Commonwealth of Virginia
Alzheimer's and Related Diseases Research Award Fund

2025-2026 ARDRAF Recipients Announced

The Alzheimer's and Related Diseases Research Award Fund (ARDRAF) was established by the Virginia General Assembly in 1982 to stimulate innovative investigations into Alzheimer's disease and related disorders along a variety of avenues, including the causes, epidemiology, diagnosis and treatment of the disorder; public policy and the financing of care; and the social and psychological impacts of the disease upon the individual, family and community.

The ARDRAF is administered by the Virginia Center on Aging in the College of Health Professions at Virginia Commonwealth University.

Awards Committee 2025

The Virginia Center on Aging acknowledges the dedicated work of this independent review panel of subject matter experts, and is grateful to them for contributing their time and expertise.

Kimberly Battle, Ph.D., FNP-BC
VCU Health

Anne Brown, Ph.D.
Virginia Tech

Frank J. Castora, Ph.D.
Eastern Virginia Medical School

Severn Churn, Ph.D.
National Institutes of Health

Leslie Davidson, OTR/L, Ph.D., FAOTA
George Washington University

Vasiliki N. Ikonomidou, Ph.D.
VecTech

Christine Jensen, Ph.D.
Riverside Center for Excellence in Aging and Lifelong Health

Uma Kelekar, Ph.D.
Marymount University

Alicia Pickrell, Ph.D.
Virginia Tech

Elvin Price, PharmD, Ph.D.
Virginia Commonwealth University

J. Tina Savla, Ph.D.
Virginia Tech

Patty Slattum, PharmD, Ph.D.
Virginia Commonwealth University

Jodi Winship, Ph.D., OTR/L
Richmond Aging and Engaging

George Worthington, M.S.
Virginia Department for Aging and Rehabilitative Services

Ning Zhang, Ph.D.
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Questions about the projects may be directed to the investigators or the ARDRAF administrator, Dr. Faika Zanjani (fzanjani@vcu.edu).

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THE ROLE OF DENDRITIC MITOCHONDRIA IN ALZHEIMER'S DISEASE SYNAPSE DYSFUNCTION

Shannon Farris, Ph.D., and Sharon Swanger, Ph.D. - Virginia Tech

The entorhinal cortex (EC) is one of the first brain regions affected by Alzheimer's disease (AD) in both patients and animal models. EC–hippocampal connections are critical for memory, and their deterioration is believed to contribute to early memory loss in AD. However, the molecular basis of this circuit's vulnerability remains unclear.

Mitochondria, the energy-producing structures in cells, are essential for synapse function, and mitochondrial dysfunction is an early hallmark of AD. Restoring mitochondrial function may help prevent or slow disease progression. In the hippocampus, mitochondria near EC inputs are larger, more numerous and show enhanced calcium signaling compared to those near other inputs. Excessive mitochondrial calcium is thought to drive AD pathology. We hypothesize that mitochondria near EC–hippocampal synapses are more vulnerable due to excess calcium uptake.

This unique mitochondrial population offers a model for studying mitochondria–synapse crosstalk in the context of calcium dysregulation in AD. Using brain slices from healthy and AD model mice, we will compare mitochondrial calcium uptake and synapse function in EC–hippocampal circuits. This pilot study will determine whether enhanced mitochondrial calcium contributes to synaptic vulnerability in AD.

Findings will support new research into upstream mechanisms of mitochondrial calcium dysregulation and downstream effects on synapses. Understanding the cellular basis of EC–hippocampal vulnerability is essential for identifying therapeutic targets to prevent synapse loss and memory impairment in AD.

Shannon Farris, Ph.D. and Sharon Swanger, Ph.D. are Assistant Professors at the Fralin Biomedical Research Institute at Virginia Tech Carilion and in the Department of Biomedical Sciences and Pathobiology at Virginia Tech. Dr. Farris's lab investigates the molecular basis of memory, focusing on how mitochondrial diversity shapes hippocampal cell- and circuit-specific functions. Dr. Swanger's lab explores receptor diversity among synapses in functionally distinct brain circuits and its role in disease susceptibility. Together, this research team aims to understand how mitochondria-synapse crosstalk contributes to entorhinal cortex–hippocampal circuit dysfunction in Alzheimer's disease.

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INVESTIGATING THE IONIC AND TRANSCRIPTIONAL MECHANISMS MEDIATING AMYLOID-BETA (AB)-INDUCED NEURONAL FUNCTIONAL INSTABILITY: IMPLICATIONS FOR TARGETED THERAPIES TO TREAT MEMORY LOSS IN DOWN SYNDROME AND ALZHEIMER'S DISEASE.

Andrew A. George, Ph.D. - Virginia Commonwealth University

Down syndrome (DS), caused by triplication of human chromosome 21, is associated with intellectual disability and early-onset Alzheimer's disease (AD). By age 40, nearly all individuals with DS exhibit hallmark AD neuropathology, including amyloid-beta (Aβ) plaques, neurofibrillary tangles and basal forebrain cholinergic neuron (BFCN) degeneration. However, these classic features alone do not fully explain the progressive cognitive decline observed in DS + AD. Pharmacotherapeutic strategies, such as cholinesterase inhibitors, have failed to produce meaningful results in ameliorating AD-related memory loss in DS patients. As AD-related pathology advances in individuals with DS, BFCNs—key contributors to memory formation in humans—continue to degenerate, and the efficacy of these treatments diminishes.

Neuronal functional stability is governed by diverse subtypes of potassium (K^+) channels, which play a crucial role in regulating neuronal excitability in many neurons, including BFCNs. Using an ex vivo mouse model of AD, we demonstrate that BFCN hyperexcitability is induced by chronic exposure to soluble, oligomeric forms of Aβ and is accompanied by functional downregulation of large-conductance (BK-type) and small-conductance (SK-type) K^+ channels. In complementary findings using the Ts65Dn mouse model of DS, we show that BFCNs from trisomic animals exhibit a hyperexcitability phenotype compared to age-matched disomic littermates.

Using the Ts65Dn mouse model of DS, this study will:

1. Investigate whether trisomy-induced BFCN hyperexcitability results from functional alterations in specific K^+ channel subtypes that regulate BFCN intrinsic excitability.
2. Determine whether trisomy-induced BFCN hyperexcitability stems from transcriptional pathway alterations, including those regulating K^+ channel expression.

Andrew A. George, Ph.D., is an Assistant Professor in the Department of Pharmacology and Toxicology at Virginia Commonwealth University (VCU). He received his B.S. in Biochemistry from Arizona State University and his Ph.D. in Neuroscience from the University of Texas at Austin. Dr. George completed his postdoctoral training in the Department of Biology and Biomedical Sciences at Washington University in St. Louis before joining the faculty at VCU. His research employs electrophysiological techniques, classical neuropharmacology and transgenic animal models to investigate why specific neuronal populations (e.g., BFCNs) and neural circuits implicated in learning and memory are especially susceptible to early functional dysregulation in amyloidogenic disease phenotypes such as Alzheimer's disease (AD) and Down syndrome with Alzheimer's disease (DS + AD).

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LEVERAGING STRUCTURED RETRIEVAL PRACTICE USING A CARE PARTNER-CENTERED INTERVENTION TO IMPROVE KNOWLEDGE, MASTERY, AND WELL-BEING

Robert Ariel, Ph.D. - Virginia Wesleyan University

Caring for someone with dementia is both challenging and stressful. Family members and friends who provide this care often lack knowledge about how to manage difficult behaviors, which adds to their stress and can affect both their well-being and the quality of care they provide. While educational resources exist for dementia care partners, most are not designed using evidence-based learning methods.

This study will test a new approach called structured retrieval practice (SRP), which helps people learn and retain information more effectively by recalling it over time with corrective feedback. Our research will compare SRP to traditional reading-based education. Care partners will learn about managing dementia symptoms, coping strategies and self-care techniques. We will then assess how well they remember this information after 2 days, 2 weeks and 2 months.

We hypothesize that care partners using SRP will:

1. Retain important information longer
2. Feel more confident in their caregiving abilities
3. Experience less stress
4. Better manage their loved one's behavioral symptoms

The study will also evaluate whether this intervention is practical and acceptable for care partners in real-world settings. If successful, this approach could be integrated into existing educational programs and digital tools to better support families caring for individuals with dementia.

Robert Ariel, Ph.D., is an Associate Professor of Psychology at Virginia Wesleyan University. His research focuses on understanding and improving self-regulated learning across the lifespan. His recent work draws on findings from the science of learning to develop more effective educational tools for informal care partners of individuals living with dementia. He has published extensively on memory, metacognition, and aging, and is committed to translating psychological research into practical solutions that enhance education, confidence and quality of life.

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UNDERSTANDING THE CONTRIBUTION OF PERINEURONAL NET CHONDROITIN-6-SULFATE TO SOCIAL MEMORY LOSS IN ALZHEIMER'S DISEASE

Elise Cope, PhD - University of Virginia

Social memory deficits—the inability to recognize or remember familiar individuals—are a debilitating symptom of Alzheimer's disease (AD), significantly reducing quality of life. Yet, the mechanisms underlying these deficits remain poorly understood. Rodent studies identify the hippocampal CA2 region as a key hub for social memory. This region is rich in perineuronal nets (PNNs), specialized extracellular matrix structures that regulate neuronal plasticity.

Our previous work showed that disrupting CA2 PNNs impairs social memory in mice. PNN loss has also been observed in both AD patients and mouse models. Notably, restoring CA2 PNNs improves social memory in AD mice, suggesting PNNs as a potential therapeutic target.

PNNs are composed of chondroitin sulfate proteoglycans, which carry sugar chains sulfated at the 4- (C4S) or 6- (C6S) carbon positions—each influencing plasticity differently. Elevated C6S levels have been reported in AD patients and correlate with disease progression, but their functional role remains unknown.

We will investigate how PNN loss and sulfation patterns contribute to social memory deficits in an AD mouse model. First, we will assess changes in CA2 PNN sulfation, focusing on C6S, using immunolabeling and confocal microscopy. Then, we will test whether reducing C6S—via enzymatic digestion or viral inhibition—restores social memory in AD mice.

These studies aim to clarify the role of sulfation in PNN dysfunction and social memory loss, potentially guiding new therapeutic strategies for AD.

Elise Cope, Ph.D., is an Assistant Professor in the Department of Neuroscience and the Brain Immunology and Glia Center at the University of Virginia School of Medicine. She earned a B.S. in Biological Science and a Ph.D. in Biomedical Sciences from Florida State University, where her dissertation focused on using zinc to improve behavioral outcomes following traumatic brain injury. She then completed postdoctoral training in Dr. Elizabeth Gould's lab at Princeton University's Neuroscience Institute, where she studied hippocampal plasticity and circuitry underlying cognitive function. In 2022, Dr. Cope launched her independent research program at the University of Virginia. Her lab investigates how structural plasticity in the hippocampus contributes to social memory circuits and whether targeting these mechanisms can alleviate social memory deficits in conditions such as Alzheimer's disease.