



THE
UNIVERSITY
OF RHODE ISLAND
GEORGE & ANNE RYAN
INSTITUTE FOR
NEUROSCIENCE

INAUGURAL REPORT 2018

Founded in 2013, the Ryan Institute brings together a pioneering team of scientists widely respected for their contributions to under-explored areas of neurodegenerative disease research. Joining forces with a closely-linked network of clinicians and innovators, the Ryan Institute has moved quickly in supporting bold new paths toward the prevention and treatment of neurodegenerative disease.





From the Executive Director

In diseases where brain cells die, it has always made sense to me to investigate the factors responsible for keeping those cells healthy. My own research for the past 30 years has focused on the role blood vessels might play in neuronal cell death. (You can read more about this on page 9.) But Alzheimer's disease is a complex disorder, and the cells and processes in the brain are highly interconnected. The enigma that is Alzheimer's won't be solved by a single breakthrough. At the Ryan Institute, I am fortunate to collaborate with scientists who are pursuing other under-explored avenues of investigation.

Last year, a landmark study by the Lancet Commission reported significant findings on lifestyle factors connected to brain health. Many of these factors support our overarching focus at the Ryan Institute on the roles of the vasculature, inflammation, and the immune system in neurodegeneration. More important, they underscore the proactive role we can all take in preventing or stalling dementia.

On these pages, I am pleased to share some accomplishments to date. We've recently welcomed three notable scientists to our team, as well as biotech startup EPhysBio, the second public-private partnership we've helped to foster at URI. In February, we joined forces with the national Alzheimer's Prevention Registry to engage the community in Alzheimer's research, and the success of our second annual URI Brain Fair in March shows that there is great public interest in learning about brain health. While there is much work ahead, every day I see more reasons to be hopeful than fearful in the fight against neurodegenerative disease.

Paula Grammas
Executive Director

Thomas M. Ryan Professor of Neuroscience

MILESTONES

November 2013

URI announces a \$15 million gift from Tom '75 and Cathy Ryan to fund the creation of the George & Anne Ryan Institute for Neuroscience.

November 2015

In a ceremony at the Rhode Island State House attended by Governor Gina Raimondo, Paula Grammas is presented as the inaugural Executive Director of the Ryan Institute.

Summer 2016

Publication of the first research article with Ryan Institute affiliation, a study of the link between early-life lead exposure and gene regulation in an animal model of Alzheimer's disease. Authors include College of Pharmacy Professor Nasser Zawia and Ryan Institute Associate Director William Renehan.

October 2016

First meeting of LIFT, the Lifestyle Interventions to Fight and Treat dementia group. (See page 21 to learn more.)

March 2017

Nearly 500 families and visitors attend the first URI Brain Fair, hosted by the Ryan Institute. Activities include a simulation of dementia, workshops on music and the brain, and walk-through 3-D brain projections.

Fall 2017 – Spring 2018

Ryan Institute's first new faculty arrive: William Van Nostrand, Katharina Quinlan, and John Robinson. Together they bring a combined \$7 million in research grant funds to URI.

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**“We need to back scientists
with different, less mainstream ideas.”**

Bill Gates, on Alzheimer’s research

DESIGN
Lakuna Design, lakunadesign.com

PHOTOGRAPHY
All photography by Kathleen Dooher (kdoohier@me.com)
unless otherwise noted.



The Dose Effect

Professors William Van Nostrand and John Robinson are taking on unanswered questions about the amount and level of exercise that can make an impact on brain health.

For years, Bill Van Nostrand and John Robinson have had a friendly debate. Between the two of them—an Iron Man and a 5K competitor, respectively—whose form of exercise is healthier?

Anyone who's read about the brain-healthy benefits of exercise might share their curiosity. "Most studies haven't been designed to determine the duration, intensity, or forms of exercise that are most beneficial," says Robinson. "How much exercise do you need? Does someone have to run on a treadmill every day, or is it enough to go out for a brisk walk? Does it make a difference if you've been sedentary your whole life? We started talking about how we could help tease apart these questions that people want to know."

Van Nostrand and Robinson are longtime collaborators, who both moved their labs to URI from Stony Brook University over the past academic year. Van Nostrand is noted for being the first to purify and characterize the precursor protein that generates amyloid-beta, which forms plaques in the brain associated with Alzheimer's. Robinson, a behavioral neuroscientist, brings key expertise to URI in cognitive mechanisms and translational research. Their work together has investigated brain function in cerebral amyloid angiopathy, a condition that contributes to Alzheimer's, as well as the role of lifestyle interventions, including brain training, in Alzheimer's disease.

And perhaps resolving those exercise questions. In 2015, they began work on a \$1.3 million grant to study "doses" of exercise in connection to Alzheimer's disease, says Van Nostrand. "This is work that hasn't been done yet to try to isolate the factors that may have a role in reducing pathology or [cognitive] impairments." By breaking down the types and durations of exercise that are most impactful in regions of the brain associated with memory, learning, and sensory input, their work could be the first to offer clear guidance on the role of exercise in preventing or protecting against neurodegenerative disease.

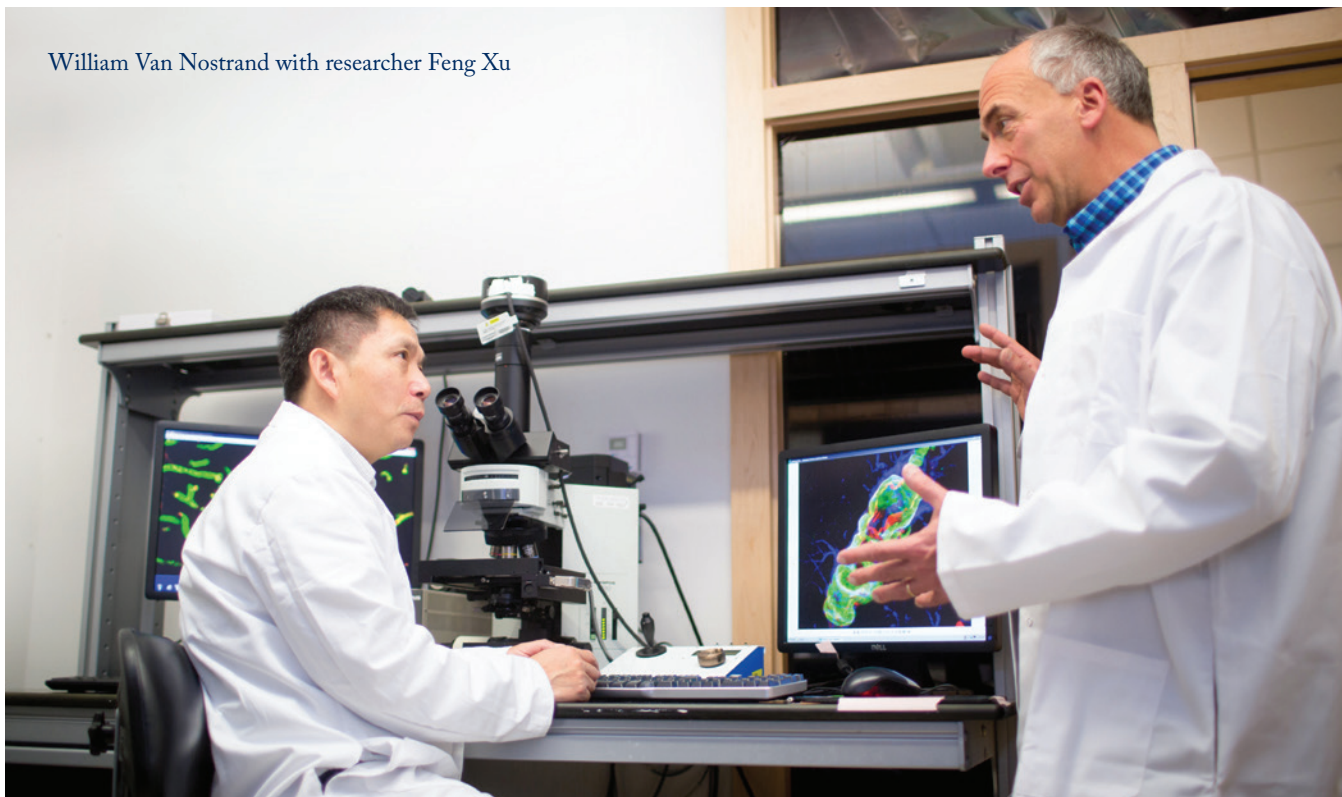
They expect to publish the results of these studies in 2019. Says Robinson, "By more clearly evaluating the impacts of exercise, we hope we can help people better understand how to take a proactive role in their brain health."



Pictured: Professors William Van Nostrand (left) and John Robinson (right)

Photo: Ryan Institute for Neuroscience

William Van Nostrand with researcher Feng Xu



The Search for Biomarkers

Neurodegenerative diseases like Alzheimer's have been difficult to treat in part because we still lack a way to identify them in their early stages—making the search for biomarkers one of the most critical areas of research. Van Nostrand's focus on understanding the molecular basis of neurodegenerative diseases has provided new insights into early stage biomarker development. Recently, his lab identified unique structural signatures of amyloid that accumulates around brain blood vessels in Alzheimer's, which could provide important information about how the disease develops and progresses. "Most of the interventions that have failed are targeting pathologies in the late stages of Alzheimer's," says Van Nostrand. "Biomarker studies of early stage disease are key for clinical diagnosis and eventually an intervention that can stop the disease."



Steps Forward

Assistant Professor Katharina Quinlan investigates the mysteries of ALS.

Katharina Quinlan has long been fascinated by the study of motor control. “Many aspects of our nervous system function—like happiness, dreams, and memories—are difficult for scientists to measure in all their richness,” she says. “In contrast, movement is a complex neural activity that can be measured and quantified.”

Quinlan studies how spinal neurons change and deteriorate in neurodegenerative diseases and disorders such as amyotrophic lateral sclerosis (ALS), spinal muscular atrophy, and cerebral palsy. Last fall, she arrived at URI from Northwestern University, where part of her research focused on potential biomarkers that could help in the diagnosis and treatment of ALS.

A still-mysterious disease that usually develops spontaneously in adulthood, ALS is difficult to diagnose and treat, in part because initial symptoms don’t appear until the disease has already advanced. Quinlan’s research has helped identify compensatory changes that may be taking place early in ALS—and masking signs of the disease. “We see the largest, fast-fatigable motor units will begin to deteriorate, but others will step up to reinnervate the abandoned muscle fibers,” she says. “We are looking to find out how various neurons could be involved in this process.”

She hopes her work could aid in detection and diagnosis, using electromyograms during walking or running to look for early physiological markers of ALS. “No one has used EMGs in this way,” she says. “I think there is potential to make them part of regular stress-test screening to get a better understanding of the disease.”

Q&A

Paula Grammas

The executive director of the Ryan Institute explains her research into the role of blood vessels in Alzheimer's disease.

“The puzzle of Alzheimer's won't be solved without exploring the multiple factors that are likely to play a role in the development of this complex disease.”



When Paula Grammas first began her investigation into the role blood vessels play in the development of Alzheimer's nearly 30 years ago, few scientists were exploring research avenues beyond the prevailing amyloid hypothesis (see page 12). Now, as increasing evidence supports the link between heart and brain health—and major research institutions have begun shifting gears to look at new targets—Grammas is no longer an outlier in the field, and the gains she has made in the past decades toward understanding the complexity of brain cell death have moved her closer to answering some critical questions.

What is the role of the blood vessels in the brain?

Blood vessels help to provide the controlled environment brain cells need in order to survive. The blood brain barrier, which is mostly made up of endothelial cells that line the blood vessel walls, helps to regulate what goes in and out of the brain. In a healthy brain, the blood vessels transport nutrients in and transport waste products out. The endothelial cells also produce proteins that support brain function.

What happens to the blood vessels in patients with Alzheimer's?

In Alzheimer's, we see that the blood brain barrier gets leaky, and the cells in the blood vessel walls—the endothelial cells—become over-activated or disturbed. Instead of making proteins that support brain function, they produce inflammatory proteins and neurotoxins that are damaging to the brain cells. It starts a vicious cycle, where these toxic molecules in turn damage the blood vessels, leading them to produce more toxic species.

What causes this over-activation?

A number of factors may injure the blood vessels and cause them to become over-activated, such as high cholesterol, high blood pressure, or high blood glucose. We know, for example, that diabetes enhances risk for developing Alzheimer's, and we know that there is blood vessel dysfunction in diabetes. This is an area that we're actively exploring.

Is there a way to stop the over-activation cycle once it starts?

In a healthy brain, the blood vessels are quiet and only become activated as a normal response to a signal or injury, such as an infection. This process becomes dysfunctional when they don't turn off and return to that quiescent state. Our research has shown that thrombin, a protein that has many functions, including in blood clotting, might be one of the key drivers in this process. It becomes over-produced, and causes inflammation that keeps the blood vessels over-activated to create the vicious cycle.

Could targeting thrombin lead to a treatment for Alzheimer's disease?

We have seen that thrombin is produced by blood vessels in Alzheimer's disease but not in the healthy brain. We also find that when we inhibit thrombin in animal models of Alzheimer's, we have been able to diminish some of the brain changes we see in Alzheimer's disease. Our next step is to test a thrombin inhibitor in people with Alzheimer's.

Why hasn't this target been tried already?

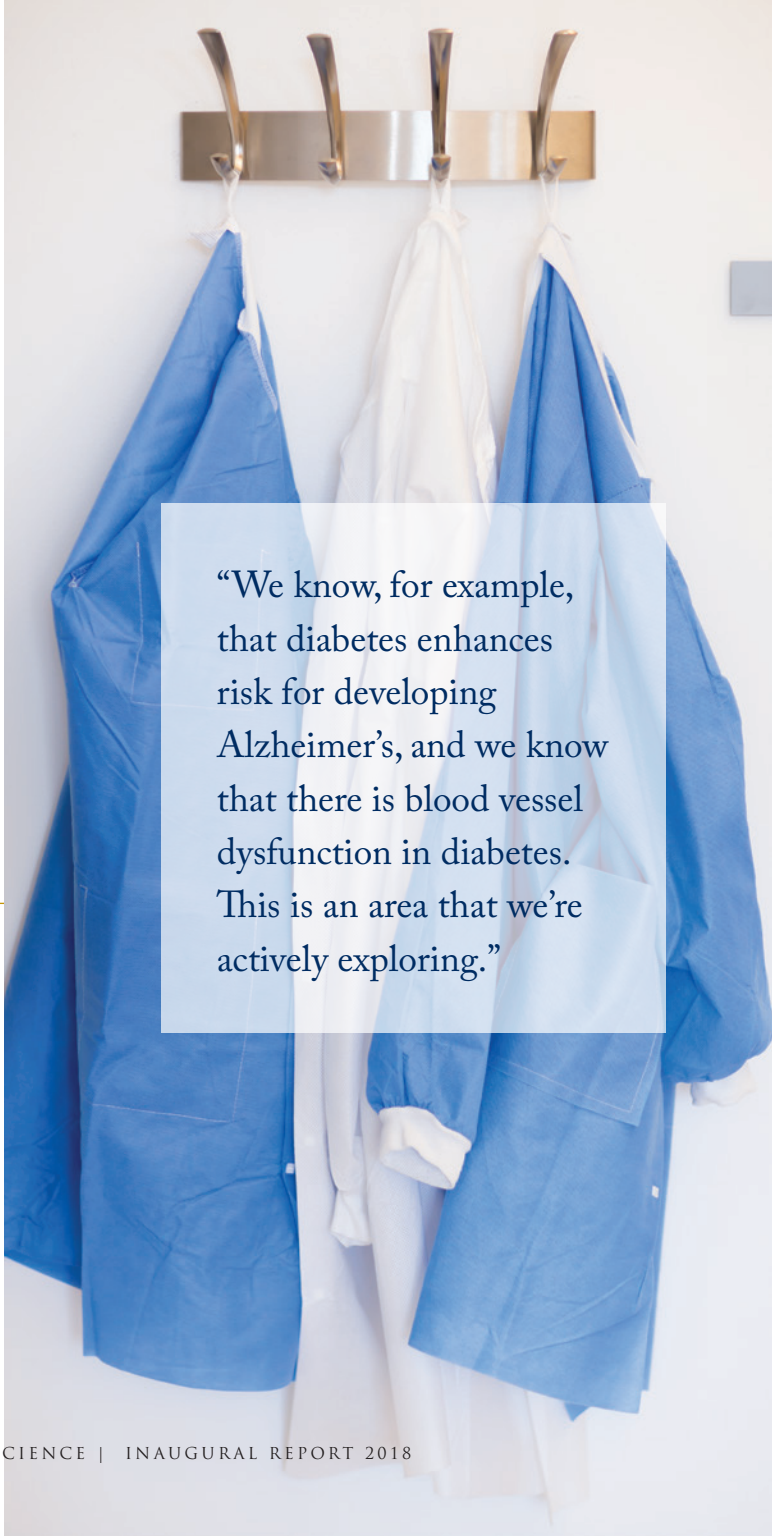
For so long, scientists have been focused on the amyloid cascade hypothesis. When you test a hypothesis and it doesn't work, it should force you to go back and re-examine, but that hasn't

happened with amyloid. There has been so much invested in trying to prove this theory, other hypotheses have not been sufficiently explored.

How did you first come to focus on the role of blood vessels in Alzheimer's disease?

In Alzheimer's, brain cells die. Either there is something intrinsically wrong with these brain cells, or something in their environment is killing them. When I started my career, there weren't a lot of people looking at the brain nerve cell micro-environment, and it seemed logical to me that it was an area we needed to know more about. Now there is more interest, and I'm glad to see that there is more support for this area of research. The puzzle of Alzheimer's won't be solved without exploring the multiple factors that are likely to play a role in the development of this complex disease.

Paula Grammas is Thomas M. Ryan Professor of Neuroscience and a professor of biomedical and pharmaceutical sciences. She was recently elected to the American Association for the Advancement of Science Circle of Fellows in recognition of her pioneering research.



“We know, for example, that diabetes enhances risk for developing Alzheimer's, and we know that there is blood vessel dysfunction in diabetes. This is an area that we're actively exploring.”

Neurodegenerative Disease by the Numbers



1 in 10

Americans over 65 with Alzheimer's dementia



123%

Increase in Alzheimer's mortality between 2000 and 2014



65

Every 65 seconds, someone new in the U.S. develops the disease.



1 of 10

Alzheimer's is the only disease among the 10 leading causes of death in the U.S. without a cure or treatment.



\$277 billion

Total health care costs in the U.S. for Alzheimer's and other dementias in 2018



1.5+ million

Americans living with other neurodegenerative diseases, including Parkinson's, multiple sclerosis, ALS, and Huntington's



Exploring New Targets in Alzheimer's Research

The amyloid cascade hypothesis has dominated research for nearly three decades, based on the theory that abnormal build-up of amyloid-beta in the brain is a primary cause of Alzheimer's. Clinical trials based on this hypothesis so far have failed to produce any disease-modifying treatments. "There is an increased acceptance of the fact that Alzheimer's is not a single disease but a group of pathologies," says Executive Director Paula Grammas. "It is critical that we support investigations into the multiple factors that are likely to play a role in the disease."

Sources: 2018 Alzheimer's Disease Facts and Figures (Alzheimer's Association), National Institutes of Health, Michael J. Fox Foundation, Harvard University

PATHFINDERS

Meet six of the Ryan Institute faculty looking into under-explored factors in neurodegenerative disease.



GUT INSTINCTS

Navindra Seeram

Associate Professor of Biomedical and Pharmaceutical Sciences

First things first: There are more than 100 trillion bacteria in your gut, and they are integral to your health. “If your gut isn’t right, you’re not right,” says Navindra Seeram, associate professor of biomedical and pharmaceutical sciences. From a well-functioning immune system to mental clarity, much depends on having a balanced, diverse population of microbiota inside you. “Plant foods help keep the balance and ensure that good bacteria thrive,” he says. Think of your microbiota, then, as a garden you cultivate—or neglect. While processed foods are major culprits in destroying your gut’s healthy balance, says Seeram, they also impair its ability to get the most nutrition from the foods you eat. “Colorful fruits and vegetables, roots, herbs, berries—foods from plant sources not only shift your microbiota, but they can also help train them to convert plant foods into beneficial compounds.”

In his current research, Seeram investigates those beneficial compounds, specifically how plant foods and natural products can help prevent or delay neuroinflammation and other chronic illnesses. Most recently, he received a grant from the U.S. Department of Agriculture to explore the anti-inflammatory properties of maple syrup with fellow associate professor Angela Slitt, and also published new research on the brain-protective effects of berries. “There’s a reason humans see color,” says Seeram, on filling your diet with a rainbow of fruits and veggies. “It’s so simple but profound. If you choose the right foods, you give yourself the best possible advantage as you age.”

**“Plant foods help keep the balance
and ensure that good bacteria thrive.”**



NEW CONNECTIONS

Yalda Shahriari

Assistant Professor of Biomedical Engineering

“We know that symptoms of Parkinson’s disease are reduced during deep brain stimulation,” says Assistant Professor Yalda Shahriari. But there are questions as to why. In her Neural Processing and Control Lab at URI, Shahriari investigates brain activity associated with neurodegenerative diseases such as Parkinson’s and ALS. As part of her research, she is working toward solving one of the perplexing puzzles of Parkinson’s disease—exactly why deep brain stimulation (DBS) helps reduce motor symptoms in patients who receive treatment. A surgically implanted device that delivers electrical pulses to specific regions of the brain, DBS can alleviate the tremors, rigidity, stiffness, and impaired movement of Parkinson’s patients. But although electrical stimulation has been used in therapies for a number of brain diseases, its mechanism in Parkinson’s remains unclear. “When we record signals from the regions of the brain that DBS targets, we can see changes in the brain’s connectivity patterns,” says Shahriari. “These signatures can ultimately help us to understand what is happening in the disease.” Her research also could pave the way for a more effective, targeted DBS treatment—currently, the device delivers ongoing treatment that is considered “blind” to patients’ needs. “In the future, we would like to be able to incorporate kinematic information from a patient’s limbs,” she says. “That’s the goal.”

The NeuralPC lab also focuses on using brain-computer interface (BCI) to develop better assistive technologies and discover insights into the brain. “Our lab is working to identify biomarkers not only with the goal of developing new therapies, but also developing personalized communication systems for patients who cannot communicate,” Shahriari says. “What if it were possible for a computer to write by reading from your brain?”

A portrait of Jodi Camberg, a woman with long, light brown hair, smiling. She is wearing a dark blue collared shirt. The background is a soft, out-of-focus indoor setting with warm lighting.

ON GUARD

Jodi Camberg

Assistant Professor of
Cell and Molecular Biology

There's a reason molecular chaperones, which help maintain equilibrium within our cells, are known as the guardians of the proteome. If this chaperone system goes awry, there is an impairment of the normal balance of functional proteins, says Assistant Professor Jodi Camberg. "For neurodegenerative diseases that are also protein misfolding disorders, the normal chaperone and clearance system that maintains [this balance] has become disrupted."

In diseases such as Alzheimer's, Parkinson's, and Huntington's, it remains unclear why certain proteins—such as amyloid-beta in Alzheimer's or alpha-synuclein in Parkinson's—abnormally accumulate and form hallmark clusters in the brain associated with the death of neurons. Camberg's research considers how chaperones, which perform quality control in our cells, could be involved in that process.

True to their name, chaperones assist and stabilize proteins as they fold or unfold into the structures that allow them to perform their functions. They also help fix, recover, or degrade proteins that have become dysfunctional. Camberg is investigating why or how the chaperones may begin to fail at these tasks, allowing harmful protein aggregates to form and persist. "If there is an imbalance of chaperones that leads to accumulation of protein aggregates, it may be possible to restore chaperones to the system to alter accumulation rate or abundance of protein aggregation," she says. She is also investigating whether chaperones could play a therapeutic role in dissolving aggregates that have formed. "Chaperones are involved in every cellular process," says Camberg. "By studying the biochemistry of these protein remodeling systems, we can better understand how protein misfolding diseases develop, and identify potential therapeutic targets."

EARLY IMPACT

Nasser Zawia

Professor of Biomedical
and Pharmaceutical Sciences

Dean of the Graduate School



“For the most part, you get your set of neurons for a lifetime,” says Nasser Zawia. As a leader in brain epigenetics (the study of how, when, and why combinations of genes are turned on and off to make proteins), Zawia was among the first to show how early-life exposure to lead may be connected to the development of Alzheimer’s dementia. “When other tissues or organs are impacted by chemicals or toxins, the cells turn over,” says Zawia. “But if your brain cells are impacted during development, you can permanently reprogram gene expression.”

This “critical window” in development, Zawia says, means not only that environmental factors, such as prenatal and early childhood exposure to toxins, can play a role in causing disease later in life, but also that positive behaviors—such as a brain-healthy diet—can have a meaningful impact on preventing disease. This year, Zawia’s lab also published research investigating the unique programming of Alzheimer’s genes. “We believe that Alzheimer’s genes might be resistant to global trends that rewrite the destiny of the genome in early life, but instead follow a special reprogramming pathway,” says Zawia, who believes this programming may be related to a still-unknown adaptive mechanism. “It is our most detailed understanding to date of this unique feature in the epigenetic programming of Alzheimer’s genes, and could help us move closer to finding a potential therapeutic target.”

A portrait of Lisa Weyandt, a woman with long brown hair and bangs, wearing a black top and gold earrings, smiling slightly. The background is a blurred stone wall.

CROSSING MINDS

Lisa Weyandt

Professor of Psychology

What can we learn about Alzheimer's or Parkinson's disease from teenagers with attention deficit/hyperactivity disorder? Professor of Psychology Lisa Weyandt studies the impact of pharmacological and non-drug interventions on ADHD, with a specific focus on how these interventions affect cognitive skills and executive function. "For example, how does treatment affect their ability to plan, their organizational skills and ability to stay focused, or their ability to inhibit impulsive responding?" says Weyandt. "My research looks at some of the same aspects of brain function that are affected by neurodegenerative diseases."

Weyandt is a leading expert in ADHD in young adults, particularly in investigating the health risks and impacts of medications or

stimulants that are intended to manage symptoms. Much of her work has focused on how prescription drugs, such as Adderall, impact cognitive performance or affect the regulation of dopamine, a neurotransmitter that may play a role in both ADHD and Parkinson's disease. "The more we understand about executive function in healthy individuals and in other types of pathology, the better our understanding of how executive function is affected in neurodegenerative diseases," she says. Recently, Weyandt co-authored a study on the role of diet as a potential risk-reducing factor for Alzheimer's, drawing on her own research in examining lifestyle interventions in ADHD. "If we can find a way to treat or prevent these disorders and diseases of the brain by making simple changes to the way we live, that is some of the most important work we can do."

Photo: Beau Jones, *Momentum* magazine

FREED SPEECH

Leslie Mahler

Associate Professor
of Communicative Disorders

Director, Interdisciplinary
Neuroscience Program



For 25 years, Leslie Mahler worked as a hospital-based speech language pathologist, treating adults with neurological disorders such as brain injury, Alzheimer’s disease, stroke, and cerebral palsy—a background that forms a meaningful foundation for her neuroscience research. “Each person presents a unique set of challenges,” says Mahler, who maintains a clinical practice in connection to her work at URI. “Many have multiple communication deficits or co-existing medical problems. Combining clinical experience with my research allows me to better identify the components of treatment that lead to an effective outcome.”

In her current research, Mahler investigates how principles of motor learning, such as intensive practice, salience, and repetition, can be applied in treatment for people with neurological diagnoses to help them build or regain their communication function. She also conducts the LOUD Crowd—a support group for people with Parkinson’s disease that focuses on communication strategies for social participation and on the role of diet and health. Additionally, she collaborates with faculty in biomedical engineering in her research on the effectiveness of deep brain stimulation on speech, as well as on the development of speech data technology to improve treatment outcomes. All together, her multi-faceted approach not only reflects the complexity of understanding and treating brain diseases such as Alzheimer’s, but the potential impact treatment can make. “To date, we don’t have a cure for dementia or these progressive neurodegenerative diseases,” she says. “But we do know that we can help maximize the level of communication function to help people with those diagnoses have the best possible quality of life.”



NEWS HIGHLIGHTS

October 2017

Biotech startup EPhysBio arrived at URI to become the second private-public partnership based at the University. EPhysBio specializes in screening potential drug therapies by evaluating their effects on brain activity (EEG biomarkers) and diseases of the central nervous system.

November 2017

A \$150,000 grant from Cure Alzheimer's Fund supports the investigation of the role of vascular activation (see page 9) in the development of Alzheimer's disease. Subsequent funding contingent upon first-year progress will support additional research.

Executive Director Paula Grammas was elected to the American Association for the Advancement of Science Circle of Fellows. She was inducted at the AAAS Annual Meeting in February 2018.

January 2018

Research begins on a project investigating the connection between diabetes and Alzheimer's disease, funded by a \$200,000 gift from the Murray Family Foundation.

A new research agreement between Pfizer, Inc. and MindImmune Therapeutics, Inc., a private drug discovery and development firm based at URI, supports research into the role of peripheral immune cells in neurodegenerative disease.

February 2018

The Ryan Institute announced a new partnership with Banner Alzheimer's Institute to help drive enrollment in the Alzheimer's Prevention Registry, a national registry of nearly 300,000 adults aged 18 and older who are interested in learning about Alzheimer's studies and prevention efforts in their communities. For more information, see page 23.

March 2018

Distinguished neuroscientist Peter Snyder, former senior vice president and chief research officer at Lifespan, began new role as vice president for research and economic development at URI. Snyder will continue his work in Alzheimer's research as a Ryan Research Professor of Neuroscience.

FOCUS ON PREVENTION

It was major news last year when one of the world's foremost medical journals, *The Lancet*, identified nine lifestyle factors that can reduce risk of dementia up to 35%—meaning one third of Alzheimer's dementias could be preventable. "The recent findings not only support the connection between heart and brain health, but they also move us toward a greater focus on prevention," says Ryan Institute Associate Director William Renehan. "It is now becoming increasingly clear that a huge portion of our risk of developing dementia is under our control."

"Dementia is by no means an inevitable consequence of reaching retirement age, or even of reaching the ninth decade. Lifestyle factors might reduce, or increase, an individual's risk."

– The Lancet Commissions, July 2017

KEY FACTORS IN BRAIN HEALTH



EDUCATION: Lower educational level leaves less "cognitive reserve" to support brain function in the presence of disease.



HEARING LOSS: Hearing loss increases long-term risk of dementia, although the mechanism underlying this relationship remains unclear.



EXERCISE AND PHYSICAL ACTIVITY: Older adults who exercise are more likely to maintain cognition than those who do not exercise.



HYPERTENSION, TYPE 2 DIABETES, & OBESITY: These combined vascular factors exacerbate inflammation, insulin resistance, and high blood sugar associated with Alzheimer's dementia.



LACK OF SOCIAL CONTACT: Social isolation increases risk of hypertension, heart disease, and depression, which are risk factors for dementia. The report also suggests that social isolation may result in cognitive inactivity, linked to cognitive decline.



DEPRESSION: Late-life (older than 65 years) depression affects two factors linked to the development of dementia: the release of stress hormones and a decrease in neuroplasticity, the brain's ability to change and adapt with age.



SMOKING: Cigarette smoke contains neurotoxins and is also a cause of heart disease, linked to dementia.

*These statements are based on The Lancet Commission conclusions published in "Dementia prevention, intervention, and care," The Lancet, July 2017.

LIFT Dementia

The Lifestyle Interventions to Fight and Treat Dementia group (LIFT) at URI brings together faculty and health professionals from various disciplines to share knowledge and foster potential research collaborations on how to slow or prevent the onset of dementia through lifestyle changes such as diet and exercise.

The group was formed in 2016 by William Renehan, associate director of the Ryan Institute, and Catherine Taylor, the Ryan Institute's senior advisor for policy, partnerships, and community engagement. "One of the most encouraging developments in the past few years has been the increased evidence to support the role of lifestyle in preventing dementia, but there is a great amount of variability in the methods that have been used to conduct these studies," says Renehan. "It has made it difficult to interpret the data in a meaningful way."

Renehan points to studies on diet and exercise conducted without input from kinesiologists, nutritionists, gerontologists, and others. "At URI, we have faculty with the experience and knowledge that is often missing from these studies," he says. "We saw an opportunity to bring together people with critical expertise across disciplines so that we can determine ways to help fill some of the important gaps in the literature."

Last year, the Ryan Institute granted \$10,000 in seed funding to each of three projects proposed by faculty in the LIFT group:

Public understanding of the connection between behavior and brain health: a survey led by Colleen Redding, research professor at URI's Cancer Prevention Research Center, to determine public knowledge and understanding of the impact of lifestyle behaviors on brain health.*

URI-affiliated senior housing community: a feasibility study led by College of Nursing Professor Patricia Burbank to explore a university-based living and learning retirement community.*

Gait and cognitive decline: a pilot study led by Kim Fournier and Christie Ward-Ritacco, assistant professors of kinesiology, to investigate gait as a biomarker for early stages of dementia.

*Funded in collaboration with the URI Institute for Integrated Health and Innovation "Big Ideas in Health" initiative.

Outreach Highlights

Brainy Day

Nearly 700 kids and adults turned out for the second annual URI Brain Fair in March, where they explored 27 interactive exhibits on the human brain. Among the day's highlights were a brain-computer interface zone, featuring technological innovations from faculty and students in biomedical engineering; games to educate on a brain-healthy lifestyle; a giant neuron that fired with audience participation; a 3-D tour of the human brain; and a virtual-reality dementia tour. The day also included lectures, live music, and a mid-day brain circus (pictured) by performer Marvin Novogrodski '84.



Photo: Michael Salerno.

Joining Forces

Nearly 80 percent of Alzheimer's studies are delayed by lack of participants. The Ryan Institute has partnered with the national Alzheimer's Prevention Registry to help connect people to studies where they live, including clinical trials at Rhode Island Hospital and Butler Hospital.

1. Join at endalznow.org
2. Choose the study opportunities and research news you want to receive.
3. Participate, if you wish, in the studies for which you're eligible and that interest you. Some focus on lifestyle factors such as diet and exercise, or can be completed online. Others are trials of investigational drug treatments.

For more information, visit ryaninstitute.uri.edu

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University of Southern California

Thank You To Our Supporters

By establishing the George & Anne Ryan Institute for Neuroscience, Tom and Cathy Ryan committed to creating a research entity that would become a center of excellence in the fight against neurodegenerative disease. Their \$15 million gift, the most generous in the University's history, has made it possible to recruit a team of leading scientists who are dedicated to fulfilling the mission of the Institute as a tribute to the Ryan family and all those who have been personally touched by these debilitating diseases.

Equally inspiring are the many individuals who joined them in supporting this critical work. We are grateful to recognize all those who have made a commitment of \$250 or more through **June 30, 2017**.

\$1 million and above

Thomas M. and Cathy Ryan

\$100,000 – \$999,999

MindImmune Therapeutics Inc.
Murray Family Charitable Foundation

\$10,000 – \$99,999

Patricia Asquith
James and Janet Field, Jr.
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and Mark Mainelli

\$1,000 – \$9,999

James and Deborah Baldwin
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and Cynthia Trieman
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