

# Alzheimer's Disease Research Current Grantees

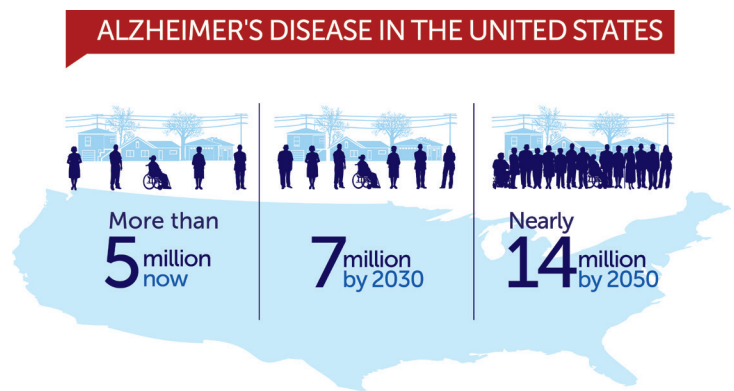
Alzheimer's disease (AD) causes irreversible damage to the brain, disrupting memory, cognition, personality, and other functions. At the very final stages, AD leads to complete brain failure, which is fatal. AD is the sixth leading cause of death in the United States. For people already suffering from health difficulties and other problems, it makes their condition much more challenging.

More than five million Americans ages 65 and older are thought to have Alzheimer's disease. By 2050, that figure is expected to triple, to nearly 14 million.

With the support of our donors, Alzheimer's Disease Research (ADR), which began in 1985, has awarded more than \$100 million to fund

research projects on the causes and potential prevention and treatment of this disease. In the past three years alone, ADR has funded 92 research projects totaling \$17.9 million. The standard ADR grant is \$300,000 over three years. We also have ADR postdoctoral fellowship grants (\$100,000 over two years), which are intended for young researchers in their final stages of mentored training.

Currently ADR is supporting 85 research projects looking into every aspect of Alzheimer's prevention, treatment, and cure. By increasing our understanding of what causes AD, how it spreads, and hastening discovery of disease-modifying drugs and therapies, ADR grantees are covering the spectrum of possibilities and bringing new hope to individuals and families who suffer from this disease. Our wide-reaching portfolio of active ADR grant projects are described on the pages following.



## Discovering New Drugs to Halt Alzheimer's

It's unlikely that a single drug, or "magic bullet," will be found to stop Alzheimer's disease. However, experts do expect that someday there will be a "cocktail" of drugs that will modify and slow the disease's course. ADR grantees are using rapid, high-tech methods to find molecules capable of stopping or slowing Alzheimer's changes, and these discoveries will be used to develop new drugs. Also, the search continues for opportunities to use already-approved drugs in new ways to arrest Alzheimer's (ie, "drug repurposing").



### Steven Estus, PhD (7/1/14 – 6/30/17)

University of Kentucky Research Foundation

*Genetics Pinpoint a Mechanism by Which a Leukemia Drug May Reduce Alzheimer's Risk*

*"Our short-term goal is to understand how a hereditary difference in a gene called CD33 acts to reduce the risk of Alzheimer's disease. Our longer-term goal is to translate this information into a drug that mimics this protective effect, and thereby reduces Alzheimer's risk."*

[www.brightfocus.org/grant/A2014210S](http://www.brightfocus.org/grant/A2014210S)



### Mark Henkemeyer, PhD (7/1/16 – 6/30/19)

University of Texas Southwestern Medical Center

*Identification of Novel Compounds to Promote Synapse Health and Prevent Alzheimer's Disease*

*"I aim to discover a new class of medicines that will halt the destruction of synapses and avert memory loss with direct implication for the prevention of Alzheimer's disease."*

[www.brightfocus.org/grant/A2016345S](http://www.brightfocus.org/grant/A2016345S)



### Tsuneya Ikezu, MD, PhD (7/1/16 – 6/30/19)

Boston University

*Validation of Drug Candidates for Enhancing the Phagocytic Clearance in the Alzheimer's Brain*

*"We propose to identify a drug, which can enhance clearance of unwanted protein buildup, consisting of degraded nerve fibers (called fibrils) and dead cells, in the Alzheimer's brain."*

[www.brightfocus.org/grant/A2016551S](http://www.brightfocus.org/grant/A2016551S)



**Patrick Kehoe, PhD** (7/1/16 – 6/30/19)

University of Bristol

*Helping the Brain to Fight Back Against Alzheimer's Disease—  
Using Old Drugs for New Purposes*

*"Our project will examine the therapeutic potential of a drug that enhances the function of a biochemical pathway that reduces high blood pressure. The same drug has additional properties that may alleviate some other destructive processes that occur in brain cells in Alzheimer's disease."*

[www.brightfocus.org/grant/A2016582S](http://www.brightfocus.org/grant/A2016582S)



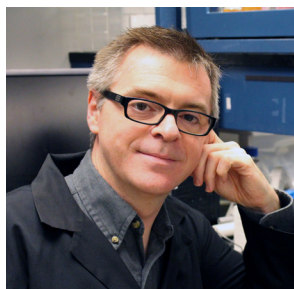
**Daniel Lee, PhD** (7/1/15 – 6/30/18)

University of South Florida

*Gene Therapy with Arginine Decarboxylase and the Regulation of Tau*

*"The goal is to identify a viable target that could be drugged or induced to provide maximum benefit with respect to tauopathies and Alzheimer's disease-like pathology, but minimal changes in a host of other body systems."*

[www.brightfocus.org/grant/A2015504S](http://www.brightfocus.org/grant/A2015504S)



**M. Paul Murphy, MA, PhD** (7/1/14 – 6/30/17)

University of Kentucky

*A Novel Therapy for Alzheimer's-Associated Dementia with Cerebrovascular Comorbidity*

*"This proposal holds potential for making progress towards a new therapeutic option for Alzheimer's disease with significant cerebrovascular comorbidity, a neurologic disorder that is both poorly understood and has only limited treatment options."*

[www.brightfocus.org/grant/A2014280S](http://www.brightfocus.org/grant/A2014280S)



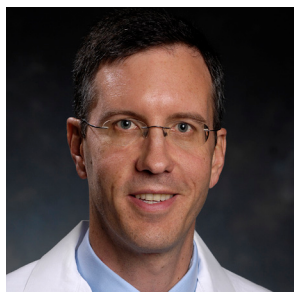
**Dianne Perez, PhD** (7/1/16 – 6/30/19)

The Cleveland Clinic Foundation

*Novel Drugs against a New Receptor Target to Treat Alzheimer's*

*"This project is important in order to validate the target, then to actually make new drugs that are the most selective for this target, and then to test these drugs in an animal model of the disease, all of which paves the way for human drug development."*

[www.brightfocus.org/grant/A2016272S](http://www.brightfocus.org/grant/A2016272S)



**Erik Roberson, MD, PhD** (7/1/15 – 6/30/18)

University of Alabama at Birmingham

*A New Approach to Targeting Tau in Alzheimer's Disease by Inhibiting Its Interaction With Fyn*

*"We are investigating compounds that would stop the interaction between two proteins, tau and Fyn. Considerable data indicates that blocking this interaction could ameliorate Alzheimer's."*

[www.brightfocus.org/grant/A2015693S](http://www.brightfocus.org/grant/A2015693S)

## Promising Treatment Approaches

Already, the painstaking work to uncover Alzheimer's pathology has paid off with a bounty of pathways to potentially treat the disease. With a disease as complex as this one, it's very good news that there are numerous intervention points where it may be possible to develop treatments to slow or halt Alzheimer's progress. Some of these involve molecular "signaling pathways" where grantees are hoping to intercept a signal, or gene transcription, to prevent a bad outcome. In other cases, they're seeking to arrest a molecular interaction to prevent a "bad player" from forming, or to remove toxic debris.



**Katrin Andreasson, MD** (7/1/14 – 6/30/17)

Stanford University

*Preventing and Treating Alzheimer's Disease by Inhibiting Tryptophan Metabolism*

*"The proposed work will determine whether degradation of the amino acid tryptophan by the enzymes TDO2 and IDO1 plays a role in the development of Alzheimer's disease."*

[www.brightfocus.org/grant/A2014423S](http://www.brightfocus.org/grant/A2014423S)



**Joachim Herz, MD** (7/1/16 – 6/30/19)

University of Texas Southwestern Medical Center

*Targeting the Molecular Cause of the ApoE4 Risk in Alzheimer's Disease*

*"Successful completion of the project would establish a novel therapeutic approach to Alzheimer's that has the potential to abolish the ApoE4 risk for developing the most frequent late-onset form of the disease."*

[www.brightfocus.org/grant/A2016396S](http://www.brightfocus.org/grant/A2016396S)



**Gail Johnson, PhD** (7/1/14 – 6/30/17)

University of Rochester Medical Center

*Stimulating Nerve Cells to Dispose of Unwanted Tau Protein*

*"Our study will look at how normal brain cells get rid of excess tau protein, and how we might be able to "restart" that vital function during Alzheimer's disease."*

[www.brightfocus.org/grant/A20140185](http://www.brightfocus.org/grant/A20140185)



**Brian Kraemer, PhD** (7/1/14 – 6/30/17)

VA Puget Sound Health Care System

*Deconstructing the Dopamine/Abnormal Tau Relationship in AD*

*"The mission of my research program is to understand the molecular mechanisms at work in age-related cognitive decline caused by neurodegeneration."*

[www.brightfocus.org/grant/A20144385](http://www.brightfocus.org/grant/A20144385)



**Bruce Lamb, PhD** (7/1/15 – 6/30/18)

Cleveland Clinic Lerner Research Institute

*The Role of TREM2, A Key Immune Regulating Protein, in AD*

*"We are working to find new drug targets in the brain and to understand the biology of the newly identified immune molecule, triggering receptor expressed on myeloid cells 2, or TREM2, and its role in the Alzheimer's disease tau pathology."*

[www.brightfocus.org/grant/A20152965](http://www.brightfocus.org/grant/A20152965)



**Yona Levites, PhD** (7/1/14 – 6/30/17)

University of Florida

*Targeting Tau with Immunotherapy in a Mouse Model of Alzheimer's Disease: A Comparison of Approaches*

*"We propose to compare the ability of genetically engineered antibodies targeted to the intracellular or extracellular levels to fight tau pathology in two mouse models."*

[www.brightfocus.org/grant/A20141055](http://www.brightfocus.org/grant/A20141055)



**Jada Lewis, PhD** (7/1/15 – 6/30/18)

University of Florida

*Decreasing Expression of a Toxic Protein in Alzheimer's Disease*

*"Using a mouse model, we are developing a way to reduce the expression of the tau protein, which is associated with frontotemporal dementia and aggregates in Alzheimer's disease."*

[www.brightfocus.org/grant/A20156885](http://www.brightfocus.org/grant/A20156885)



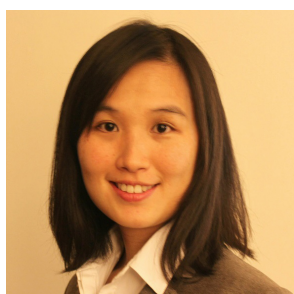
**Stephen Martin, PhD** (7/1/16 – 6/30/18)

University of Texas at Austin

*A New Approach to Treating Alzheimer's Disease*

*"Because there is an urgent, unmet need for drugs to treat both the symptoms and the disease, the proposed program will explore a novel strategy to treat Alzheimer's by targeting a biological pathway different from those of all existing drugs and all but one known clinical candidate."*

[www.brightfocus.org/grant/A2016443S](http://www.brightfocus.org/grant/A2016443S)



**Ming-Hsuan Ou-Yang, PhD** (7/1/15 – 6/30/17)

Northwestern University

*How BACE1 Regulates Learning and Memory Through Molecular Scissors*

*"Through this study we can better understand how BACE1 functions and whether there may be potential side effects of future BACE1 drugs, thus devising ways to ensure the safety and effectiveness of such agents."*

[www.brightfocus.org/grant/A2015289F](http://www.brightfocus.org/grant/A2015289F)



**Ana Pereira, MD** (7/1/16 – 6/30/19)

The Rockefeller University

*Enhancing Glutamate Levels as a Way to Treat Alzheimer's Disease*

*"Our project studies how dysfunction of the major glutamate transporter in the brain, GLT-1, is an important mechanism in several toxicities in Alzheimer's disease, thus potentially validating GLT-1 as a novel and specific target for drug development."*

[www.brightfocus.org/grant/A2016478S](http://www.brightfocus.org/grant/A2016478S)



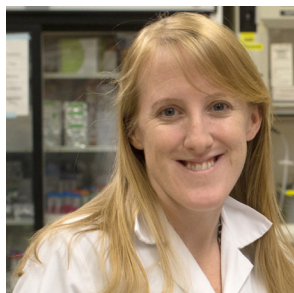
**Ryohei Yasuda, PhD** (7/1/15 – 6/30/18)

Max Planck Florida Institute for Neuroscience

*Identifying the Role of a Brain-specific Protein Centaurin-a1, in Alzheimer's Disease*

*"We recently identified that a signaling protein called centaurin-a1 (CentA1) causes AB-induced dysfunction of neurons and thus potentially contributes to brain dysfunction in Alzheimer's disease."*

[www.brightfocus.org/grant/A2015251S](http://www.brightfocus.org/grant/A2015251S)



**Tracy Young-Pearse, PhD** (7/1/15 – 6/30/18)

Brigham and Women's Hospital, Harvard University

*A Personalized Medicine Approach to Develop New Alzheimer's Disease Treatments*

*"The project combines a series of modern technologies and a deep understanding of Alzheimer's disease that will, for the first time, enable us to answer some very practical questions about how we can make future Alzheimer's treatments more effective."*

[www.brightfocus.org/grant/A20156075](http://www.brightfocus.org/grant/A20156075)



**Huda Zoghbi, MD** (7/1/16 – 6/30/19)

Baylor College of Medicine

*A Genetic Screen to Identify New Drug Targets for Alzheimer's Disease*

*"The major goal of this project is to discover new therapeutic targets for Alzheimer disease. We propose a research program centered on genes and genetic networks that control amyloid precursor protein (APP) levels using innovative screens of the 'druggable' genome."*

[www.brightfocus.org/grant/A20161515](http://www.brightfocus.org/grant/A20161515)

## **Why Do AB and Tau Mis-shape and Collect in the Alzheimer's Brain?**

Amyloid beta (AB) and tau are proteins that serve a useful role in healthy brains. Yet in Alzheimer's disease, they undergo molecular changes and take on altered shapes, which causes them to collect into tangles, plaques, and vascular deposits. Scientists are trying to pinpoint what goes wrong when amyloid fibers go from "normal" to becoming misshapen and toxic to surrounding tissues. Progress in these areas will lend insights into new front-line treatment strategies.



**David Brody, MD, PhD** (7/1/14 – 6/30/17)

Washington University School of Medicine

*Purifying the Most Toxic Forms of Beta Amyloid from the Brains of Patients with Alzheimer's Disease*

*"The goal of this project is to purify and characterize small, toxic proteins that may be the root cause of dementia in Alzheimer's disease."*

[www.brightfocus.org/grant/A20142705](http://www.brightfocus.org/grant/A20142705)



**Nicholas M. Kanaan, PhD** (8/16/13 – 6/30/17)

Michigan State University

*Are Tau Oligomers the Elusive Toxic Species of Tau within Alzheimer's Disease?*

*"Upon completion of this project, we hope to show that tau oligomers are the toxic tau species involved in Alzheimer's disease and other tauopathies."*

[www.brightfocus.org/grant/A2013364S](http://www.brightfocus.org/grant/A2013364S)



**Paul Seidler, PhD** (7/1/16 – 6/30/18)

University of California, Los Angeles

*Blocking Assembly of Tau Protein into Toxic Structures Associated with Alzheimer's Disease*

*"This project will expand our understanding of Alzheimer's disease by delineating the structural assemblies that are available to tau, another amyloid protein that, along with amyloid beta, is associated with Alzheimer's disease."*

[www.brightfocus.org/grant/A2016588F](http://www.brightfocus.org/grant/A2016588F)



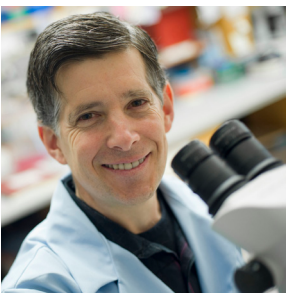
**Dominic Walsh, Bsc (Hons), PGCE, PhD** (1/1/14 – 12/31/16)

Brigham and Women's Hospital

*Identifying the Disease-Causing Form of the Amyloid Beta-Protein in Human Brain*

*"In this proposal, we aim to tackle one central issue - the identification from the human brain of toxic forms of Abeta that cause Alzheimer's disease."*

[www.brightfocus.org/grant/A2013059S](http://www.brightfocus.org/grant/A2013059S)



**Benjamin Wolozin, MD, PhD** (7/1/15 – 6/30/18)

Boston University

*Harnessing Reversible Protein Aggregation to Treat Alzheimer's Disease*

*"We hypothesize that the process of aggregation associated with stress granules might actually stimulate pathology in Alzheimer's disease."*

[www.brightfocus.org/grant/A2015256S](http://www.brightfocus.org/grant/A2015256S)





**Guilian Xu, PhD** (7/1/14 – 6/30/17)

University of Florida

*Are There Global Changes in Protein Metabolism with Alzheimer's Disease and Do These Changes Affect Cognition?*

*"We hypothesize that tau protein is one of the 'secondary misfolded' proteins that happens as human Alzheimer's disease progresses."*

[www.brightfocus.org/grant/A2014108S](http://www.brightfocus.org/grant/A2014108S)

## **How Alzheimer's Disease Develops in the Brain (Pathogenesis)**

Even now, more than 100 years after Alzheimer's disease was first diagnosed, we still are looking for clues as to how it starts. It's believed that chemical changes in the configuration of amyloid proteins (both tau and amyloid beta) may cause it to aggregate and become dysfunctional and toxic to the brain's neurons and other tissues. Once that happens, the damage grows. We need to learn how Alzheimer's spreads inside the brain through diseased nerve fibers and connections.



**Francesca Bartolini, PhD** (7/1/15 – 6/30/18)

Columbia University

*Microtubule Stabilization Pathways in Alzheimer's Disease*

*"Our studies will potentially identify new diagnostic markers and introduce a new class of cytoskeleton regulators that may be targeted in drug therapies aimed at rescuing cells from both amyloid beta and phospho-tau toxicity in Alzheimer's disease."*

[www.brightfocus.org/grant/A2015508S](http://www.brightfocus.org/grant/A2015508S)



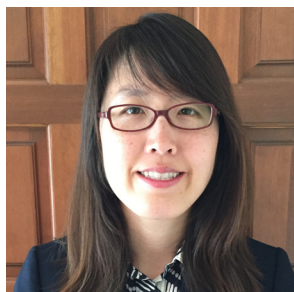
**Virginie Buggia-Prevot, PhD** (7/1/14 – 1/31/17)

University of Chicago

*Understanding the Role of Novel Endocytic Proteins in Alzheimer's Disease Pathogenesis*

*"The goal of my research is to better understand the causes of Alzheimer's disease by studying the mechanisms that contribute to the underlying pathology of the disease, in order to identify potential therapeutic targets."*

[www.brightfocus.org/grant/A2014316F](http://www.brightfocus.org/grant/A2014316F)



**Karen Chiang, PhD** (7/1/15 – 6/30/17)

University of California, San Diego

*The Impact Of AB on the Spread of Tau Toxicity in the Brain*

*"My research aim is to explain how the relative synaptic localization of amyloid beta expression contributes to the development of tau pathology."*

[www.brightfocus.org/grant/A2015595F](http://www.brightfocus.org/grant/A2015595F)



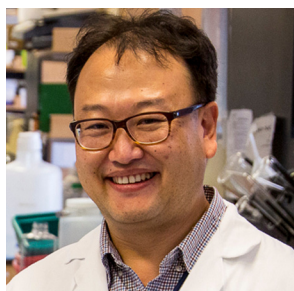
**Umesh Jinwal, PhD** (7/1/15 – 6/30/17)

University of South Florida

*Defining the Role of Tau and Kinase Regulator Chaperone Protein Cdc37 in Alzheimer's Disease*

*"The main focus of this research is to investigate the role of Cdc37, a chaperone protein, in tau abnormalities using cellular and animal models of Alzheimer's disease."*

[www.brightfocus.org/grant/A2015666S](http://www.brightfocus.org/grant/A2015666S)



**Doo Yeon Kim, PhD** (7/1/16 – 6/30/19)

Massachusetts General Hospital, Harvard University

*A Human Cellular Alzheimer's Disease Model Based on 3D Culture Technology*

*"In this project, we will further develop and characterize a human 'Alzheimer's disease in a dish' model based on a unique three-dimensional (3D) human neural progenitor cell culture technique."*

[www.brightfocus.org/grant/A2016362S](http://www.brightfocus.org/grant/A2016362S)



**Harry LeVine, III, PhD** (7/1/14 – 6/30/17)

University of Kentucky

*Identifying the Type of Neuron Accumulating Amyloid in Early Alzheimer's Disease*

*"The goal of our project is to identify the type of neuron in humans that is first affected by human-specific pathology in Alzheimer's disease (AD) and to determine how further changes in humans as AD progresses differ from those in a mouse model of AD pathology."*

[www.brightfocus.org/grant/A2014044S](http://www.brightfocus.org/grant/A2014044S)



**Jiri Safar, MD** (7/1/16 – 6/30/19)

Case Western Reserve University

*Profiling Prion-Like Strains of AB that Control Alzheimer's Progression*

*"We believe our project addresses a confounding puzzle: Why clinical symptomatology, severity, and progression rates of late-onset Alzheimer disease frequently do not coincide with the total beta amyloid (AB) load?"*

[www.brightfocus.org/grant/A2016085S](http://www.brightfocus.org/grant/A2016085S)



**Stephen Salton, MD, PhD** (7/1/16 – 6/30/18)

Icahn School of Medicine at Mt. Sinai

*Role of VGF in Alzheimer's Disease Pathogenesis and Progression*

*"This project can be expected to result in a strong foundation for future exploration of approaches that deliver VGF and/or VGF-derived peptides to patients with Alzheimer's disease."*

[www.brightfocus.org/grant/A2016508S](http://www.brightfocus.org/grant/A2016508S)



**Juan Troncoso, MD** (7/1/15 – 6/30/17)

Johns Hopkins University

*Alzheimer's Disease, Before Plaques and Tangles*

*"We propose to study the postmortem brain of individuals between 30 and 50 years of age and to identify those brains with the very early pathologic changes or lesions of Alzheimer's disease."*

[www.brightfocus.org/grant/A2015332S](http://www.brightfocus.org/grant/A2015332S)



**Hongmin Wang, PhD** (7/1/14 – 6/30/17)

University of South Dakota

*Using Genetically Modified Mice to Study the Role of Ubiquilin-1 in Alzheimer's Disease*

*"The goal of this project is to determine whether the onset and progression of Alzheimer's disease in mice can be altered by the manipulation of a protein called ubiquilin-1."*

[www.brightfocus.org/grant/A2014420S](http://www.brightfocus.org/grant/A2014420S)

## Role of Immune Factors and Clearance Mechanisms

One theory about Alzheimer's disease is that it may be triggered, in part, by a breakdown in the brain's immune system. Normally our brain has ways of clearing damaged cells and other unwanted particles in its midst and disposing them into the bloodstream—which can be thought of as “taking out the garbage.” However, a chronic rise in unwanted debris, including toxic AB and tau proteins, can short-circuit the immune system. Grantees are looking at what causes the immune response to become unbalanced and whether there are ways to help the brain's immune cells and system do a better job of fighting Alzheimer's.



**Randall Bateman, MD** (10/1/14 – 9/30/17)

Washington University

*A New Method to Measure Tau Kinetics in Humans with Alzheimer's Disease*

*“Our study elucidating human central nervous system tau kinetics will enable better designs for prevention and treatment of Alzheimer's disease in the future.”*

[www.brightfocus.org/grant/A2014384S](http://www.brightfocus.org/grant/A2014384S)



**Swetha Gowrishankar, PhD** (7/1/16 – 6/30/18)

Yale University

*Role of Axonal Lysosome Transport in Alzheimer's Disease Pathology*

*“Collectively, our efforts are expected to provide understanding of the mechanisms whereby amyloid plaques trigger the accumulation of lysosomes in surrounding axons, as well as the contribution of such lysosomes to the disease process.”*

[www.brightfocus.org/grant/A2016411F](http://www.brightfocus.org/grant/A2016411F)



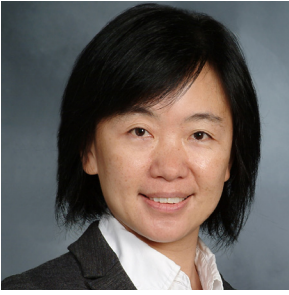
**Jean-Vianney Haure-Mirande, PhD** (7/1/16 – 6/30/18)

Icahn School of Medicine at Mt. Sinai

*Role of Microglia in Alzheimer's Disease: Deleterious or Helpful?*

*“The aim of our project is to: 1) understand the role of the immune system in the pathogenesis of Alzheimer's, and 2) provide new insight for a therapeutic target to control Alzheimer's disease.”*

[www.brightfocus.org/grant/A2016482F](http://www.brightfocus.org/grant/A2016482F)



**Wenjie Luo, PhD** (7/1/16 – 6/30/19)

Weill Cornell Medical College

*Cellular Mechanisms Underlying Microglia-Mediated Amyloid Degradation*

*"Accumulation of abnormal amyloid and tau proteins in the brains is believed to be detrimental for brain functions; thus, searching for ways to reduce these bad proteins from the brain may help us find a promising drug for Alzheimer's disease."*

[www.brightfocus.org/grant/A2016399S](http://www.brightfocus.org/grant/A2016399S)



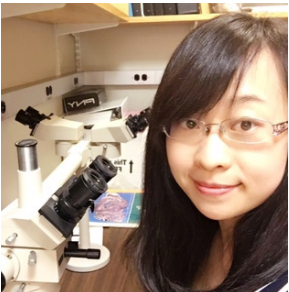
**Zixu Mao, PhD** (7/1/16 – 6/30/19)

Emory University

*Understanding Brain Inflammation in Alzheimer's Disease*

*"The goal of my project is to understand the critical signaling pathways that underlie microglial inflammatory response in the context of Alzheimer's disease pathogenesis."*

[www.brightfocus.org/grant/A2016501S](http://www.brightfocus.org/grant/A2016501S)



**Qiaoqiao Shi, PhD** (7/1/16 – 6/30/18)

Brigham and Women's Hospital, Harvard University

*New Mouse Models to Study the Role of Complement in Brain Aging and Neurodegeneration*

*"Complement component 3 (C3) [an immune molecule] is up-regulated in Alzheimer's disease and may contribute to the synapse loss that underlies cognitive decline. To further understand when and where C3 plays a role in Alzheimer's disease, we have generated two novel mouse models."*

[www.brightfocus.org/grant/A2016425F](http://www.brightfocus.org/grant/A2016425F)

## Harnessing The Body's Own Protective Mechanisms

Even when they share the same set of risk factors, such as a genetic mutation or presence of amyloid beta plaques, not all people develop Alzheimer's disease. Scientists are looking for the reasons why, with the hope that the body's innate defense mechanisms may provide clues and could possibly be replicated or manipulated as a prevention or treatment strategy.



**Jason Gestwicki, PhD** (7/1/15 – 6/30/18)

University of California, San Francisco

*Control of Normal Tau Levels by Molecular Chaperones*

*"In the current project, we are trying to understand how the body's own proteins (termed molecular chaperones) protect us against Alzheimer's disease."*

[www.brightfocus.org/grant/A2015297S](http://www.brightfocus.org/grant/A2015297S)



**Stacy Grunke, PhD** (7/1/15 – 6/30/17)

Baylor College of Medicine

*Network Disruption and Recovery Following Cell Loss in Early Stage Alzheimer's Disease*

*"We will determine whether there are mechanisms for repair of the brain circuitry that can facilitate functional recovery of memory processing once entorhinal neurons are lost."*

[www.brightfocus.org/grant/A2015016F](http://www.brightfocus.org/grant/A2015016F)



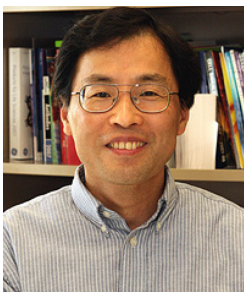
**Marie-Victoire Guillot-Sestier, PhD** (11/1/15 – 10/31/17)

University of Southern California

*Relative Contribution of Central vs. Peripheral Immune Cells in Cerebral Amyloid Beta Clearance*

*"Currently, my ambition is to identify whether resident central and/or infiltration of peripheral immune cells in the brain are responsible for amyloid beta clearance."*

[www.brightfocus.org/grant/A2015309F](http://www.brightfocus.org/grant/A2015309F)



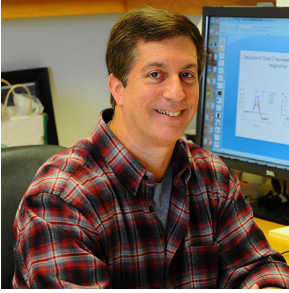
**Chien-liang Lin, PhD** (7/1/14 – 6/30/17)

The Ohio State University Research Foundation

*Regulating Glutamate Levels as a Therapeutic Strategy for Alzheimer's Disease*

*"The goal of this research is to develop restoration of EAAT2, a glial glutamate transporter, function as a therapeutic strategy for Alzheimer's disease."*

[www.brightfocus.org/grant/A2014315S](http://www.brightfocus.org/grant/A2014315S)



**Richard Morrison, PhD** (7/1/14 – 6/30/17)

University of Washington School of Medicine

*Restoring a Novel Multifunctional Protein in Neurons to Enhance Cognitive Function in Alzheimer's Disease*

*"The goal of our project is to enhance cognitive function and reduce cell damage in Alzheimer's disease by restoring expression of a novel multifunctional protein in neurons that is lost during the progression of Alzheimer's."*

[www.brightfocus.org/grant/A2014237S](http://www.brightfocus.org/grant/A2014237S)



**Charles Sanders, PhD** (7/1/15 – 6/30/18)

Vanderbilt University Medical Center

*How Does TREM2 Help the Brain Clean out Molecular "Garbage" that Contributes to Alzheimer's Disease*

*"Triggering receptor expressed on myeloid cells 2, or TREM2, is a protein that plays a major role in helping the brain to cleanse itself of toxic substances that contribute to Alzheimer's disease and related disorders. We seek to unravel the role that the TREM2 protein plays in helping to prevent Alzheimer's disease."*

[www.brightfocus.org/grant/A2015565S](http://www.brightfocus.org/grant/A2015565S)

## **Controlling Risk Factors to Delay or Prevent Alzheimer's Onset & Progression**

Alzheimer's disease (AD) is a frustratingly complex disease of mixed origins that expresses itself in different ways. At least 70% of its variation remains unexplained, so numerous routes of discovery are being explored. Type 2 diabetes mellitus, one of the most common medical illnesses impacting our society, increases the risk of developing AD. Concussions, affecting one million North Americans each year, are another risk factor strongly associated with AD. In addition, AD has been associated with poor sleep quality, which appears to affect amyloid beta clearance; and with a sort of "brain diabetes" that might be addressed by a special diet. These factors are being explored in the hope that better understanding can lead to new therapies.



**Michal Schnaider Beeri, PhD** (7/1/14 – 6/30/17)

Sheba Medical Center and Interdisciplinary Center

*Blood Vessel Function in Cognitive Impairment with Diabetes*

*"The overall goal of this study is to investigate the contribution of arterial wall functions (AWF, the flexibility of the vasculature in the brain and in the periphery) to cognitive function in individuals with type 2 diabetes, who are at particularly high risk of developing dementia."*

[www.brightfocus.org/grant/A2014268S](http://www.brightfocus.org/grant/A2014268S)



**Jason Brandt, PhD** (7/1/16 – 6/30/19)

Johns Hopkins University

*A High Fat, Low Carbohydrate Diet for MCI and Early Alzheimer's Disease*

*"This study will test whether a special diet that has been used to treat other brain disorders may be useful to treat Alzheimer's disease."*

[www.brightfocus.org/grant/A2016073S](http://www.brightfocus.org/grant/A2016073S)



**Frank LaFerla, PhD** (7/1/15 – 6/30/18)

University of California, Irvine

*Investigating Mechanisms that Link Diabetes Mellitus and Alzheimer's Disease*

*"This proposal seeks to elucidate how diabetes affects Alzheimer's disease."*

[www.brightfocus.org/grant/A2015535S](http://www.brightfocus.org/grant/A2015535S)



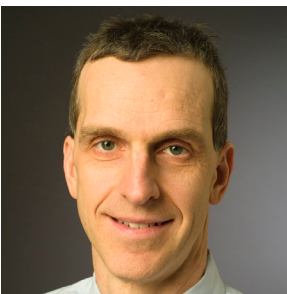
**Brendan Lucey, MD** (7/1/16 – 6/30/19)

Washington University School of Medicine

*Sleep Quality and Decreasing Amyloid-Beta Levels in the Human Brain*

*"This study proposes to answer several of these questions: 1) does poor sleep quality increase amyloid beta and 2) does improving sleep quality in poor sleepers decrease amyloid beta?"*

[www.brightfocus.org/grant/A2016180S](http://www.brightfocus.org/grant/A2016180S)



**Donald Redelmeier, MD** (7/1/15 – 6/30/18)

Sunnybrook Research Institute

*A New Way to Recover From a Concussion and Avoid Alzheimer's Disease*

*"We propose a computerized study linking multiple healthcare databases (anticipated sample size = 30,000, median follow-up duration = 10 years) to test whether statin treatment might decrease the risk of subsequent dementia in seniors following a concussion."*

[www.brightfocus.org/grant/A2015284S](http://www.brightfocus.org/grant/A2015284S)



## Preserving the Brain Network in Alzheimer's

The human brain has an estimated 100 billion neurons, meaning we have quite a few to spare, and extended from each of them is a long fiber, known as an “axon,” which can run half the length of our bodies. These axons form connections known as “synapses” with other nerve cells, creating an incredible communications network through which our body talks to our brain, and vice versa. Despite the loss of individual neurons, it isn't until the brain's entire communications network malfunctions that we experience Alzheimer's most typical symptoms—things like forgetting common words, or becoming lost in familiar places. Even then, the brain is amazingly adaptable and “plastic,” meaning it actively remodels itself to meet new demands. If a part of the network becomes too damaged to connect with other neurons by the most direct route, it will find detours and form new connections known as indirect neural pathways. ADR grantees are devising new ways to study how this network works and preserve its function for as long as possible, even after the onset of Alzheimer's disease.



**Iman Aganj, PhD** (7/1/16 – 6/30/19)

Massachusetts General Hospital, Harvard Medical School

*Imaging Biomarkers for Alzheimer's: New Methods to Account for Indirect Brain Connections*

*“In this project, I'll develop necessary computer tools and imaging techniques to model neuronal connections in the human brain, and use the results to better understand how the brain architecture and function are influenced by Alzheimer's disease.”*

[www.brightfocus.org/grant/A2016172S](http://www.brightfocus.org/grant/A2016172S)



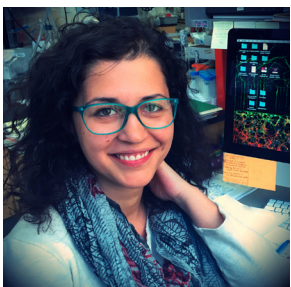
**Ulrich Hengst, PhD** (7/1/15 – 6/30/18)

Columbia University

*Spread of Alzheimer's Disease Along Neuronal Connections*

*“We plan to study the question of what precisely is happening within an axon that connects to a diseased area of the brain, and how is the neurodegenerative signal transmitted back to the neuron.”*

[www.brightfocus.org/grant/A2015093S](http://www.brightfocus.org/grant/A2015093S)



**Selene Lomoio, PhD** (7/1/16 – 6/30/18)

Tufts University School of Medicine

*Reorganizing the Neuronal Highway in the Alzheimer's Brain*

*“Our goal is to try to understand what is disrupting the neuronal traffic in Alzheimer's disease brains, find a way to prevent it, and possibly find a treatment for Alzheimer's.”*

[www.brightfocus.org/grant/A2016379F](http://www.brightfocus.org/grant/A2016379F)



**Tara Tracy, PhD** (7/1/16 – 6/30/18)

The David J. Gladstone Institutes

*Investigating the Impact of KIBRA Protein Loss on Synapse Function and Memory*

*"My research will advance our understanding of how the loss of a kidney and brain-expressed (KIBRA) protein at synapses contributes to cognitive decline in Alzheimer's disease."*

[www.brightfocus.org/grant/A2016360F](http://www.brightfocus.org/grant/A2016360F)

## **Pioneering New Ways to Look Inside the Brain**

Solving Alzheimer's requires knowing as much as possible about how the brain works throughout our lifespan, including normal aging, and how its different regions become impacted by the disease. Thanks to new imaging techniques being developed and tested by our grantees, the Alzheimer's brain no longer has to be visualized "in theory," or through postmortem examination, but can be observed through live imaging. Grantees are working to develop and validate high-definition imaging that will differentiate between early disease and normal aging, and are also using imaging to assess the impact of tau tangles on neuronal networks and the associated onset of depressive symptoms that might serve as an indicator of early disease. Ultimately these techniques will help us monitor brain health, diagnose Alzheimer's onset at the earliest possible point, and track the progress of cures.



**Jennifer Gatchel, MD, PhD** (7/1/16 – 6/30/18)

McLean Hospital, Massachusetts General Hospital

*Depressive Symptoms, Proteins AB and Tau, and Neuronal Network Activity in Prodromal and Early Alzheimer's Disease*

*"The goal of my project is to investigate whether specific brain changes, including both the accumulation of two of the main Alzheimer's disease associated proteins, — amyloid beta (AB) and tau — and associated changes in connections between neurons, might underlie these symptoms in early Alzheimer's."*

[www.brightfocus.org/grant/A2016434F](http://www.brightfocus.org/grant/A2016434F)



**Xiong Jiang, PhD** (7/1/16 – 6/30/19)

Georgetown University

*A Novel Non-Invasive MRI-Based Biomarker of Early Stages of Alzheimer's Disease*

*"Here we propose to develop and validate multimodality magnetic resonance imaging (MRI) techniques that can help to detect and quantify asymptomatic Alzheimer's progression."*  
[www.brightfocus.org/grant/A2016251S](http://www.brightfocus.org/grant/A2016251S)



**Rik Ossenkoppele, PhD** (7/1/14 – 6/30/17)

Alzheimer Center of the VU University Medical Center

*Testing the Amyloid Cascade Hypothesis In Humans Using a Novel Tau PET Tracer*

*"We propose to study the role of tau in Alzheimer's disease patients by investigating its relationships with beta amyloid, shrinkage of the brain and communication between brain cells."*  
[www.brightfocus.org/grant/A2014083F](http://www.brightfocus.org/grant/A2014083F)



**Emilie Reas, PhD** (7/1/16 – 6/30/18)

University of California, San Diego

*Novel Biomarkers of Brain Microstructure in Aging and Mild Cognitive Impairment*

*"Our research aims to characterize the small-scale structural changes that occur during the earliest stages of Alzheimer's disease, and describe how they relate to cognitive decline during aging and neurodegenerative disease."*  
[www.brightfocus.org/grant/A2016241F](http://www.brightfocus.org/grant/A2016241F)



**Laura Wisse, PhD** (7/1/16 – 6/30/18)

University of Pennsylvania

*Separating Early Alzheimer's Disease and Aging Effects in Search of Markers to Track Alzheimer's Treatment Effects*

*"My ultimate goal is to find a good biomarker in the brain that can help track disease progression and treatment effects in an early stage of the disease, before the damage has become too severe."*  
[www.brightfocus.org/grant/A2016432F](http://www.brightfocus.org/grant/A2016432F)

## Biomarkers for Early Screening and Detection

Alzheimer's disease (AD) has a "preclinical" phase, consisting of a gradual changes in the brain 10-20 years before the first outward symptoms. The best hope of stopping AD is during this phase. There are drugs now in development that may potentially slow or stop its course but may only work if started early. All this means that early screening and detection of AD are critical. Our grantees are developing and testing biomarkers that would make it possible to screen for AD using a simple blood test, through cerebrospinal fluid, or through a molecular probe that would detect the earliest changes in amyloid beta's molecular shape.



**Veer Bala Gupta, PhD** (7/1/15 – 6/30/17)

Edith Cowan University

*Studying Proteins in Blood to Detect Alzheimer's Disease at an Early Stage*

*"We propose that investigating familial form of Alzheimer's disease will be an innovative approach to identify and develop reliable blood-based biomarkers for this disease."*

[www.brightfocus.org/grant/A2015641F](http://www.brightfocus.org/grant/A2015641F)



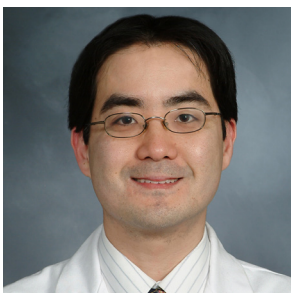
**Francis Hane, PhD** (7/1/15 – 6/30/17)

Lakehead University

*Using a Molecular Probe to Predict the Onset of Alzheimer's*

*"Our goal is to develop magnetic resonance imaging (MRI) techniques that can be used to detect Alzheimer's disease up to 10 years before the start of Alzheimer's symptoms."*

[www.brightfocus.org/grant/A2015344F](http://www.brightfocus.org/grant/A2015344F)



**Makoto Ishii, MD, PhD** (7/1/15 – 6/30/18)

Joan and Sanford I. Weill Medical College of Cornell University

*Identifying How Fat Hormones That Regulate Body Weight Are Affected in Alzheimer's*

*"The goal of this project is to understand why Alzheimer's disease leads to loss in body weight, particularly early in the disease, and by doing so explore new avenues for developing novel therapeutic targets and diagnostic tools."*

[www.brightfocus.org/grant/A2015485S](http://www.brightfocus.org/grant/A2015485S)



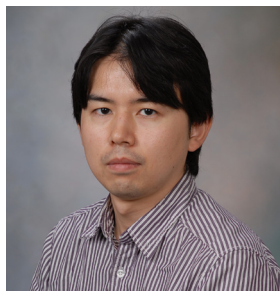
**Richard Perrin, MD, PhD** (7/1/14 – 6/30/17)

Washington University School of Medicine

*New Ways to Detect, Monitor, and Predict Early Alzheimer's with Spinal Fluid*

*"The goal of this study is to discover a timeline of protein changes within the cerebrospinal fluid (the fluid that bathes the brain) that can be used to diagnose and monitor Alzheimer's disease, even years before symptoms of the disease appear."*

[www.brightfocus.org/grant/A2014296S](http://www.brightfocus.org/grant/A2014296S)



**Mitsuru Shinohara, PhD** (7/1/14 – 11/30/16)

Mayo Clinic, Jacksonville

*Synaptic Regulation of Beta Amyloid Metabolism and Associated Biomarkers in Body Fluids*

*"By shedding light on how synapses regulate the production and behavior of toxic beta amyloid in brains at a molecular level, and what proteins or molecules in body fluids are associated with that process, the results of our proposed research will help identify appropriate biomarkers that indicate disturbances in synaptic beta amyloid metabolism in brains, before the development of Alzheimer's disease."*

[www.brightfocus.org/grant/A2014137F](http://www.brightfocus.org/grant/A2014137F)



**Philip Verghese, PhD** (5/1/16 – 4/30/18)

C2N Diagnostics

*Silk ABeta Spot Test*

*"The primary objective is to develop novel and simple blood tests using the SILK™ and SISAQ platforms that can be used for 1) aiding in selection of patients for inclusion in AD trials, 2) demonstrating drug target engagement, and 3) tracking disease progression."*

[www.brightfocus.org/grant/CA2016636](http://www.brightfocus.org/grant/CA2016636)



**Zhentao Zhang, MD, PhD** (7/1/15 – 6/30/17)

Emory University

*A New Method to Predict Alzheimer Disease*

*"The goal of the project is to find new biomarkers that can detect preclinical Alzheimer's disease."*

[www.brightfocus.org/grant/A2015359F](http://www.brightfocus.org/grant/A2015359F)

## Genetic Factors

Genes represent another possible chance to intervene in the preclinical phase of Alzheimer's disease. Mutations to the *APOE* gene, already associated with Alzheimer's risk, may affect protein metabolism and lead to deposits of amyloid and other aberrant proteins. Another genetic variant affects amyloid precursor protein, one of the raw ingredients of AB, and appears to double the risk for late onset Alzheimer's disease. And inherited forms of Alzheimer's are being studied for genetic mutations and variations that may help explain the far more common, late-onset form of the disease. Genetic contributors, once discovered, are potentially modifiable.



**Carlos Cruchaga, PhD** (7/1/13 – 9/30/16)

Washington University in St. Louis

*A New Method to Identify Protein and Genes Involved in Alzheimer's Disease*

*"The long-term goal is to use novel and powerful approaches to identify new genes and protein variants associated with Alzheimer's disease."*

[www.brightfocus.org/grant/A2013359S](http://www.brightfocus.org/grant/A2013359S)



**Albert Davis, MD, PhD** (7/1/15 – 6/30/17)

Washington University School of Medicine

*Does the APOE Gene Regulate Protein Aggregation in PD?*

*"This project will study how brain proteins clump and will hopefully pave the way for new treatments for brain diseases including Alzheimer disease, Parkinson disease (PD), and related brain disorders."*

[www.brightfocus.org/grant/A2015577F](http://www.brightfocus.org/grant/A2015577F)



**David Irwin, MD, MS** (7/1/16 – 6/30/18)

University of Pennsylvania School of Medicine

*Non-Amnestic Alzheimer's Disease Biology*

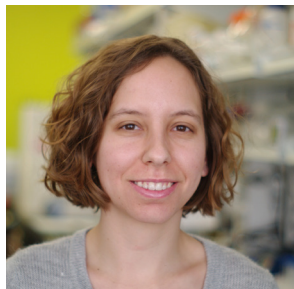
*"The goal of our project is to study the pattern of disease spread and genetic risk for non-amnestic clinical variants of Alzheimer's disease."*

[www.brightfocus.org/grant/A2016244S](http://www.brightfocus.org/grant/A2016244S)



**Catherine Kaczorowski, PhD** (7/1/16 – 6/30/19)  
The University of Tennessee Health Science Center  
*A New Method to Identify Genes Critically Involved in Alzheimer's Disease*

*"The main goal of our project is to identify new genes that may modify the age at which a person will develop symptoms of Alzheimer's disease, termed 'age-at-onset.'"*  
[www.brightfocus.org/grant/A2016397S](http://www.brightfocus.org/grant/A2016397S)



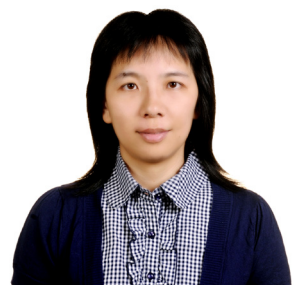
**Celeste Karch, PhD** (7/1/15 – 6/30/18)  
Washington University School of Medicine  
*Defining the Role of Phospholipase D3 in Alzheimer's Disease*

*"We have identified genetic variants in phospholipase D3 (PLD3) that double the risk for late-onset Alzheimer's disease. The goal of this study is to use cell and mouse models to begin to define the molecular mechanisms by which PLD3 variants influence APP metabolism and contribute to Alzheimer's pathology."*  
[www.brightfocus.org/grant/A2015411S](http://www.brightfocus.org/grant/A2015411S)



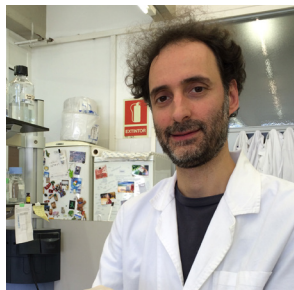
**Joseph H Lee, PhD** (7/1/15 – 6/30/18)  
Columbia University  
*Genome Search for Genetic Modifiers of Alzheimer Disease Age at Onset*

*"The main goal of this project is to identify new genes that may lead to either earlier or later age at onset of Alzheimer disease by examining Puerto Rican families that have at least one person who carry a unique mutation in the PSEN1 gene."*  
[www.brightfocus.org/grant/A2015633S](http://www.brightfocus.org/grant/A2015633S)



**Chia-Chen Liu, PhD** (7/1/16 – 6/30/18)  
Mayo Clinic, Jacksonville  
*The Effects of ApoE Isoforms on Brain Functions and Alzheimer's Disease*

*"Using a unique inducible mouse model, our studies will for the first time test how peripheral ApoE affects brain functions and Alzheimer's disease progression."*  
[www.brightfocus.org/grant/A2016346F](http://www.brightfocus.org/grant/A2016346F)



**Carlos Saura, PhD** (7/1/14 – 6/30/17)

Universitat Autònoma de Barcelona

*Genetic Mechanisms Underlying Memory Loss in Alzheimer's Disease*

*"The goal of this project is to investigate novel molecular mechanisms that regulate gene expression programs causing memory impairments at early Alzheimer's disease stages."*

[www.brightfocus.org/grant/A2014417S](http://www.brightfocus.org/grant/A2014417S)

## Blood Vessels and Alzheimer's Disease

Most Alzheimer's disease is "mixed," meaning that in addition to amyloid plaques and tangles, there are changes in the brain's blood vessels that interfere with normal circulation. Amyloid can deposit in vessels—a condition known as cerebral amyloid angiopathy (CAA)—and Alzheimer's-related inflammation causes vessels to grow "sticky," stalling the blood flow. In both cases the reduced flow compromises the supply of oxygen and nutrition to the brain. Tau protein has been associated with increased expression of proteins involved in new vessel growth (impact still unknown). In addition, the extremely tight junctions of the blood-brain barrier (BBB), which close the brain's circulation from the rest of the body, may also be affected in Alzheimer's. It may be possible to manipulate tight junctions to allow better clearance of toxins and facilitate drug delivery through the BBB.



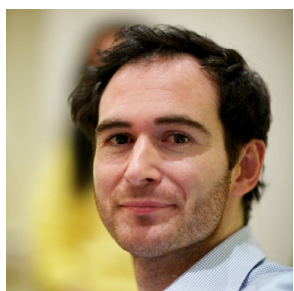
**Rachel Bennett, PhD** (7/1/16 – 6/30/18)

Massachusetts General Hospital, Harvard University

*Blood Vessel Changes in Tauopathy*

*"This research aims to determine if blood vessel alterations are an early or late event in the disease process and to use 'off the shelf' drugs to prevent blood vessel growth."*

[www.brightfocus.org/grant/A2016404F](http://www.brightfocus.org/grant/A2016404F)



**Matthew Campbell, PhD** (7/1/15 – 6/30/18)

Trinity College, Dublin

*A Novel Way of Removing Toxic Material from the Brain in Early Alzheimer's Disease*

*"The primary goal of this research project is to develop a greater understanding of the integrity of blood vessels in the brain in the context of Alzheimer's disease."*

[www.brightfocus.org/grant/A2015548S](http://www.brightfocus.org/grant/A2015548S)





**Jorge Ghiso, PhD** (7/1/15 – 6/30/18)

New York University School of Medicine

*Effect of Aging and Dysfunction of Cerebral Microvasculature in Alzheimer's Disease*

*"The central focus of this project is how brain AB removal is additionally influenced by aging and by the dysfunction of the cerebral microvasculature."*

[www.brightfocus.org/grant/A2015275S](http://www.brightfocus.org/grant/A2015275S)



**Pietro Michelucci, PhD** (1/1/16 – 12/31/16)

Human Computation Institute

*Crowd-powered Microvascular Modeling*

*"Our Phase I goal is to see whether thousands of public participants can analyze the research data just as accurately as lab experts, but by working together, do it much faster."*

[www.brightfocus.org/grant/CA2016629](http://www.brightfocus.org/grant/CA2016629)



**Jerome Robert, PhD** (1/1/16 – 12/31/17)

University of British Columbia

*Development of a New Model of Brain Vasculature in the Test Tube*

*"We are developing novel methods to analyze in the test tube the importance of the brain vessels in the development of Alzheimer's disease."*

[www.brightfocus.org/grant/A2015324F](http://www.brightfocus.org/grant/A2015324F)

## Caregiving Research

Caregiving goes hand in hand with Alzheimer's disease—an estimated 70 percent of all people with Alzheimer's are cared for at home, often with care provided by family members and friends. As the disease worsens, there's an impact on caregivers that puts them, too, at risk of depression and cognitive problems. The impact of that is being studied, as are ways to improve home-based dementia care.



**Corinna Lathan** (4/15/16 – 4/14/17)

AnthroTronix

*Health-eBrain Study: Addressing Depression and Cognitive Functioning in Dementia Caregivers*

*"The Health-eBrain Study seeks to understand the cognitive functioning of dementia caregivers who are heavily burdened and who are at risk for mood disorders."*

[www.brightfocus.org/grant/CA2016602](http://www.brightfocus.org/grant/CA2016602)



**Constantine Lyketsos, MD, MPH** (8/15/15 – 12/31/16)

Johns Hopkins Bayview Medical Center

*Home Is Where the Future Is: BrightFocus Foundation Consensus Panel on Home-Based Dementia Care*

*"Panel findings show the evidence for providing wholistic, individualized home-based dementia care for patient and caregivers benefits is robust and growing."*

[www.brightfocus.org/grant/CA2016637](http://www.brightfocus.org/grant/CA2016637)

### BrightFocus Grants at a Glance

55%

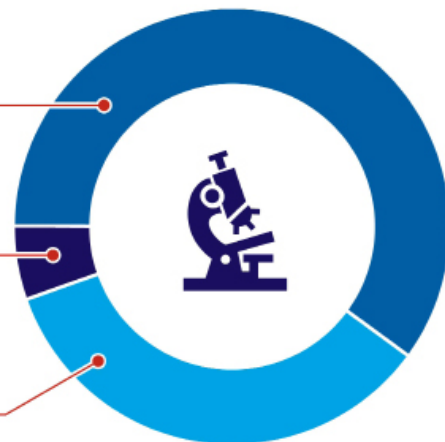
BASIC RESEARCH GRANTS

10%

CLINICAL RESEARCH GRANTS

35%

TRANSLATIONAL RESEARCH GRANTS



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*\*Note: These are active Alzheimer's Disease Research grants as of 7/1/2016.*



Alzheimer's Disease Research, a BrightFocus Foundation program