D.
National Institute of Mental Health
2002 Ruane Prizewinner

Our clinical studies of childhood onset schizophrenia, a rare and severe form of the disorder defined as onset before age 13, indicate that these children have high rates of several biological risk factors. 80% have a variety previous neurodevelopmental disorders suggesting an overall "brain vulnerability." Also, 14% of our childhood onset patients have various chromosomal duplications or deletions called Copy Number Variants (CNVs)–higher than the 2-3% rate in later onset disorders. These abnormalities are non-

specific, as they increase the risk for autism, developmental delay as well as schizophrenia. Going forward the actual mechanism of these variants will be important in understanding schizophrenia and other neurodevelopmental disorders.

John L. R. Rubenstein, M.D., Ph.D.
University of California, San Francisco
2016 Ruane Prizewinner

Progress in identifying genes that contribute risk for Autism Spectrum Disorder (ASD) has opened the door to a deep understanding of mechanisms that can cause severe childhood psychiatric disorders. These genetic studies have indicated that ASD can be caused by disruption of several biological processes (gene regulation, synapse development/function, and neural excitability). Fundamental research in these areas will provide insights into how disruption of development and function of the brain contributes to disease risk. Furthermore, these basic studies will facilitate translational investigations into rational therapeutic approaches. Because a clear pathway for discovery is now open, I am optimistic that progress will be made on more precise diagnosis and treatment for ASD. Furthermore, because there is evidence that ASD may share similar genetic mechanisms with other psychiatric disorders, such as schizophrenia, I am hopeful that insights gained from understanding ASD will help researchers and clinicians make progress to more broadly advance understanding psychiatric disorders.

Matthew W. State M.D., Ph.D.
Yale University
2012 Ruane Prizewinner

There has been a recent explosion of progress in the genetics of autism spectrum disorder. These advances have been marked by the discovery of around 70 specific risk genes subject to rare, large effect de novo (new, spontaneous) mutations that disrupt protein synthesis or function. This differentiates ASD from other psychiatric disorders, such as schizophrenia or bipolar disorder, where the lion's share of recent progress has been via the identification of common, small-effect alleles in the non-protein-coding segments of the genome. These findings have led to some distinctive opportunities in ASD and other neurodevelopmental disorders showing similar results. Because the mutations fall directly within genes and carry many-fold increases in risk, there is a relatively direct path to neurobiological studies. And these have quickly begun to identify key biological pathways and spatio-temporal aspects of ASD risk, with multiple laboratories identifying excitatory neurons in mid-fetal prefrontal human cortex as one of likely many--important anatomical regions and developmental epochs. Over the next several years, reliable gene discovery will surely continue, as there is strong evidence of many more risk genes in the genome. Routine sequencing of the entire human genome (whole genome sequencing) will help with these efforts and provide additional biological insights. Finally, developmental, systems and computational neurobiological studies are destined to offer new and important insights into both the genetic and environmental contributors to ASD, leveraging a growing set of definitive molecular clues provided at last by successful genomic studies.

Eric Andrew Taylor, M.D.
King's College London Institute of Psychiatry, Psychology and Neuroscience
2008 Ruane Prizewinner

In the 50 years that I've been involved with the science and practice of mental health there have been revolutionary changes for the better. The large, old, depriving institutions have mostly gone. Effective medicines and psychotherapies have arrived. We have realized that most severe mental illnesses start young and are rooted in brain dysfunctions of various kinds. There is much still to do, and the future should bring translation of hard-won scientific knowledge into better clinical tools. Longitudinal studies will have identified the factors making for better or worse outcomes. Neuroimaging, neuropsychology and machine learning applications will all have advanced to the level of characterizing individual cases. Diagnosis can then become more precise and describe the individual's profile of dysfunctions rather than a heterogeneous syndrome. Treatment and the monitoring of treatment will become more personal, useful at an earlier stage of disorder, and correspondingly more effective. In another 50 years we could be looking back at another revolution.

Anita Thapar, M.D., Ph.D.
Cardiff University School of Medicine
2014 Ruane Prizewinner

Research findings have been so important for ADHD–a disorder that can be misunderstood by some. Huge international efforts mean we now know there is a strong genetic contribution; specific genes that are involved are also being identified. It now is clear that there is strong biological and clinical overlap with other brain disorders that first show in childhood, such as autistic spectrum disorder, communication and learning difficulties. This group is now called the childhood neurodevelopmental disorders. Future research and clinics in some countries are beginning to investigate and assess these conditions together. Research also has highlighted the plight of adults; for many, ADHD remains a problem beyond childhood. As a result, new methods of assessment and diagnoses that are more age appropriate are beginning to emerge. Investigations of the entire population are also proving exciting. These are telling us that ADHD behaves like a spectrum. Sophisticated new methods are being used to identify environmental as well as genetic causes. Future discoveries will be crucial for informing prevention and early intervention programs. ❐

The Goldman-Rakic Prize

for Outstanding Achievement in Cognitive Neuroscience

Amy F. T. Arnsten, Ph.D.
Yale University
2015 Goldman-Rakic Prizewinner

A major goal for understanding the neurobiology of mental illness will be the bridging of cognitive neuroscience (including findings from neuropathological and neuroimaging studies in patients) and cellular and molecular neuroscience, so that we can understand how a wide variety of genetic insults can lead to a shared phenotype. As many mental disorders target newly evolved brain circuits, we must respect that these neurons are often uniquely regulated at the molecular level. We have been making progress in many arenas, for example, seeing how atrophy of the deep layer III microcircuits in dorsolateral prefrontal cortex leads to symptoms of thought disorder, and how molecular insults to these circuits render them vulnerable to dendritic spine loss. It is hoped that insights from neuropathological studies in patients can provide "top-down" guidance for basic research, and that basic research can in turn illuminate how genetic insults lead to circuit dysfunction. In this way we can keep on course to better learn how to protect brain networks, and develop treatments to prevent or slow the course of mental disorders.

Karl Deisseroth, Ph.D.
Stanford University
2015 Goldman-Rakic Prizewinner

The development between 2004 and 2009 of optogenetics (controlling specific neural elements during behavior using microbial opsin genes, fiberoptics, and cell targeting tools) was supported in its early stages by my NARSAD Young Investigator Award, and has helped thousands of investigators around the world advance our shared understanding the circuit underpinnings of adaptive and maladaptive behavior. In one example (depression), specific cellular connections–spanning the entire adult mammalian brain–have been identified that are causally involved in precise control of anhedonia- and hopelessness-related behaviors. More broadly, exploration is now possible of a virtually limitless range of ideas and hypotheses regarding the causal and global circuit dynamics of both normal behavior and psychiatric disease mechanisms, including also states related to anxiety, addiction, and altered social behavior. Though my first steps that led to these advances were supported by NARSAD, these were not part of a traditional disease-related research program. Rather, interdisciplinary basic-science collaboration has been the hallmark of these efforts, characterized by joint efforts among physicians, biologists, physicists, materials scientists, biochemists, chemists, and chemical engineers. We and others have discussed how to balance funding of early versus late-stage research, and we have suggested that scientists must communicate to the broader public that any specific goal of a research portfolio–be it disease treatment or national interest–is best served with a major basic research component where direct links between research and goal are not known, or even knowable. Looking to the future, this approach (which NARSAD/BBRF has pioneered and exemplified) will continue to deepen our understanding of psychiatric disease symptoms and, more broadly, the sensations, cognitions, emotions, memories, and actions that contribute to the common experience of humanity.

Joaquin M. Fuster, M.D., Ph.D.
University of California, Los Angeles
2006 Goldman-Rakic Prizewinner

Cognitive neuroscience is the neuroscience of the human mind. It is the science that explores the brain mechanisms of the five basic cognitive functions: attention, perception, memory, language, and intelligence. These functions work with the cognitive networks of the cerebral cortex ("cognits") and their relations with subcortical centers of the brain. The essential elements of those networks or cognits consist of widely distributed neurons and the fiber connections within and between them. The points of contact of those connections are called synapses, which are little switches that by chemical and electrical changes make cognitive functions and their networks work or shut off–as in sleep or inattention. Synapses are modifiable (plastic) by learning and memory. They grow and their power of transmission is enhanced by education and life experience. In aging and degenerative diseases of the brain, like Alzheimer and Parkinson, they deteriorate, along with the neurons and nerve fibers that connect them. With a large variety of methods (neuroimaging, neurochemistry, neuropharmacology, neurophysiology, nutrition science and genetics), modern cognitive neuroscience investigates the basic and translational aspects of cognitive function. The principal objectives are two: (a) to promote healthy development of the brain; and (b) to prevent and heal the ravages of aging, psychosis and dementia. Progress in their pursuit is costly in money and human resources.

Michael E. Goldberg, M.D.
Columbia University
2011 Goldman Rakic Prizewinner

We know little about the mechanisms underlying human behavior. Modern cognitive neuroscience views the brain as a network for turning perception into action, to facilitate earning reward. Some of the greatest insight into this process comes from studying the activity of individual brain cells while monkeys perform difficult cognitive tasks. For example we know a lot about the cortical and subcortical networks involved in visual attention, and the generation of the eye movements that humans make to facilitate attention. We are beginning to understand how the brain labels things in the environment that will cause pleasure and things that will cause pain. What we do not understand are the mechanisms that drive human choice and motivation. Why do people make bad choices, such as an addict who has gone through rehabilitation who chooses to become addicted again? What are the derangements of brain networks that cause the anhedonia of severe depression so that nothing gives pleasure? What are the genetic and neurochemical factors that cause humans to act in self-destructive ways? What is the network change that causes cognitive distortion? The promise of cognitive neuroscience is that understanding the processing in the normal brain will enable us to understand what goes wrong in the psychologically damaged brain, and help us to heal it.

Robert C. Malenka, M.D., Ph.D.
Stanford University
2010 Goldman-Rakic Prizewinner

Major advances in methodologies that allow scientists and physicians to interrogate and manipulate neural circuit activity in awake behaving animals and humans hold the promise to revolutionize our understanding of the pathological brain mechanisms that mediate many of the most prominent symptoms of major mental illnesses. Leveraging advances from human genetics, we are now able to generate animal models with the same genetics as human patients and using these models, define and even repair the circuit dysfunctions mediating the pathological behaviors at the core of many mental illnesses. The new insights generated by this basic research will guide human brain imaging studies, the results of which will be used to stratify patients based on a combination of their symptoms and brain activity fingerprints to ensure that they receive the optimal treatments for their specific condition. These new insights will also guide efforts using novel platforms to develop new medications that improve symptoms via currently unknown, novel mechanisms of action as well as guide direct brain interventions to modify dysfunctional circuits using non-invasive techniques such as transmagentic stimulation or targeted ultrasound stimulation. With the amazing advances that have occurred over the last decade and with many more on the horizon, I am confident that we are on the cusp of a revolution in how we diagnose and treat mental illness. Within 20 years we will have vastly improved diagnostic capabilities and completely novel treatments that we can administer clinically in much more effective and sophisticated ways. Although challenging, the future is bright for those of us who care about reducing and treating brain and behavior illnesses.

Bruce S. McEwen, Ph.D.
The Rockefeller University
2005 Goldman-Rakic Prizewinner

Our discovery in 1968 of cortisol receptors in the hippocampus provided a gateway into later discoveries, throughout the brain, of receptors and actions of stress, sex as well as metabolic hormones upon cognitive function, self regulatory behavior, mood and many other aspects of brain function. This has not only broadened the definition of "neuroendocrinology" to include the continuous, reciprocal communication between the brain and the body via hormonal and neural pathways but it has also contributed translationally to neurological and psychiatric investigations showing plasticity and vulnerability of the human brain. The brain is the central organ of stress and adaptation to stress because it perceives and determines what is threatening, as well as the behavioral and physiological responses to the stressor. The adult and developing brain possess remarkable structural and functional plasticity in response to stress, including neuronal replacement, dendritic remodeling, and synapse turnover. Neurotransmitters, neuromodulators, neuroendocrine, autonomic, immune and metabolic mediators are essential for epigenetic regulation of brain and body adaptation via the active process of allostasis. However, chronic stress causes an imbalance of neural circuitry subserving cognition, decision-making, anxiety and mood that can alter expression of those behaviors and behavioral states. This imbalance, in turn, affects systemic physiology via the same mediators and leads to increased allostatic load and overload. Thus, in the short term, as for increased fearful vigilance and anxiety in a threatening environment, these changes may be adaptive. But, if the danger passes and the behavioral state "gets stuck" along with the changes in neural circuitry, such maladaptation may need intervention with a combination of pharmacological and behavioral therapies, as is the case for chronic anxiety and depression. The entire brain also has receptors for sex hormones that influence many functions, along with developmentally programmed sex differences, and there are, as a result, important sex differences in brain function that are now being explored. Moreover, the life course has taken on new meaning as a determinant of trajectories of life-long health; and adverse early-life experiences, interacting with genotype, produce lasting epigenetic effects on brain and body over the lifespan, among which included increased risk for depression, diabetes, substance abuse and other disorders. While prevention is most important, the plasticity of the brain gives hope for therapies that take into consideration brain–body interactions.

Earl K. Miller, Ph.D.
Massachusetts Institute of Technology
2016 Goldman-Rakic Prizewinner

I see cognitive neuroscience becoming increasingly integrative both within and across levels of inquiry. When I began my career, the focus was on the brain's individual parts (individual neurons, brain areas, etc.). It was as if the brain was a clock and if we could figure out each "gear," we would figure out the whole. But we have seen increasing awareness that any understanding of brain function is going to depend on a network understanding, i.e., how the parts work together. For one thing, it is becoming clear that the brain's individual neurons and areas do not have single functions, that their signals only make sense in the context of what other neurons and areas are doing. This has necessitated the rise of computational approaches and theory needed to describe and understand network interactions. Finally, we are seeing innovation of techniques and approaches that cut across different levels. Molecular tools are providing greater insight into networks' properties by allowing us to perturb networks in precise ways. In short, I see different strands of neuroscience that have traditionally been separate beginning to weave together. This is what happens when any field of science matures.

Eric J. Nestler, M.D., Ph.D.
Mount Sinai School of Medicine
2008 Goldman-Rakic Prizewinner

It was a tremendous honor for me to receive the Patricia S. Goldman-Rakic Award from BBRF. It was particularly meaningful since Dr. Goldman-Rakic was a close colleague of mine at Yale for many years. The key goals of my laboratory's research are to understand how drugs of abuse or stress change the brain in lasting ways to induce addiction- or depression-related behavioral abnormalities in animal models and to use that information to develop improved

treatments for these conditions. Work in my laboratory, and in many others, over the past couple of decades has identified numerous molecular and cellular adaptations induced in brain in response to chronic exposure to a drug of abuse or stress, with an increasing number being related causally to behavioral symptoms in animal models. A major goal of current research is to move beyond studies of single adaptations to understand how a myriad of molecular changes summate to underlie specific changes in neural and synaptic function in a given brain region. Likewise, it will be important in turn to understand how these neural and synaptic changes summate to alter the functioning of the brain's circuitry to mediate specific behavioral abnormalities that define an addicted or depressed state. This delineation of molecular, cellular, and circuit mechanisms of addiction and depression will require increased attention to the specific cell types (both neuronal and non-neuronal) where the drug- and stress-induced adaptations occur and to the specific microcircuits within brain pathways affected by those adaptations. Finally, we must do a far better job of translating our increasing knowledge of the neurobiological basis of drug addiction and depression to the clinic. We have arguably not made appreciable improvements in addiction and depression treatments over the past several decades. This is due mostly to the unique complexity of the brain—which goes far beyond that of all other organ systems. However, it also is due to the field's dramatically reduced ability to readily study the effects of drugs with novel mechanisms in humans. The hope is that the transformational advances in our ability to study molecular, cellular, and circuit mechanisms in the brain, together with a renewed investment in experimental human pharmacology, will lead to the fundamental improvements in therapeutics that are so sorely needed.

Michael Posner, Ph.D.
University of Oregon
2004 Goldman-Rakic Prizewinner

During the past decade research in cognitive neuroscience has provided an important foundation for future advances in understanding and treating mental illness. The development of resting state MRI and of connectomics has allowed the possibility of tracing the development of human brain networks from birth through the life span. This provides a foundation for understanding atypical development in childhood, the risk taking and disorders of adolescents, and the loss of function in old age within a single framework of brain development. This research is fostering new methods of treatment while at the same time improving our understanding of existing treatments. Distinguishing between changes in the depressed brain arising from drug treatments and those resulting from cognitive behavioral therapy marks an important advance in our ability to choose among or combine treatment modalities. Understanding the mechanisms of self-regulation may allow treatments for substance abuse that do not rely on the intention of the addicted person. An increase in our knowledge of how genetic and epigenetic mechanisms relate to brain networks paves the way for possible individualized therapies. These dramatic basic research findings have already influenced patient care and the future should see a dramatic increase in these efforts.

Larry R. Squire, Ph.D.
University of California, San Diego
2012 Goldman-Rakic Prizewinner

This is a great time for cognitive neuroscience and particularly for study of those features of cognition that are relevant to mental illness: attention, memory, planning, decision making, and the organization of action. We are learning about these functions–how they operate and sometimes fail–in ever increasing detail. Studies in humans help identify the components of these functions and the underlying brain systems. Studying in monkeys can illuminate how these functions operate. And these problems are now being brought to rats and mice with increasing success, where it has become possible using extraordinary new techniques to study the cellular and molecular events, and the circuitry, that support many cognitive functions. Basic science will play a vital role in the development of better diagnosis, prevention, and treatment of mental illness. It is sometimes said that we want to fix the car but we need to know how the car works. ❐

The Maltz Prize

for Innovative and Promising Schizophrenia Research

(formerly known as the **Baer Prize**)

Stephen J. Glatt, Ph.D.
State University of New York, Upstate Medical University
2010 Baer Prizewinner

We have long known that the risk for mental illness runs in families and, at least in part, is attributable to genes. With support from the Brain and Behavior Research Foundation, we have made in the last decade undeniable progress in identifying some of the risk genes for schizophrenia, bipolar disorder, major depressive disorder, and other major mental disorders. Continued collaborative science is key to assembling samples large enough to have confidence in the identification of additional genes, but eventually this effort will "max out." Once the majority of genetic risk factors have been identified, we will be in a unique position to identify and study resilience factors, as many individuals who carry a large number of mental illness risk genes do not develop any disorder. These individuals should be studied in depth so we can identify the protective genes and environments from which they have benefitted, and to determine ways in which the biological pathways that promote resilience can be fostered in at-risk individuals, and in the population at large.

Jeremy Hall, M.D., Ph.D.
Professor of Psychiatry and Neuroscience
Director, Neuroscience and Mental Health Research Institute
2007 Baer Prizewinner

Success in genomic discovery is now transforming our understanding of the causes of severe psychiatric disorders such as schizophrenia. Advances in neuroscience and stem cell biology are also increasingly providing the tools to translate these genetic discoveries into mechanistic and therapeutic insights. The challenge we face now is to use these approaches in an integrated and recursive fashion to translate genetic discovery into meaningful impacts on the treatment and lives of patients. This will require the development of programs of research that our focussed on delivering clinical outputs and integrating with drug discovery and the pharmaceutical industry. Our own work at the Cardiff Neuroscience and Mental Health Research Institute focusses on key pathways of risk for schizophrenia identified from genetic studies (e.g., synaptic proteins; neuro-immune targets) and on developing a translational platform linking genetic, cellular, animal and human studies against which to prosecute future drug screening and development. Overall we aim contribute to the global efforts at develop genuinely new therapeutic approaches for this disabling disorder.

William P. Horan, Ph.D.
University of California, Los Angeles
2016 Maltz Prizewinner

I am optimistic that new treatments that help people with psychosis achieve more productive and personally meaningful lives in the community will be available in the next decade. We already have a good understanding of some of the key factors that hold people back from achieving their goals in areas such as relationships, independent living, and work/school. Social cognition has emerged as one of the most important factors. Social cognition refers to the diverse mental operations underling social interactions such as perceiving, interpreting, and managing responses to the behaviors of other people. Findings from many research groups in the United States and abroad strongly support targeted psychosocial interventions as a way to improve social cognitive skills. Further, there is emerging evidence that these interventions can impact the brain systems involved in social cognition. A new wave of studies is now evaluating creative approaches to enhance skill acquisition in these interventions, such as combining them with complementary pharmacological treatments and new technologies. These studies are also developing new ways to translate newly acquired social cognitive skills into meaningful improvements in daily life functioning. Social cognitive treatments will likely be important new tools to help people with psychosis maximize their functional recovery and personal fulfillment.

M. Camille Hoffman, M.D.
University of Colorado
2015 Baer Prizewinner

As an obstetrician and maternal-fetal medicine subspecialist, I am hopeful that the field of schizophrenia research is moving towards prevention. A hypothesis called the developmental origins of health and disease, or DoHaD, has a robust body of data to support the impact of genetics, epigenetics, and environmental influences on fetal life with respect to the downstream development of chronic diseases of adulthood, including psychiatric conditions. We must intercept adverse brain development very early- during gestation-to prevent a number of conditions including some mental illnesses. Our research team has an ongoing double-blind randomized trial assessing the impact of maternal choline supplementation (in the form of phosphatidylcholine) during pregnancy on early child neurodevelopment. Based on our pilot data, we anticipate improved child outcomes including behavior, attention, and social interaction in the children whose mothers received choline while they were developing in the womb versus children whose mothers received a placebo. These are early markers of schizophrenia. If this supplement, which is safe during pregnancy and easy to take, is preventive, we can significantly reduce the human suffering related to schizophrenia and other severe mental illnesses.

Amanda J. Law, Ph.D.
University of Colorado School of Medicine
2011 Baer Prizewinner

2017 marks the 30th anniversary of the neurodevelopmental hypothesis of schizophrenia. The original theory, offered independently in the papers of Weinberger (1987) and Murray and Lewis (1987), proposed that a prenatal/early developmental event could disrupt normal growth and maturation of the brain, leading to manifestation of schizophrenia in later life. Remarkably, even 30 years ago, it was recognized that genetic and in-utero environmental factors (such as prenatal infection and obstetric complications) may contribute to later risk. Despite the wealth of epidemiological evidence to support the hypothesis, still, little is known about the mechanistic basis of how these factors impact early brain development. Today, we are in an unprecedented era of psychiatric genetics research and the knowledge generated is expected to transform our biological understanding of the disorder. Although most efforts to date have focused on identifying genes associated with risk, a critical next step is translating these findings into molecular and neurobiological mechanisms and understanding how they interact with early developmental adversity. For more than a decade, our research has focused on understanding the neurobiological role of genes associated with schizophrenia, incorporating developmental studies in humans and mice. Looking forward, we propose that the next generation of animal studies in schizophrenia will integrate gene models with in-utero models of environmental risk. Such studies have the potential to provide novel insight into the early neurodevelopmental origins of schizophrenia, the in-utero mechanisms involved, and allow for the identification of prevention or intervention strategies.

Amanda McCleery, Ph.D.
University of California, Los Angeles
2016 Maltz Prizewinner

Over the last two decades there has been a dramatic shift in the conceptualization of schizophrenia. There is now a growing recognition that cognitive impairments are a core feature of the illness. Cognition, which includes domains such as attention, learning, and memory, is highly predictive of community functioning in schizophrenia, and hence, it is a prime target for intervention. The overarching aim of my research has been to identify and understand cognitive predictors of community functioning in schizophrenia and related conditions. My work is informed by developmental psychopathology, and I aim to gain a nuanced understanding of the trajectory of cognition over the course of illness in order to identify critical periods, key targets for intervention, and mechanisms of change. With the support of funding from the Brain & Behavior Research Foundation, I have expanded my research program to include novel electrophysiological methods to probe the processes that underlie impaired cognition in schizophrenia, including neuroplasticity. Through this line of research, I aim to increase our understanding of the bases of cognitive impairment in schizophrenia, and to guide development of treatment strategies to remediate impaired cognition. I am grateful for the continued support of the Brain & Behavior Research Foundation. The Foundation provides critical funding for pioneering research that aims to elucidate disease pathophysiology, reduce disability, and improve outcomes for people with mental illness.

Barnaby Nelson, Ph.D.
Orygen & University of Melbourne
2015 Baer Prizewinner

Although progress has certainly been made in symptom management in schizophrenia, the underlying causes of the disorder remain elusive. In my view, progress on this front requires a resuscitation of detailed study of psychopathology, a return to understanding core phenomenological features of the disorder (i.e., characteristic disturbances of subjective experience in schizophrenia beyond "symptom counting") and improved integration of the different domains of research (e.g., the phenomenological, neurocognitive, neurobiological domains). Apart from the obvious benefit of improved understanding of the disorder, these efforts will guide research into pathogenetic mechanisms, lead to increased ability to identify patients at highest risk of the disorder, and improve early intervention and targeted therapies.

Stephen Ripke, M.D.
Psychiatric Genomics Consortium
2014 Baer Prizewinner

To date, genome-wide association studies (GWAS) have identified more than 100 schizophrenia-associated loci in the genome and have led to novel insights into the biology of this common psychiatric disorder. Many of the identified genes fall into functional categories of synaptic function and plasticity, glutamatergic neurotransmission, neuronal calcium signaling, neurodevelopment and immune processes. Since the early days of my medical training I have had an interest in pursuing computational methods in genetic research. The combination of strong computational and statistical background with medical/clinical training allows me to execute all necessary steps from receiving raw genotypic data up to drawing medical/clinical conclusions from the results. Having lead many analyses of the Psychiatric Genomics Consortium in recent years–the largest collaborative experiment in psychiatric genetics to date–I have developed a strong understanding of what successful studies in the future would look like. With larger sample sizes and novel methodological approaches, we are well positioned to continue our strong record of discovery.

Lorna W. Role, Ph.D.
Stony Brook University
2009 Baer Prizewinner

I was lucky enough to be brought into the NARSAD/BBRF "family" by one of the Foundation's early forays into trying to attract people into the field who were *not* working on schizophrenia or other neuropsychiatric diseases. It has been a transformative experience for me, taking my research from (very) basic science studies of mechanisms of synaptic transmission to (now 12 years) of effort that is much more translationally directed. This has been a tremendously positive expansion of our research efforts and interests, setting new goals to our studies of how genes implicated as susceptibility factors in schizophrenia might influence circuit excitability. As such, I think an important direction for innovation in the field is to continue to encourage radically new perspectives and fusion of different disciplines. Collaborative interactions optimize the power of diversity in thinking and approaches. In my view, neuropsychiatric therapies of the future can only benefit from the convergence of perspectives as apparently disparate as computational neuroscience, genetics and biomedical engineering.

Daniel Wolf, M.D., Ph.D.
University of Pennsylvania
2009 Baer Prizewinner

Biological research in psychiatry and in schizophrenia in particular is benefiting from a new focus on unpacking the heterogeneity within diagnostic categories by relating specific symptom dimensions to particular cognitive-emotional processes and their underlying brain circuitry. My own work, supported by BBRF and the Baer Award, along with work by many others in the field, has helped identify reduced function in brain reward circuitry as a neural correlate of motivation deficits in schizophrenia. These motivation deficits, along with other "negative symptoms," cause much of the disability in schizophrenia, and unfortunately remain resistant to current treatments. However, hope lies ahead. The identification of specific brain circuit abnormalities is providing new biomarkers and targets for treatment development, and together with our rapidly expanding understanding of basic neuroscience and human genetics, will lead to transformative new therapies. As the field moves increasingly toward understanding the earliest stages in the development of schizophrenia, where dysfunction in brain motivation circuitry may provide one early signal of risk, we will ultimately achieve the ability to prevent schizophrenia and other illnesses from arising in the first place. In the meantime, there is an enormous amount of work that needs to be done to get there. ❐

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Robert Wayne Emerson, Ph.D.
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Christina Marie Gremel, Ph.D.
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Indiana University School of Medicine, IN
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Institute of Living/Hartford Hospital, CT
Institute of Neurology/University College London, United Kingdom, England
Institute of Neuroscience, Spanish Research Council, Spain
Institute of Psychiatry/King's College London, United Kingdom, England
Institute Pre Mata (IISPV -HPU), Spain
Institut National de la Santé et de la Recherche Médicale, Marseille, France
Institut National de Recherche en Agronomie, France
Instituto de Biomedicina de Valencia (IBV-CSIC), Spain
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National Institute of Mental Health (NIMH/NIH), MD
National Institute of Mental Health and Neurosciences, Bangalore, India
National Institute of Neurological Disorders and Stroke (NINDS/NIH), MD
National Institute on Aging (NIA/NIH), MD
National Institute on Alcohol Abuse & Alcoholism
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National Institute on Drug Abuse (NIDA/NIH), MD
National Institutes of Health (NIH), MD
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National Research Council, Institute of Neuroscience, Italy
National Scientific and Technical Research Council of Argentina (CONICET), Argentina
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