



MCG
Medicine

SPRING | SUMMER 21

at AUGUSTA UNIVERSITY

GREY MATTERS

MCG Medicine

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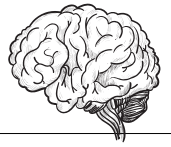


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FOOD FIGHT

Plant-based diet protects from hypertension, preeclampsia

(from left) Drs. John Henry Dasinger, Justine M. Abais-Battad and David L. Mattson



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FROM THE DEAN

You are what we are known for. If you travel Georgia, the Southeast, the nation and beyond, people in the know about medical education know that the Medical College of Georgia is exceptional at educating physicians. The best residency programs want our students, communities want our graduates, and we sure want to keep our students as residents and our graduates as faculty. Educating great physicians is our core, and we just graduated our 185th class. So when you have a time-tested method, do you try to make it even better? The best physicians, you our graduates, have lifelong learning in your DNA so I know your answer would be yes.

The 3+ curriculum at MCG, which began with our Class of 2024, was the most significant change in our significant history. One year in, it's still a work in progress but I believe it is work that will benefit both your medical school and the state we are privileged to serve. We call our new curriculum tailor-made medical education because it gives our students four excellent pathways to choose from. Succinctly put, it offers an expedited three-year medical degree for those who choose to do their primary care training in Georgia, the idea here is to enable more students to select primary care by enabling them to get to know the people and health care needs of our state even better and by reducing financial pressure to make a more lucrative specialty choice when your heart is in frontline care.

Thanks to our first major supporter, Peach State Health Plan (see page 42), along with matching funds included in this year's budget from the state of Georgia, we have established an endowment that will provide scholarship support for these students — a year of scholarship support for each year they commit to serve in Georgia's most medically underserved areas — to ensure that the debt they graduate with is reduced. We have 8 students in the

Class of 2024 who have committed to this expedited 3+ primary care pathway, and vice dean for academic affairs Dr. Doug Miller, who has been a driver of this major initiative, tells me that is among the largest first-time commitments among other medical schools in the nation pursuing some iteration of a three-year program for some of their students. By the way, the Consortium of Accelerated Medical Pathways Program tells us there are more than 17 medical schools in the U.S. and Canada pursuing innovative offerings to address increasing student debt and workforce shortages and the lineup includes some other great medical schools like The Ohio State University College of Medicine and Duke University School of Medicine.

We anticipate that ultimately about 50 students will be enrolled in this pathway at any one time here, which is great, and will require about a \$125 million endowment

"We plan to get to 240 students per class in Augusta, a total class size of 300, with the 60 students per class in Athens, by the time your medical school turns 200 in 2028."

to sustain scholarship support for all three years of medical school.

About 60% of our students already choose a primary care specialty — family medicine, internal medicine, pediatrics, general surgery, OB/GYN, psychiatry and emergency medicine — although, as we know here and everywhere, many who opt for internal medicine are ultimately headed to specialties like cardiology or rheumatology, and those who pick general surgery, may ultimately opt for something like plastic surgery or cardiothoracic surgery. However, we believe this primary care pathway we are creating will help ease the primary care crunch in the dynamic state we are privileged to serve, which is consistently among the top 10 in the nation in population, but still about 40% rural, medically underserved, and

riddled with major health concerns like cardiovascular disease and stroke.

Pathway 2 is a dual degree program, which enables our students to also earn a master's degree in business, public health, education or biomedical science, within the four-year timeframe. Pathway 3 enables a fourth-year focus on advanced residency preparation and Pathway 4, the one I probably would have taken, enables students to focus on research in their final year that will ultimately strengthen their clinical work without doing a full MD/PhD. I think the choices are the best part of this 3+ curriculum and I feel like we have covered the choices our students want to make.

All this work has meant rethinking how we educate, ensuring that all the information we share with our students is directly relevant to their future as great physicians. As our class has grown, we have generally shrunk the size of many

lectures, so that more often there are eight to 12 students learning in small groups. As you can imagine, this is a lot more work, particularly for our basic science faculty and requires more participation by our physician faculty, but we are getting very good feedback from our students. Our faculty being so accessible to students is already part of what we do so well, but this gives them even more individual face time and enables us to better catch early any signs of problems, academic or otherwise. And, like when I was in medical school and maybe missed a few large lectures, it will be harder for our students to miss class when class is so small. You can't be anonymous in this curriculum. And we will still have large lectures, including the stroke lecture I am privileged to give each year along with one



of my patients, they are just a little shorter and more focused, which is probably a good thing too.

Meanwhile, we are increasing our already large medical school class, which is — at this moment — the nation’s 10th largest. We went up by 10 students with the Class of 2024, another notable point for this class since it was the first increase in class size since the second four-year campus, the AU/UGA Medical Partnership in Athens, opened in 2010. We grew from 40 to 50 students per year there and this year we are adding 10 students at the main campus here in Augusta and 10 more in Athens. It has been a while since we grew in Augusta. We went from 180 to 190 students per class in Augusta in 2006, the first increase since 1974. That makes this year’s increase the first at our main campus in 15 years. We plan to get to 240 students per class in Augusta, a total class size of 300, with the 60 students per class in Athens, by the time your medical school turns 200 in 2028.

It has been both an unusual and exceptional six months at your medical school, and I am happy about our progress in so many directions and hope you are too. We have an extraordinary mission and legacy at the Medical College of Georgia and I am eternally thankful for your insight and support as we move ever forward. 🍀

Respectfully yours,

David C. Hess, MD

Dean, Medical College of Georgia

Executive Vice President for Medical Affairs and Integration,

Augusta University

Presidential Distinguished Chair

FROM THE PRESIDENT

Though I could easily fill this column with praise for the hard work, dedication and leadership our faculty, staff and remarkable students have demonstrated during the year-plus fight against COVID-19 — and let me be clear: I am incredibly proud of the work we've done — I'd like to use this space instead to look forward and talk about what I feel is potentially the most exciting research opportunity in the history of this university. It's called TRIBA — the Transdisciplinary Research Initiative in Inflammaging and Brain Aging — and it promises to be a game changer not only for us, but for the health of those we're entrusted to protect.

This exciting new initiative, which came from the faculty, will explore the ways chronic inflammation impacts aging so that we can improve health outcomes for people suffering from Alzheimer's, dementia and other age-related diseases. It will be supported by an aggressive three-year, \$15 million investment to recruit between 15 and 20 new faculty researchers, building on our already significant strengths in aging, neuroscience and vascular biology. A further investment of \$5-\$15 million in research facility renovations will follow.

While these new researchers will primarily reside in the Medical College of Georgia, this cluster hire will be spread over five other colleges and schools — the Dental College of Georgia, the College of Science and Mathematics, the colleges of Nursing and Allied Health Sciences, and the School of Computer and Cyber Sciences — making it a multidisciplinary collaboration in the truest sense. The recruitment steering committee is being led by Dr. Babak Baban, immunologist and associate dean for research in DCG, and Dr. Mark Hamrick, bone and muscle biologist and senior associate dean for research in MCG. Both have already made significant contributions to the study of age-related inflammation. Dr. Baban and a team of investigators from MCG and DCG recently reported on CBD's role in reducing inflammation and plaque levels associated with Alzheimer's (see page 8), and Dr. Hamrick is part of an \$11.1 million Program Project grant from the National Institutes of Health exploring the role oxidation plays in age-related damage to bone, which has implications for brain health as well. Thanks to the hard work of

researchers from several areas of our institution, funding from the National Institute on Aging has increased threefold in the last 10 years, and we want to capitalize on that in a big way.

Why the focus on inflammaging and brain aging? Because seldom do you find something that offers so many benefits to so many people in so many different ways.

First, consider the benefit to our patients, who along with our students are always our first priority. By the year 2030, the entire Baby Boomer generation will be over 65 years old, and while many adults are expected to live to 85 or beyond, there is little evidence to support the idea that these additional years will be better years, given the prevalence of age-related diseases. Though we know that most age-related diseases, including cardiovascular disease and hypertension, involve inflammation, much about inflammation remains unknown, including its potential cause and its role in adverse health outcomes. The related field of brain aging includes specific aspects of inflammation within the brain and how that affects cognitive activities and disease progression.

Concentrating on this area of study allows us to build on our existing research expertise in brain injury and learning and memory disorders to help move the needle on these serious health concerns disproportionately affecting Georgians, particularly minority and underserved populations. In doing so, we can also kick start our research enterprise, which we all know is not where we'd like it to be. Because we anticipate significant opportunities to compete for funding in this area, this initiative will be a major driver in our effort to bridge the \$14 million gap in NIH funding that will bring us into the top 60 ranked medical schools in the country, a goal I want us to achieve by 2030.

The initial faculty for this interdisciplinary cluster hire will be located on the fourth floor of the new College of Science and Mathematics building, located on the Health Sciences Campus. Thanks to efficiencies in other parts of the project, we were able to build out the shelled space with money allocated for the initial project, giving us an additional 20,000-square-feet of much-needed research space. Here, investigators can collaborate with each other in a building primarily populated by undergraduates, giving these young students valuable opportunities to explore a future in research.

I'm excited about the impact this new initiative will have on the health of Georgians and on the research ranking of this university. Expect to hear a lot more about this in the coming months as we work toward making this vision a reality. 🍀

Sincerely,



Brooks Keel, PhD

President, Augusta University

Chair, Augusta University Medical Associates



KIDNEY RISK

Young pregnant women, who appear to have fully recovered from an acute injury that reduced their kidney function, have higher rates of significant problems like preeclampsia and low birthweight babies, problems which indicate their kidneys have not actually fully recovered.

Now MCG scientists have developed a rodent model that is enabling studies to better understand, identify and ideally avoid this recently identified association, they report in the *Journal of the American Society of Nephrology*.

“We are talking about a population of young women that we usually think about as protected and healthy, and they are not if they have had a kidney insult,” says Dr. Jennifer C. Sullivan, pharmacologist and physiologist in the Department of Physiology and interim dean of The Graduate School at Augusta University.

Acute kidney injuries, or AKIs, result from the rapid, temporary loss of adequate oxygen-rich blood to the organ that constantly filters our blood of toxins and resorbs essentials like water, glucose and potassium. An AKI can result from a literal blow to the kidney, like a car accident, from pharmaceuticals like the chemotherapy drug cisplatin, or from a major surgery or sickness that puts you in intensive care, like COVID-19, which is known to have a direct effect on the kidney.

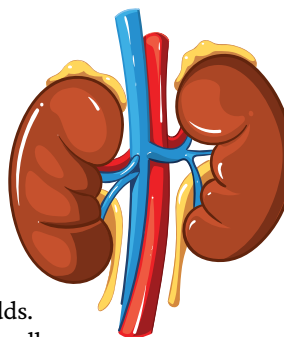
The incidence of AKI has been on the increase, and the COVID pandemic very likely will accelerate that, says Dr. Ellen E. Gillis, senior postdoctoral fellow in Sullivan’s lab and the study’s corresponding author. A COVID-related AKI is associated with high mortality and considered an independent risk factor for death in patients with COVID.

Their new model is helping identify how calamity can occur when the two conditions collide, even if they are separated by years.

During pregnancy, maternal circulation must support fetal circulation so cardiac output increases, total body volume increases as well as plasma volume so the

kidneys have an increased load to filter, Gillis says. “There is an increase in plasma volume to ensure the high metabolic demands of both baby and mother are met,” Sullivan adds. In this scenario, the baby actually gets preferential protection.

Two recent clinical studies and their new model suggest that even years later, some young women have lingering damage that can prevent these usual responses and protections. It can happen despite the fact that the mother’s kidney function appeared normal before pregnancy, including normal creatinine levels, a waste product that healthy kidneys filter from your blood that is measured in the blood and/or urine as a standard assessment of kidney function. ♡



Editor's Note:

It is with great sadness that we share that Dr. Gillis passed away suddenly Wednesday, June 23.



(from left) Drs. Ellen E. Gillis and Jennifer C. Sullivan



FOOD FIGHT



A plant-based diet appears to afford significant protection to rats bred to become hypertensive on a high-salt diet. When the rats become pregnant, the whole grain diet also protects the mothers and their offspring from deadly preeclampsia.

Although we have all heard to avoid the salt shaker, an estimated 30-50% of us have a significant increase in blood pressure in response to high-salt intake, percentages that are even higher and more impactful in Blacks.

Two new studies provide more evidence that the gut microbiota, which contains trillions of microorganisms that help us digest food and plays a key role in regulating the response of our immune system, is also a player in the unhealthy response to salt, investigators at the Medical College of Georgia and Medical College of Wisconsin report in the journals *ACTA PHYSIOLOGICA* and *Pregnancy Hypertension: An International Journal of Women's Cardiovascular Health*.

The findings provide more evidence of the “potential power” of nutritional intervention, and consequently our long-term health, says Dr. David L. Mattson, chair of the MCG Department of Physiology, Georgia Research Alliance Eminent Scholar in Hypertension and senior author on the two studies.

They result from the unexpected observation that the protection works even in a well-established model of salt-sensitive hypertension: The Dahl salt sensitive rat, which is bred to develop hypertension and progressive kidney disease on a high-salt diet.

In 2001, the Medical College of Wisconsin in Milwaukee shared their colony of Dahl SS rats, who were fed a milk-based protein diet, with Charles Rivers Laboratories. Once the rats arrived there, they were switched to a grain-based diet. Both diets are relatively low in sodium, although the protein, or casein-based, diet actually has a little less salt.

It was soon noted that when high-salt content

was added to their diet, the relocated rodents developed significantly less high blood pressure and related kidney damage than the rat colonies that remained in Wisconsin. More than a decade of research documented these differences, Mattson and his colleagues at MCG and MCW write, and now has shown them that developing salt-sensitive hypertension isn't just about sodium consumption.

“The animal protein amplified the effects of the salt,” says Mattson, a longtime hypertension researcher, who along with Dr. Justine M. Abais-Battad, physiologist, and postdoc Dr. John Henry Dasinger, came to MCG from Wisconsin two summers ago.

“Since the gut microbiota has been implicated in chronic diseases like hypertension, we hypothesized that dietary alterations shift the microbiota to mediate the development of salt-sensitive hypertension and renal disease,” they write in the journal *ACTA PHYSIOLOGICA*.

The gut microbiome is designed to metabolize what we eat, break it down and put it in a form that gives us nutrition, first author Abais-Battad says, and reciprocally it reflects what we eat.

When they looked at the microbiomes in the rats: “Sure enough, they were different,” she says.

They sequenced the genetic material of both rat colonies and found they were “virtually identical,” but their response to a high-salt diet was anything but, Mattson says.

As they anticipated at this juncture, the Wisconsin rats developed renal damage and inflammation — both indicators of high blood pressure — but on the same high-salt diet, the Charles River rats

experienced significantly less of these unhealthy results. The distinct differences they saw in their microbiota, reflected the difference in disease incidence and severity.

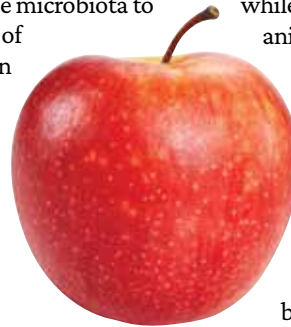
There also is evidence that even on a low-salt diet, Dahl salt sensitive rats are inclined to develop preeclampsia, a potentially lethal problem during pregnancy where the mother's blood pressure, which typically was normal before, soars and organs like the kidneys and liver show signs of damage.

To look at the impact of diet in this scenario, the Dahl SS rats were kept on their respective plant- or animal-based protein diet and both groups had three separate pregnancies and deliveries.

Rats on the whole wheat based-chow were protected from preeclampsia while about half of the rats on the animal-based casein diet developed this significant complication of pregnancy. They experienced a significant increase in the protein spilled into their urine, an indicator of kidney trouble, which worsened with each pregnancy; increased inflammation, a driver of high blood pressure; increased pressure inside the renal artery; and showed significant signs of kidney destruction when the organs were studied on follow up. They died of problems like stroke, kidney disease and other cardiovascular problems.

The work Abais-Battad, Dasinger and Mattson already have done shows that the protein-based diet produces more proinflammatory molecules, where the plant-based diet seems to suppress these factors.

They are further exploring the impact of diet of the renin-angiotensin system, which helps regulate blood pressure. They also want to better dissect the blood pressure-raising bacteria and the factors they produce. ♻️



SALT SENSITIVE

Obesity and a high-salt diet are both bad for our hearts but they are bigger, seemingly synergistic risks for females, scientists report.

“We see younger and younger women having cardiovascular disease and the question is: What is the cause?” says Dr. Eric Belin de Chantemele, physiologist in the MCG Vascular Biology Center and Department of Medicine. “We think the fact that females are more salt sensitive and more sensitive to obesity are among the reasons they have lost the natural protection youth and estrogen are thought to provide.”

His message to women based on the sex differences they are finding: “First reduce your consumption of salt, a message the American Heart Association has been pushing for years, which should also result in a reduction in your intake of highly processed, high-calorie food and drink.”

Belin de Chantemele, whose research team has been exploring why so many young women are now getting cardiovascular disease, presented their findings during the Henry Pickering Bowditch Award Lectureship at the American Physiological Society Annual Meeting at Experimental Biology 2021. The award, which honors the scientist who created the first physiology lab in



the country and was the American Physiological Society's first president, recognizes original and outstanding accomplishments in the field of physiology by a young investigator.

The sex hormone estrogen, which has some protective powers like keeping blood vessels more flexible, is considered a natural protection for premenopausal women yet, along with soaring rates of severe obesity in young women, heart disease is now the third leading cause of death in females between the ages 20-44 — fourth for males in that age group — then moves up to second place for the next 20 years in both sexes, and is the number one killer for both men and women looking at all ages, according to the National Vital Statistics Reports.

While Belin de Chantemele refers to bad nutrition as the “world's biggest killer” and obesity as a major risk factor for hypertension in both sexes, his lab has mounting evidence that obesity and high salt intake are even bigger risks for females, who have naturally higher levels of two additional hormones, leptin and aldosterone, setting the stage for the potentially deadly cardiovascular disparities. ♡

Dr. Eric Belin de Chantemele



NEW SURGERY CHAIR



Dr. Caprice C. Greenberg, a renowned health services researcher and surgical oncologist, is the new chair of the MCG Department of Surgery. She also is the Moretz/Mansberger Distinguished Chair in Surgery.

Greenberg, who previously served as the Morgridge Distinguished Chair in Health Services Research in the Department of Surgery at the University of Wisconsin School of Medicine and Public Health in Madison, joined the medical school last spring.

Her career focus has been using research to improve the quality and safety of surgical care. She has been at the forefront of the push to move surgical investigation, including video capture and analysis, into operating rooms to study the way care is delivered and to focus on system, team and individual provider performance.

Greenberg created the Wisconsin Surgical Outcomes Research Program (WiSOR), a multi-investigator research program she directed until 2019. WiSOR was developed in 2011 to improve the quality, safety, effectiveness and efficiency of surgical care through research and innovation.

It is an effort she intends to replicate at MCG and across Georgia. ♡

NEWS & VIEWS



PRIME TECHNOLOGY

The latest gene editing technology, prime editing, expands the “genetic toolbox” for more precisely creating disease models and correcting genetic problems, scientists say.

In only the second published study of prime editing’s use in a mouse model, MCG scientists report prime editing and traditional CRISPR both successfully shut down a gene involved in the differentiation of smooth muscle cells, which help give strength and movement to organs and blood vessels.

However, prime editing snips only a single strand of the double-stranded DNA. CRISPR makes double-strand cuts, which can be lethal to cells, and produces unintended edits at both the work site as well as randomly across the genome, says Dr. Joseph Miano, genome editor, molecular biologist and J. Harold Harrison, MD, Distinguished University Chair in Vascular Biology at the MCG Vascular Biology Center.

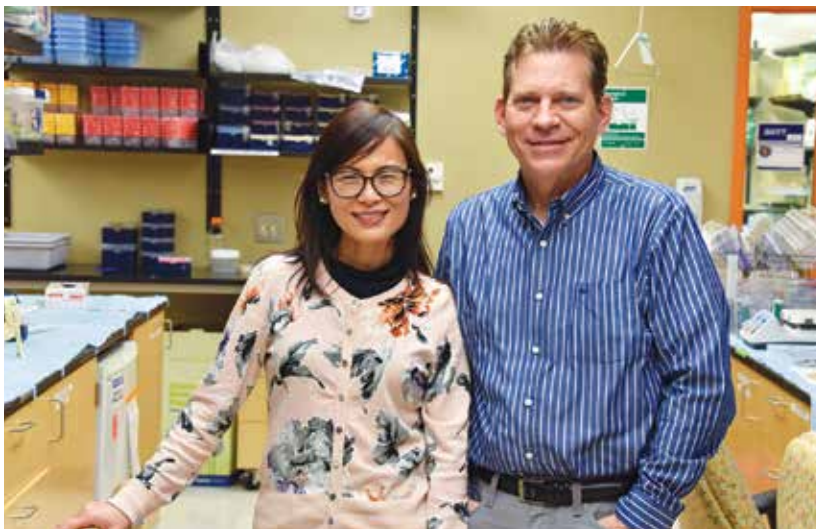
Prime editing is the latest gene-editing technology, and the MCG scientists report in the journal *Genome Biology* that they were able to use it to

remove expression of a gene in smooth muscle tissue, illustrating prime editing’s ability to create cell-specific knockout mice without extensive breeding efforts that may not result in an exact model, says Dr. Xiaochun Long, molecular biologist in the Vascular Biology Center. Miano and Long are corresponding authors of the study.

“It’s actually less complicated and more precise than traditional CRISPR,” Miano says of prime editing, which literally has fewer components than the game-changing gene-editing tool CRISPR.

Miano was among the first wave of scientists to use CRISPR to alter the mouse genome in 2013. Two scientists were awarded the 2020 Nobel Prize in Chemistry for the now decade-old CRISPR, which enabled rapid development of animal models, as well as the potential to cure genetic diseases like sickle cell, and potentially reduce the destruction caused by diseases like cancer, in which environmental and genetic factors are both at play. 🌱

Drs. Xiaochun Long and Joseph Miano



CBD VERSUS ALZHEIMER'S

A two-week course of high doses of CBD helps restore the function of two proteins key to reducing the accumulation of beta-amyloid plaque, a hallmark of Alzheimer’s disease, and improves cognition in an experimental model of early onset familial Alzheimer’s, investigators report.

The proteins TREM2 and IL-33 are important to the ability of the brain’s immune cells to literally consume dead cells and other debris like the beta-amyloid plaque that piles up in patients’ brains, and levels of both are decreased in Alzheimer’s.

The investigators report for the first time that CBD normalizes levels and function, improving cognition as it also reduces levels of the immune protein IL-6, which is associated with the high inflammation levels found in Alzheimer’s, says Dr. Babak Baban, immunologist and associate dean for research in the Dental College of Georgia and the study’s corresponding author.

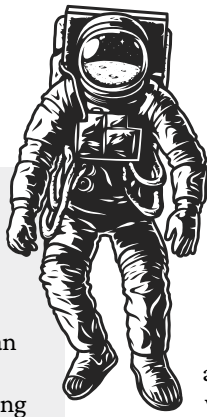
There is a dire need for novel therapies to improve outcomes for patients with this condition, which is considered one of the fastest-growing health threats in the United States, DCG and MCG investigators write in the *Journal of Alzheimer’s Disease*.

“Right now we have two classes of drugs to treat Alzheimer’s,” says Dr. John Morgan, neurologist and director of the Movement and Memory Disorder Programs in the MCG Department of Neurology. One class increases levels of the neurotransmitter acetylcholine, which also are decreased in Alzheimer’s, and another works through the NMDA receptors involved in communication between neurons and important to memory. “But we have nothing that gets to the pathophysiology of the disease,” says Morgan, a study coauthor.

The DCG and MCG investigators decided to look at CBD’s ability to address some of the key brain systems that go awry in Alzheimer’s.

They found CBD appears to normalize levels of IL-33, a protein whose highest expression in humans is normally in the brain, where it helps





THE GRAVITY OF THE SITUATION

sound the alarm that there is an invader like the beta-amyloid accumulation. There is emerging evidence of its role as a regulatory protein as well, whose function of either turning up or down the immune response depends on the environment, Baban says. In Alzheimer's, that includes turning down inflammation and trying to restore balance to the immune system, he says.

That up and down expression in health and disease could make IL-33 both a good biomarker and treatment target for disease, the investigators say.

CBD also improved expression of triggering receptor expressed on myeloid cells 2, or TREM2, which is found on the cell surface where it combines with another protein to transmit signals that activate cells, including immune cells. In the brain, its expression is on the microglial cells, a special population of immune cells found only in the brain where they are key to eliminating invaders like a virus and irrevocably damaged neurons. ♡

Extended time in space weakens astronauts' bones, so scientists are working to better understand how bone senses and responds to the usual forces placed upon it with the goal of keeping their bones strong.

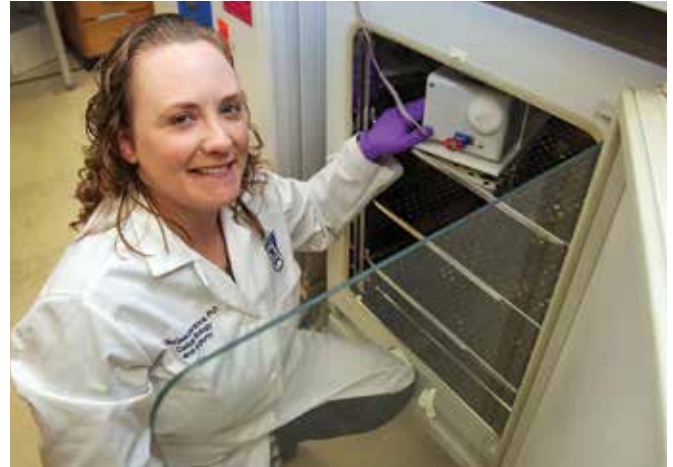
The skeleton's ability to adapt to mechanical loading — the forces put on bone by both gravity and muscle in response to movement — is critical to bone health, and circumstances like spaceflight or a spinal cord injury can interfere, says Dr. Meghan E. McGee-Lawrence, biomedical engineer in the MCG Department of Cellular Biology and Anatomy.

McGee-Lawrence received a \$750,000 grant from NASA to help her better understand how lack of gravity and other causes of disuse affect the usual dynamics of the skeleton, and ways to restore a healthy dynamic when usual options like increased physical activity and weight lifting are not viable.

Her focus is the natural sensors of mechanical loading on the bone called osteocytes. They are the most common cell

in the bone, and McGee-Lawrence's lab was the first to report that tears, called plasma membrane disruptions, occur in osteocytes with mechanical loading, and that, when all goes well, the result is healed tears and building bone.

They have shown that plasma membrane disruptions happen in healthy osteocytes in under a minute in response to load and immediately initiate changes like letting in more calcium, which in turn alters expression



Dr. Meghan E. McGee-Lawrence

of molecules that regulate the activity of bone-forming osteoblasts and bone-consuming osteoclasts.

Calcium found in abundance in the extracellular fluid around osteocytes rushes through the new opening where the mineral, best known for building strong bones and teeth, instead functions as a call to action for these cells.

When there are few tears and little mechanical loading, the message to osteoblasts is that denser bone is not needed and the osteoclasts will resorb some of the bone matrix. Conversely, when osteocytes sense a lot of loading and tearing, they will instruct osteoblasts to shore up bone density and osteoclasts to cut back on resorption.

Now, they are looking directly at what happens to plasma membrane disruption formation and repair in osteocytes in response to disuse. They also are looking for ways to normalize this apparently essential process in maintaining healthy bone in the face of disuse that occurs in space as well as on Earth. ♡



Drs. Babak Baban, John Morgan and Hesam Khodadadi



GREY MATTERS

Enhancing care of the brain

The MCG Department of Neuroscience and Regenerative Medicine, under the leadership of Dr. Xin-Yun Lu, department chair since July 2019, has been making strategic hires of scientists with expertise in and passion for life-changing neurodegenerative conditions like **spinal cord injury**, **Parkinson's**, **Alzheimer's**, **ALS** and **PTSD**. The Departments of Neurology and Medicine in collaboration with the Georgia Cancer Center have hired neuro-oncologist Dr. John Henson to enhance treatment of **brain tumors** and other nervous system tumors. Prevention expert Dr. Martha Tingen and Psychiatry Chair Dr. Vaughn McCall, an expert in the trifecta of suicide, depression and insomnia, are taking on **suicide**, an increasing cause of death in the United States.

Reconnecting the Brain and Body

BY TONI BAKER

Despite what we may have heard, there are a handful of places where adult humans can generate neurons.

The obviously highly busy olfactory bulb, which sits in our forebrain at about the same level as the top of our nose and where we process odors detected by the cells in our nasal cavity, is really good at it. Until we get old anyway, so is the dentate gyrus, part of the hippocampus, which is critical to learning and memory.

But not our spinal cord, which endlessly relays messages between our brain and our body.

Dr. Hedong Li, molecular neuroscientist, studied retinal and brain development early in his career, and remains fascinated by how neurons, or nerve cells, a fundamental working unit of the central nervous system that enable everything from sight to thought to movement, develop. “I am so curious about why it happens, how it happens,” he says.

Much of his focus today is on encouraging the injured spinal cord to make it happen.

“We need better treatment,” Li says flatly, and he has good evidence that cell reprogramming — in his case turning astrocytes, star-shaped cells that normally support neurons, into actual neurons — can

help reestablish lost brain and body connection.

Like a well-functioning highway or high-speed internet, the spinal cord has ascending and descending fibers to and from the brain. “If you have an injury in the spinal cord you basically block the highway,” he says. So he is building a workaround.

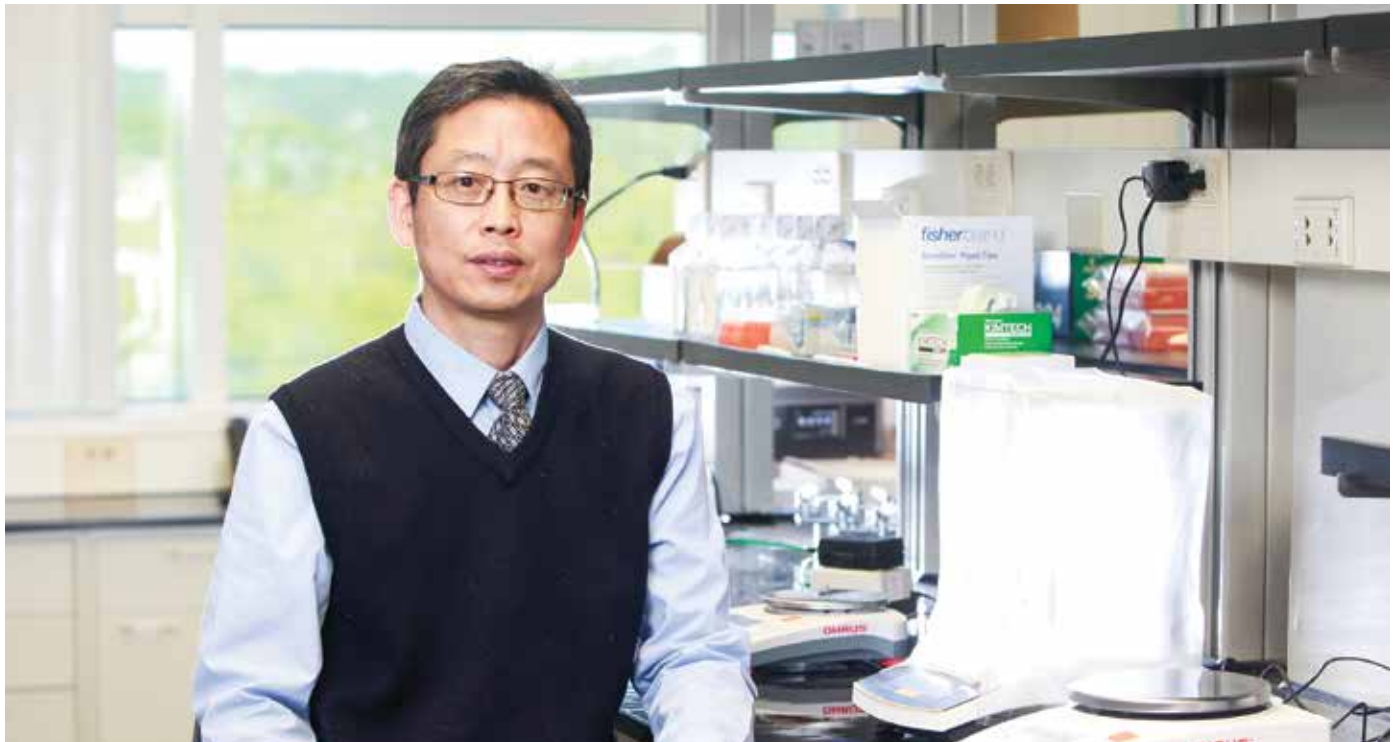
“We know cell reprogramming is feasible and we can do it. Now we need to know how do we make this optimal in order to translate this into a clinic so it can lead to functional recovery in spinal cord injury,” he says. “We have a lot to do.”

If it sounds astounding that scientists can help produce functioning new neurons that will make synapses so they can reach out to each other and reach around an injury site, Li says it isn’t really.

“It’s all in the genome,” says Li. “Every cell has the same genome, right? All the sequences are there you just have to turn on the right program,” he says referencing the four base pairs of our DNA, — adenine, cytosine, guanine and thymine — which line up in endlessly different combinations to make us.

In his case, Li is looking back to his fascination with how neurons first develop and making use of key components to make more of them.

Dr. Hedong Li



neuron noun

neu·ron | \ nū-răn , nyū-; nūr-ăn, nyūr-\

a grayish or reddish granular cell that is the fundamental functional unit of nervous tissue transmitting and receiving nerve impulses and having cytoplasmic processes which are highly differentiated frequently as multiple dendrites or usually as solitary axons which conduct impulses to and away from the cell body.

Merriam Webster

Uncommon support

Healthy neurons in our nervous system normally have a lot of good support, about a 10 to 1 ratio of glial cells like astrocytes. Within the central nervous system, which includes the brain and spinal cord, there are at least two other glial cell types — oligodendrocytes and microglia.

But astrocytes are the most common of these supportive cells, and their many essential duties include supporting the synapses, including recycling neurotransmitters, the chemical messengers that neurons pass between each other to incite action; cleaning up after neurons die; and helping ensure neurons get the right amount of blood and fuel so they live and function well. When astrocytes don't work well, it can contribute to neurodegenerative diseases like amyotrophic lateral sclerosis, or ALS, and Parkinson's.

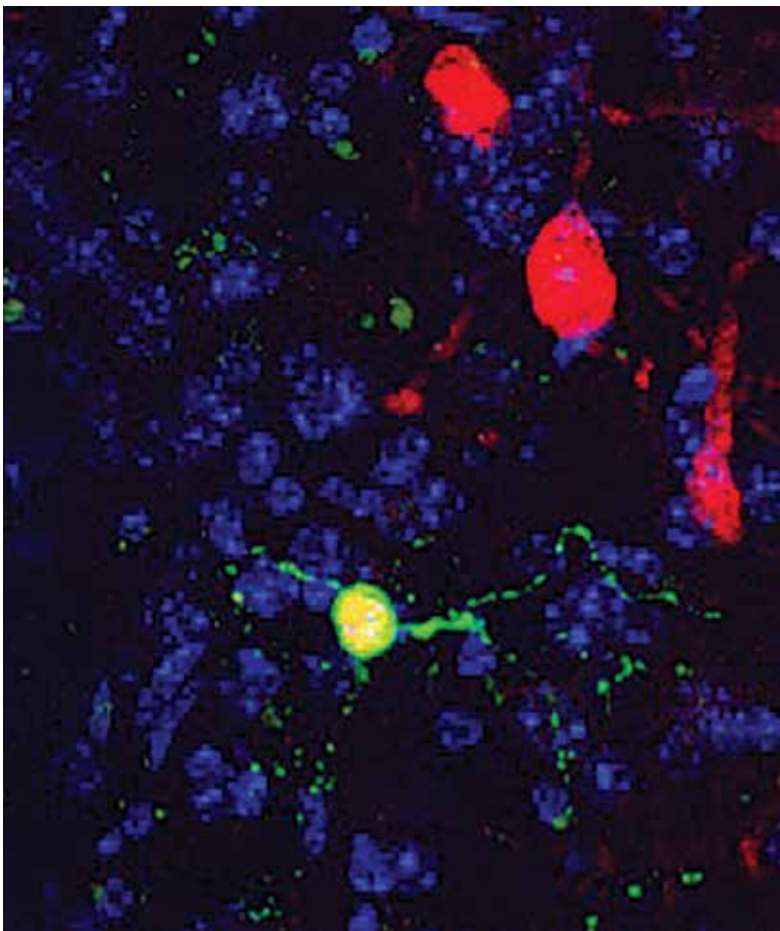
When injury happens, like the impact of a car wreck that cracks the boney spine which then severs the spinal cord it was designed to protect, astrocytes appear to further step up their game, growing their number and becoming more reactive. Meanwhile, the neurons they are there to support tend to be more vulnerable to injury and death and do not spontaneously regenerate.

Astrocytes' proliferation and ramped up stance in the face of injury is likely a function of their natural protective role, as immune cells move in, inflammation increases and all the glial cells react to multiple factors produced, Li says. Astrocytes even literally change shape, deposit a glue-like extracellular matrix around the injury site, likely with the idea of helping contain the injury like a tourniquet can stop bleeding, but ultimately the glue becomes the foundation and astrocytes themselves become part of a scar, which can further impede communication through the injury site.

The intention likely is to "prevent the injury from spreading. It's the natural response," Li says. The more significant the injury, the more significant the response, which is typically overdone, he says. Astrocytes also lose their physiological function when they become reactive, and it's not clear how easy it is for them to return to normal, Li says.

But he has developed a way to help tweak this scenario, taking advantage of the proximity of the apparent astrocyte surplus while helping normalize their function in the process.

Li is principal investigator on a two-year, \$423,000 R21 Exploratory/Development Research grant from the National Institutes of Health that is enabling he and his team to use a construct he has engineered to aid repair of an injured spinal cord by converting those astrocytes into neurons.



This image is showing a GFP-labeled, reprogrammed neuron (green) from a glial cell in the injured mouse spinal cord. The reprogrammed neuron elicits neuronal morphology and expresses the neuronal marker NeuN (red). DAPI (blue) is used to label cell nuclei.

It's all about balance

The construct takes advantage of the common adeno-associated viruses' ability to target a cell, in this case to deliver to astrocytes a gene that will increase the amount of NeuroD1 present. NeuroD1 is a neuron-specific transcription factor that helps turn on genes directly related to stem cells becoming neurons during our development. Part of the package is also an element that will make NeuroD1 more responsive to microRNA-124, which is skilled at turning genes down and plays a critical role in the formation, differentiation and maturation of neurons, once astrocytes become neurons.

Li received a \$1.8 million NIH grant in August 2020 that helped his team engineer the construct called ND1-124T.

The pairing seems logical because Li and his team have shown NeuroD1 can convert — one to one — the multitude of reactive astrocytes that show up after injury into new neurons in the brain and spinal cord. But as complex as that is, it's not that simple.

Li was corresponding author of a paper published in December 2020 in *Frontiers in Cell and Developmental Biology* that reported NeuroD1 converts astrocytes into neurons following a spinal cord injury, and that these converted neurons could mature and become part of the spinal cord wiring — even long after an injury. However, the process results primarily in excitatory neurons.

Li has evidence that part of the reason for the resulting paucity of inhibitory neurons is that while high levels of NeuroD1 are needed to make the desired reprogramming happen, levels remain high afterward. Normally following development, NeuroD1 levels are low and what those persistent high levels will do to the new neurons is another unknown.

That's where his construct comes in to help normalize the resulting mix. The construct has a binding site for microRNA-124, a natural gene suppressor which is not active in astrocytes. Once his construct boosts production of NeuroD1 in astrocytes, which enables the

reprogramming to neurons, the microRNA-124 binding site on his construct makes NeuroD1 more vulnerable to the naturally abundant microRNA-124 in the new neurons so NeuroD1 levels drop.

The bottom line is his construct helps ensure a more natural, healthy balance of excitatory neurons, which promote neuron firing so we can do things like walk and think, and inhibitory neurons, which suppress excessive firing, which can result in problems like seizures.

"We want to get things back to normal," Li says. "We think it's a smart strategy. The R21 grant is basically to test that idea," Li says. If he is right, they will have, not the only way, but a working way to retool an injured spinal cord.

The new grant is enabling analysis of the efficiency of his construct in an injured spinal cord, including the makeup of the resulting neurons and whether the new neurons improve function. The long term goal is a treatment for patients.

He and Dr. Sergei Kirov, neuroscientist and director of the 2-Photon Microscopy Core in the neuroscience department, have watched in three-dimension as NeuroD1 transforms astrocytes into neurons in the brain over a couple of weeks, and seen the reactive cells change shape and become a smaller cell body with long processes, or synapses, that can connect with other neurons, toward the end of week two. They are working on a way to also view this transformation within the thin, cylindrical — about the circumference of your little finger in humans — spinal cord.

While other scientists focus on regenerating broken axons — the long, slender nerve fibers that conduct electrical activity away from a neuron — through the spinal cord injury site, Li and his colleagues are working on more of a workaround, like the roundabouts popping up on roadways to help keep traffic moving.

Like those roundabouts with our vehicle speed, the workarounds may slow the speed of neuronal communication a bit, but should reestablish those lost connections. 🧡

Li says he has traditionally viewed himself as a doer not a dreamer, but he is working on some personal reprogramming to become one.

One of the first things he did after arriving at MCG in late 2020 was to visit the Spinal Cord Injury Unit at the Charlie Norwood VA Medical Center just across the street from MCG's teaching hospitals, the AU Health System.

The 40-year-old unit is one of the VA's largest for both veterans and active duty personnel. That visit kindled a dream for his next construct to be a spinal cord injury research center in Augusta.

Li received his undergraduate and master's degree in biochemistry from China's prestigious public research university Nankai University. Then came to the United States where he earned a master's in biochemistry from Wayne State University followed by a PhD in molecular neuroscience, also at the Detroit-based university. He did his postdoctoral work at Keck Center for Collaborative Neuroscience at Rutgers University, and became a research associate in 2001 and joined the faculty in 2005. He went back to China in 2009 and became a professor at West China Women and Children's Hospital of Sichuan University before returning to the U.S. to join the faculty at Penn State University in 2015. 🧡



C. elegans, a nematode, with a gene number similar to humans

Improving a Sticky Situation

BY TONI BAKER

Dr. Danielle Mor says the tiny, transparent *C. elegans* can help us better see how a little understood protein becomes sticky, piles up and destroys our neurons.

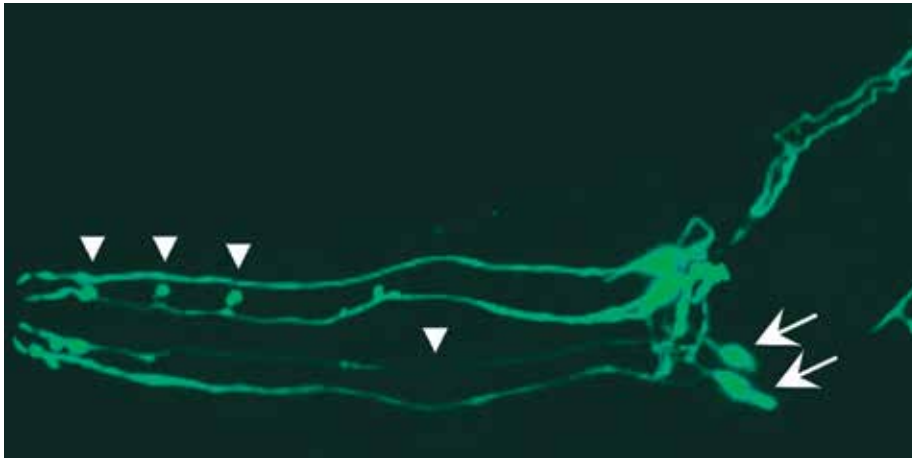
“I am trying to figure out how those neurons are dying,” Mor, neuroscientist, says of key brain neurons destroyed in Parkinson’s disease.

In her new lab in the Medical College of Georgia Department of Neuroscience and Regenerative Medicine, she has an RNA interference library with bacterial clones representing most of the genes of the *C. elegans*, a nematode, with a gene number similar to humans, many of which have functional counterparts to humans. These worms, which measure about 1 millimeter, or .039 inches, live about two to four weeks and stay transparent throughout life, also have a lot of nerve cells, or neurons — about one-third of their total cell count — and have long been considered excellent models for brain and aging research.

In the search to better understand what goes wrong in Parkinson’s, the worms enable Mor to test specifics, like a bit of a bacterium, and look at its impact, and use the real magic of high throughput screening to efficiently identify the most logical culprits in the devastation of Parkinson’s.

One clear problem in this neurodegenerative disease is with that little understood protein α -synuclein, which is primarily found in neurons and thought to have a role in synaptic vesicles, which are essentially biological carrying cases that deliver important messages so we can do things like raise our hand.





Degenerating neurons

The educator in Mor makes her head straight to the whiteboard in her office where she shares how neurons share information. Like outstretched arms, neurons have dendrites that take in information from the thin, long axon of another neuron. While their extensions don't technically touch, their essential exchange occurs through points called synapses and vesicles that, in this case, deliver the neurotransmitter dopamine. *C. elegans* have a similar setup.

The protein α -synuclein is all around these vesicles, but while exactly what they are doing is unclear, it likely has a role in the organization and recycling of these synaptic vesicles, Mor says. To add to the puzzle of just what this protein is up to, mice seem generally normal when it's knocked out.

But in the estimated 10 million people in the world, living with Parkinson's, the harm it can do is clear.

Protein power

Proteins are the workhorse of cells and they must be properly folded, or configured, to do their heavy lifting throughout the body. In this case, α -synuclein becomes misfolded in a small percentage of people because of genetics and in others likely from some sort of environmental triggers or other unknowns.

Whatever the cause, what is essentially the sticky innards of the amino acids that comprise the protein, become exposed, so the proteins start sticking together, starting with just a few then potentially building like a rolling snowball. But any size is potentially deadly to neurons. And the smaller ones likely are the most adept at traveling.

Much like the infamous amyloid plaque that grows in Alzheimer's and other neurodegenerative diseases, misfolded α -synuclein forms plaques in this case inside our neurons called Lewy bodies, which also are known to impact cognition and other basic brain functions.

Why microglial cells, immune cells resident in the brain, don't just eliminate the unnatural, growing protein wads early on is probably because, like most body functions, the quality control function of cleanup diminishes as our age increases, Mor says.

So Mor, who just completed her postdoctoral fellowship at Princeton University before joining the MCG faculty in October, is enthusiastically committed to helping find a way to prevent or at least stop the snowball and spread in her professional lifetime.

There is much work to do. "The short answer is, we still don't know," but simply put, neuron death seems to result from the protein aggregates gumming up its inner workings, like sticky art resin might threaten the life of a beautiful piece of art.

How sticky starts

How/why the initial stickiness starts, remains another million dollar question. Seemingly ironically, Mor and others — Mor showed it for the first time in a mouse brain — have seen that the simple interaction of dopamine, which is packaged in the synaptic vesicles, and α -synuclein, which hovers around those vesicles, can prompt the unhealthy aggregation that ultimately kills off the neurons producing dopamine.

Particularly problematic is that the stickiness appears contagious, spreading to α -synuclein in other neurons, although how remains another million dollar question, says Mor. It may be that a neuron literally spits out an aggregate before it gets too big. Then that sticky wad may travel quite a distance.

In fact, some of the latest evidence indicates that the trouble may actually start in the gut.

Mor theorizes, and there is some evidence in patients to support her ideas, that we are either consuming some already sticky form of α -synuclein and/or something like a bacterium in our gut is making the protein in the neurons in our gut sticky. However it starts, these agile sticky wads may be traveling up the natural communication paths from the digestive tract to the brain, which is known to have two-way conversations with the gut.

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Which takes her back to the *C. elegans*.

Mor is developing a model that recreates that sequence of events in the worm, then she can use the proven, incredible flexibility of the worm model to do high throughput screening of thousands of potential modifiers and genetic interactors looking for early instigators of the destructive, traveling protein aggregations.

She hopes by working both to see what might stop the stickiness and what makes it worse, she can better understand the problem and potential solutions for people.

The fact that she can do such large scale analysis means this high-energy individual can multiply her impact and better focus her future effort.

“With the worm, what you can do is test for thousands of chemicals or drugs or genetic manipulations to see what is important or what has an effect on what you want to study.”

Something must initiate the problematic misfolding, she says. Some theorize a “leaky gut” is enabling bacteria and inflammatory molecules to seep into surrounding tissue, triggering inflammation and ultimately that unhealthy protein aggregation. She hopes it is something we are eating, drinking or maybe one or more bacteria in our microbiome so it’s something we can change and so reduce the likelihood of developing Parkinson’s.

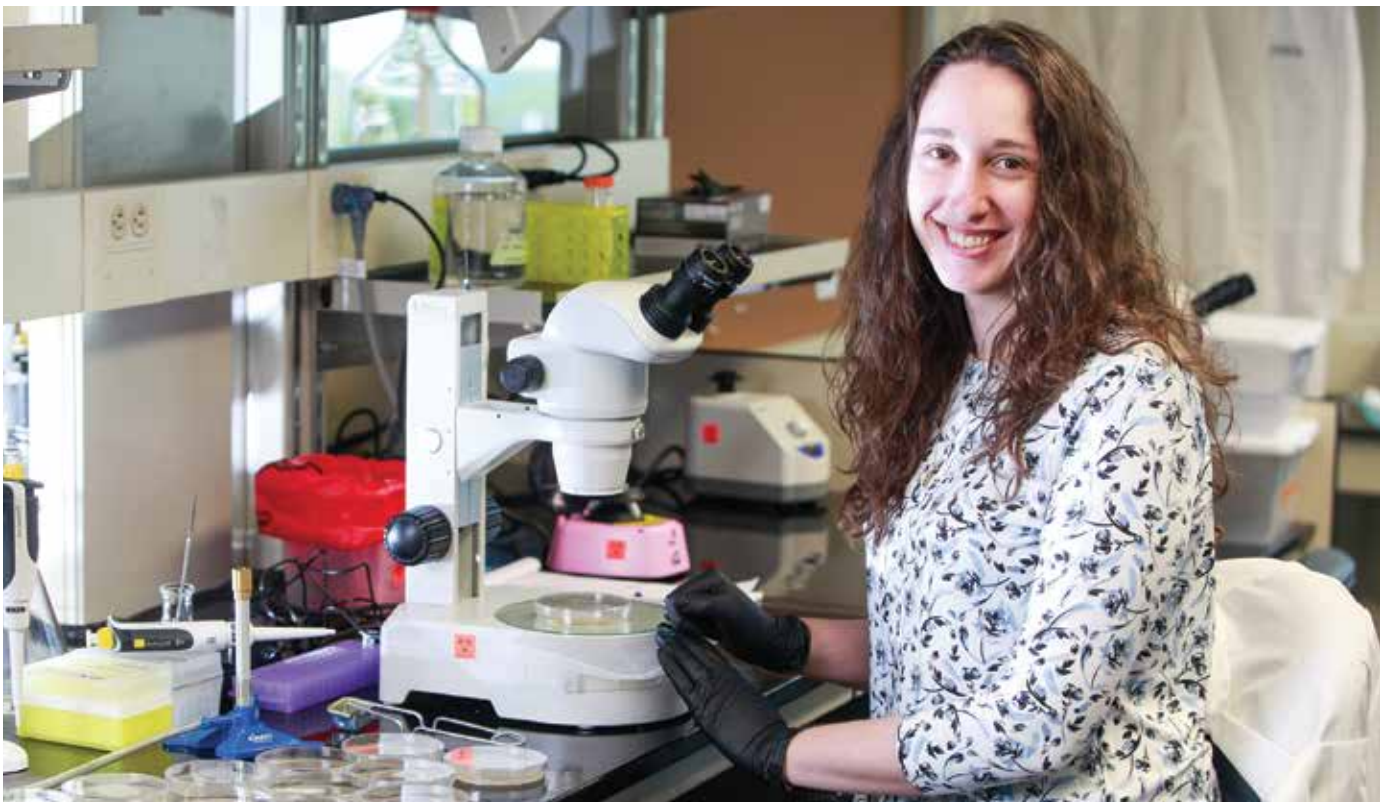
“These are horrible problems that people have to endure. Hopefully we will get to the point where they don’t have to endure it.” 🍀

Mor is a New Yorker by birth, who captures the energy and enthusiasm of America’s largest city. She was smitten with the brain early, intrigued by concepts like how the clearly physical brain enables such intangibles as emotions.

“To me the brain is unmatched,” is an early truth she holds to this day. When she asked parents Eva and Gabi Mor about how the brain worked, they rightly told her nobody 100% knows and she could go do the research to find her answers. Mom, Dr. Eva Mor, is an epidemiologist and published author of works like “Making the Golden Years Golden,” who ultimately focused her career on helping older individuals live better. Her life’s work likely fueled Mor’s in the difficult diseases of aging like Parkinson’s. “She kind of instilled in me a fascination and desire to protect.” Her mom’s dad, Leib Rozen, also lived for 14 years with Parkinson’s. He died before his granddaughter made her career decisions, but no doubt he was with her when she did.

Mor earned her undergraduate degree in neuroscience and behavior from Wesleyan University in Middletown, Connecticut, a PhD in neuroscience at the University of Pennsylvania School of Medicine, and completed her postdoctoral fellowship at Princeton University in New Jersey in September 2020 before joining the MCG faculty. 🍀

Dr. Danielle Mor



A CRITICAL JUNCTION

BY TONI BAKER

Dr. Ferenc "Frank" Deak describes a chemical synapse as "really where all the action happens in the brain," a point where an action potential, basically an electrical message from the neuron, triggers the release of a chemical message, or neurotransmitter.

The first synaptic connections we form enable basics like eating and going to the restroom; most of our cognitive functions peak at about age 25, but we actually have the highest number of synapses in our terrible twos, Deak says. "Those two-year-olds are sponges they can learn so quickly," he adds.

Conversely, losing synapses is a leading indicator of Alzheimer's disease, something he is working to change.

Aging is the biggest known risk factor for Alzheimer's, according to the Alzheimer's Association, and the buildup of beta amyloid plaque, a hallmark of the disease.

In what feels like a twisted tale, neuroscientist Deak studies how the synaptic connections influence the buildup that ultimately destroys them.

Beta amyloid is a piece of amyloid precursor protein, one of the highest expressed proteins in a healthy brain. The protein is essential to the developing brain and apparently to the developed brain as well, including providing all sorts of signaling functions, although exactly what remains unclear, Deak says. However it does appear that to do its jobs, amyloid precursor protein needs to be cut into smaller sections, and under the wrong circumstances, one of the pieces cut out is sticky beta amyloid.

The sticky pile can build over maybe 20 years or more, until it becomes a substantial plaque outside neurons that interferes with their ability to communicate. The pile also triggers destructive inflammation. As damage progresses outside, inside the neurons another twisted collection of the protein tau, called neurofibrillary tangles, coincides with the onset of dementia.

“Connections get lost between neurons,” says Deak, and by then it’s too late. “We want to do something before that.”

The way communication is supposed to happen is that with the help of a family of proteins called SNARE, a biological carrying case called a vesicle fuses to the membrane of the presynaptic neuron —

the neuron sending the message. That creates an opening in the cell membrane at its outer edge and near the receiving end of the postsynaptic neuron, so the neurotransmitter and other contents of the vesicle can be released, in a process called exocytosis. Then in rapid succession, the empty vesicle essentially collapses into the membrane of the presynaptic neuron so it can be taken back inside the neuron, retooled and reused.

But like the rest of our body, vesicles change with age and with Alzheimer’s.

Untwisting the tale

In a bit of a perfect storm, synaptic vesicles in the older brain tend to pick up more amyloid precursor protein from the membrane of the neuron during their refresh. When the flattened biological carrying case gets brought back inside the neuron, the increased amount of amyloid precursor protein it brings with it can encounter enzymes called beta and gamma secretases, which process membrane proteins. But in this case, they are problematic, excising the sticky beta amyloid. Inside the neuron, the sticky neurotoxic amyloid starts making bad changes, including impacting the vesicles. Later other vesicle types will start hauling it out, dumping the neurotoxin at the

synapse. One unfortunate failure in Alzheimer’s treatment was drugs that tried to improve the scenario by inhibiting the secretases that would make two cuts instead of one, so beta amyloid would not be freed, but there were odd consequences like bleeding from the gut and turning hair permanently orange.

Deak has mounting evidence that a protein on the surface of the vesicle’s membrane, might be a better point of action.

He collaborated with the Mayo Clinic Brain Bank to look in diseased and healthy brains for genetic variants, including synaptic protein genes, which might be players in Alzheimer’s.

He found that with age the amount of synaptobrevin-1, or VAMP1, increases in the membrane, or surface, of the vesicle. As the name implies, synaptobrevin-1 is a usual constituent of synaptic vesicles, aiding their fundamental function of neurotransmitter release.

Single nucleotide polymorphisms, or SNPs, are comparatively simple genetic variations and the most common variations found in humans; and genetic screens of humans have found SNPs for synaptobrevin-1 are “significantly” associated with late onset Alzheimer’s disease. Also, SNPs associated with

Dr. Frank Deak



increased synaptobrevin-1 expression increase the risk of Alzheimer's, and those that reduce its expression appear to reduce the risk. And, in synaptobrevin-1 knockout mice, beta amyloid levels are significantly reduced.

Deak and his colleagues hypothesize that variations in the synaptobrevin-1 level alter the relationship between synaptic transmission and the processing of amyloid precursor protein and consequently have a profound effect on beta amyloid production. A bottom line is that high synaptobrevin-1 levels mean amyloid precursor protein is cleaved in a way that produces more of that neurotoxic amyloid.

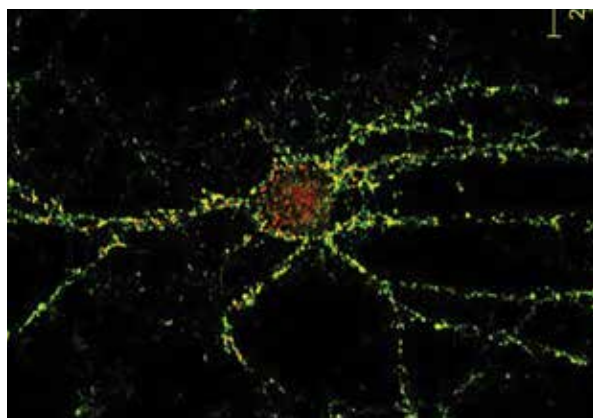
With age, all of us collect some amyloid plaque as well as neurofibrillary tangles, but in Alzheimer's there is so much that it interferes with neuron function.

Deak thinks the vesicle's role in the destructive tale is an early one

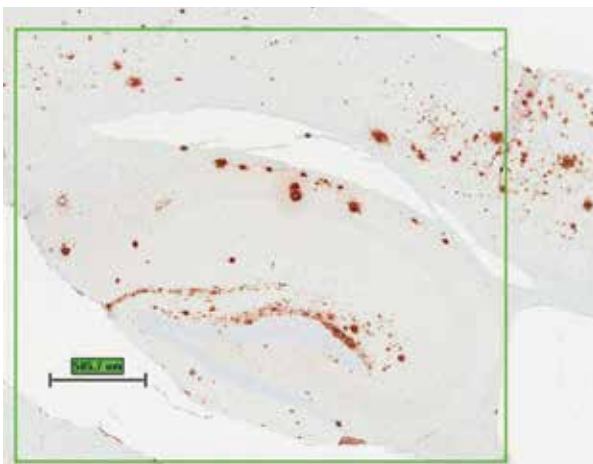
that a \$1.9 million grant from the National Institute on Aging is helping him further explore.

Right now he and his research team are looking at the impact of synaptobrevin-1 levels on the activity of synapses and production of beta amyloid in their knockout mice, and what reducing synaptobrevin-1 levels does to reduce cognitive impairment in a mouse model of Alzheimer's. They hypothesize that lower synaptobrevin-1 will reduce the unhealthy association between the level of synaptic activity, how amyloid precursor protein gets processed and how much neurotoxic amyloid ultimately gets dumped on synapses.

If they are correct, synaptobrevin-1 may emerge as an important regulator of amyloid production in the synapse and a novel target for diagnosing and treating Alzheimer's. 🌱



A healthy cultured mouse neuron.



The brain of an Alzheimer's mouse model; brown spots are destructive plaques.

Deak was born in Hungary, a country about the size of Indiana in Central Europe. He earned a doctor of medicine degree, summa cum laude, from Semmelweis University of Medicine in Budapest, in 1994, and a PhD in the university's Department of Physiology, also summa cum laude, in 2000. To support himself during graduate school, Deak was also a faculty member and lecturer in the Physiology Department, and on the weekends he pulled long shifts on ambulances, delivering babies, working accident scenes and making house calls. He came to the United States right after grad school, to complete postdoctoral studies with Dr. Thomas C. Südhof in the Center for Basic Neuroscience at the University of Texas Southwestern in Dallas. Five years later, Südhof would win a Nobel Prize for his work to understand the machinery regulating vesicle traffic, including how they stay in place, ready to release signals at the right moment with the help of calcium sensing proteins. Deak had originally loved the idea of being a neurologist — which made him a bit of the odd man out in a family focused on engineering — but decided that by being a neuroscientist he could discover more about the fundamentals that go wrong in the brain, like problems with those vesicles.

So next, Deak joined the faculty of the Department of Neuroscience at the Mayo Clinic in Jacksonville to focus on Alzheimer's research. In 2012, he joined the faculty of the Reynolds Oklahoma Center of Aging and Oklahoma Center for Neuroscience at the University of Oklahoma Health Sciences Center in Oklahoma City and came to MCG late last year. Over the years, he has served as a member of numerous NIH study sections, including the Chronic Dysfunction and Integrative Neurodegeneration Study Section. His work has been published in top journals like *Science*, *Neuron* and *Nature Cell Biology*. He is associate editor of *Geroscience*, the journal of the American Aging Association. The word geroscience is defined by the National Institute on Aging as the intersection of basic aging biology, chronic disease and health. As his long-term goal, Deak aims to use geroscience approaches to prevent multiple chronic brain disorders of the elderly. 🌱

Suppressed
by

STRESS

BY TONI BAKER

Our body is always about balance, and HCN1 is like a natural moderator in the hippocampus, our center of learning and memory.

HCN1 helps keep neuron activity and connectivity from getting excessive and communication from becoming garbled and disruptive, like what happens in a seizure.

Its levels are naturally high in the hippocampus, and neuroscientist Dr. Chung Sub Kim has evidence that even a single episode of significant stress can further increase HCN1 expression in the CA1 region of the hippocampus, which is critical to making, consolidating and retrieving memories, and push the excitability of neurons there down.

A few weeks of chronic stress pushes HCN1 expression way up and neuron excitability further down and appears to contribute to mental health pathology like depression and anxiety and PTSD.

A single, unforgettable life-changing event, like being the target of a sniper at a school shooting or a series of traumatic events like a tour of heavy combat duty, sexual assault or child sexual abuse, can result in long-term behavior changes, depression, nightmares, flashbacks, loss of interest in the things that normally provide pleasure, increased risk of substance abuse, suicide risk and more. The National Center for PTSD says that 7-8% of Americans will have PTSD at some point in their lives.

Back to balance, Kim wants to better understand how traumatic events induce PTSD and whether lowering HCN1 levels, or otherwise boosting the ability of our

neurons to get excited and take action like making a memory, can restore resilience in people whose lives have been potentially permanently rocked.

“We don’t know the cellular mechanisms underlying developing PTSD, which is why there is no specific treatment,” Kim says. Existing therapies, like using serotonin reuptake inhibitors, the most commonly prescribed antidepressant, for example, help maybe 20-30% of individuals with PTSD, he says, noting that depression and PTSD most typically go hand in hand.

HCN1 is the most common form of the hyperpolarization-activated cyclic nucleotide-gated nonselective cation channels, or HCN channels. H-current is generated by these HCN channels and is referred to as a pacemaker current in neurons and heart cells alike, because it helps sustain their intrinsic, essential

rhythm. Mutations in HCN1 are known to cause epilepsy, and if you completely block the channel you likely would lose interest in your usual activity and might see an increase in epileptic activity, he says.

Kim and his colleagues have shown that knocking out HCN1 in the CA1 region of the hippocampus reduces anxiety and depression behaviors in rats, changes associated with an increase in both neuron excitability and the synaptic activity that connects neurons and enables their communication and action.

They also reported in June 2020 in the journal *Cell Press* that one way just a single dose of ketamine, an anesthetic and pain reliever that gets misused as a “club drug,” may also produce a robust antidepressant effect is by reducing or preventing the current HCN1 generates. With tremendous side effects, including hallucinations,



Dr. Chung Sub Kim

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looming with ketamine and no Food and Drug Administration approved drug to directly target HCN1, Kim is looking for a safe way to do that in a very specific region of the hippocampus.

More is not always better

He notes that by changing just a single ion channel in one area of the complex brain, it will yield a change in behavior, and he has evidence that a virus may help him do that.

Kim already has shown in a depression model, a properly armed virus can help restore more normal neuron excitability and other positive responses, like more activity in that specific brain region and more normal behavior.

Now he is pursuing novel studies to do the same in PTSD. His tools include using a virus that is activated by blue light using a brain research tool called optogenetics, which in this case includes taking advantage of the infecting skill of a common cold virus to seek and find the CA1 region of the hippocampus.

The virus expresses channelrhodopsin, a protein that enables light to control electrical excitability and other processes — including enabling algae to move in response to light — to help turn

neuron excitability levels up toward normal.

He also is looking at a viral modality to directly knock HCN1 levels down, also to a more normal, pre-PTSD level, which also will ultimately turn up excitability. This approach targets expression of the HCN1 gene by disrupting its natural DNA pairing — how the DNA's building blocks are supposed to stack up — so it cannot produce the HCN1 protein.

His models include both chronic social defeat, like a larger mouse usurping the authority of another, as well as a witness stress model, where a mouse who knows the previous hierarchy witnesses — rather than directly experiencing — the resulting aggression as the new boss takes over.

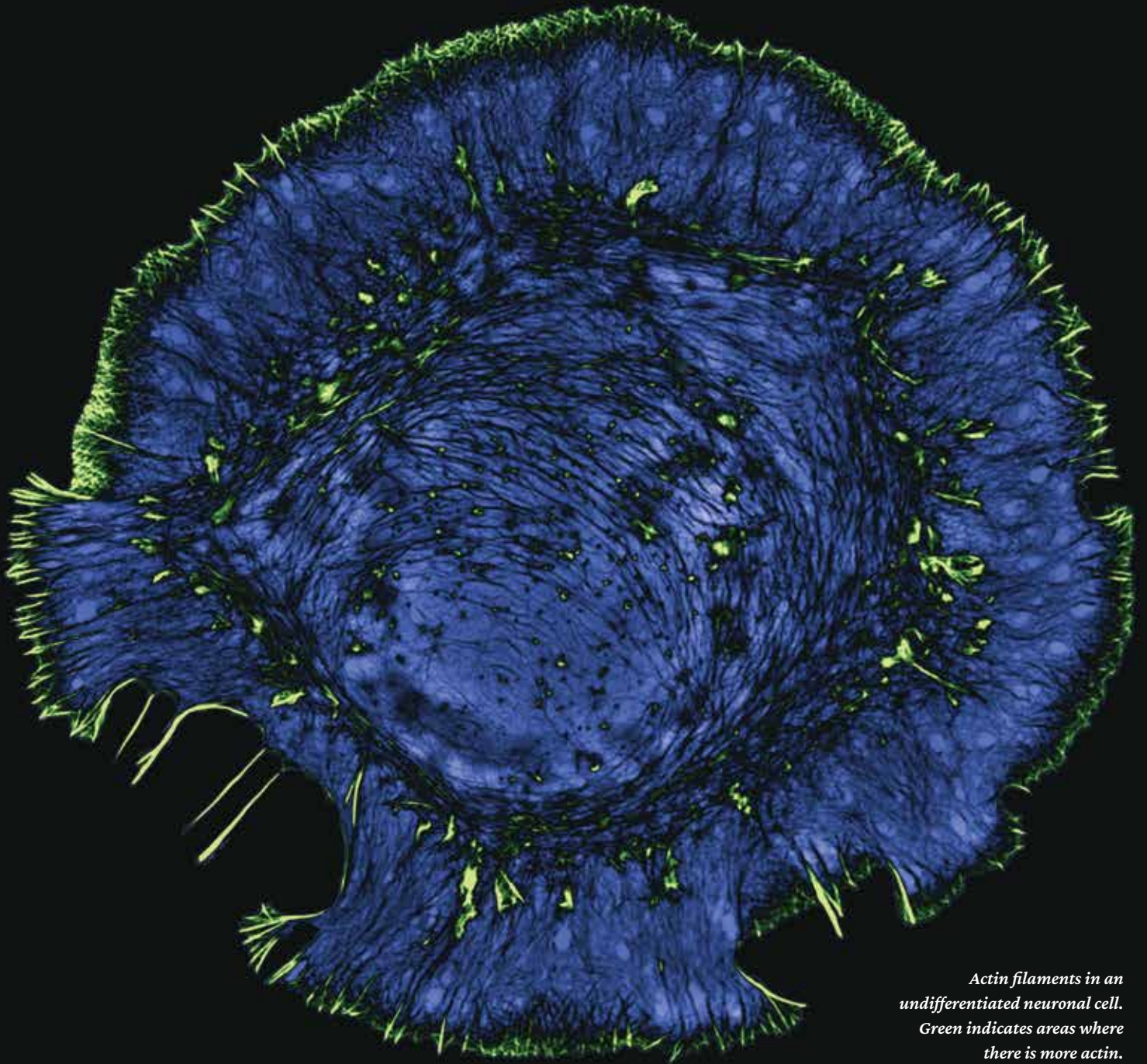
He also is looking further at what HCN channels are doing in the models of PTSD, what cellular mechanisms underlie the changes that occur and what happens to PTSD symptoms in the model when neuronal excitability and/or HCN channel expression are changed.

These studies will provide more insight into both susceptibility and resiliency to PTSD, Kim says, and whether lowering HCN1 levels and/or increasing neuron excitability can convert susceptibility into resilience. 🍀

Kim is a native of South Korea, a country of about 52 million people that is the geographic size of Dr. Frank Deak's homeland Hungary but with more than five times its population. He earned a master's of medical science degree from Yonsei University in Seoul then came to the United States for his PhD. He wanted to study at the University of Texas, Austin with Dr. Daniel Johnston, a world class expert in electrophysiology, who studies the cellular and molecular mechanisms of synaptic integration and long-term plasticity of neurons in areas of the brain, like the hippocampus. Kim worked his way into the Johnston lab and a PhD by starting as a lab technician who brought a different set of technical skills like immunostaining and biochemical assays. Johnston is now professor and director of the Institute for Neuroscience in the university's College of Natural Sciences. Despite Kim's significant determination, it wasn't easy being in a new country where the work was highly complex and the language barrier significant, but Johnston often used the old adage with him: No pain, no gain. Those words still echo in Kim's head as a motivator although he hasn't used them yet in his own lab at MCG, where he is building a team that now includes postdoc Dr. Jiwon Kim and technician Talisha Davis. Kim also did his postdoctoral studies with Johnston in Texas, became a research associate in 2015 and moved to MCG in May 2020. 🍀

Action of Actin

BY TONI BAKER



Actin filaments in an undifferentiated neuronal cell. Green indicates areas where there is more actin.

Dr. Eric Vitriol pulls up microscopy video of the actin cytoskeleton of a motor neuron, and its surface resembles the dancing edges of the sun with its solar flares.

The dynamic cytoskeleton enables a developing neuron to sense its environment, and steers it to the exceedingly precise location where it should connect with a muscle cell, a connection which can be more than three feet away. It generates forces and gives motor neurons structural support. It allows the cells to communicate with each other and internally, to divide and to move.

“The cell is constantly sensing its surrounding, so it’s moving all the time even though it’s not changing position,” says research partner Dr. Tracy-Ann Read.

The protein actin, found in and made by essentially all cells, in its single molecule, or monomer, state is the essential building block of the linear filaments that are the scaffolding for the dynamic cytoskeleton.

Actin monomers are constantly stacking up to form filaments that organize into these elaborate, dynamic architectures in cells, then disassembling as well in orderly fashion back to single actin molecules.

Motor neurons are making filaments all the time, whether you are sedentary or a marathon runner, because like us, filaments wear out and change with age. The actin filaments Vitriol and Read study assemble in seconds and last for one to five minutes before disassembly.

The wonders of modern microscopy, with the help of fluorescent labels, enables Vitriol and Read, both cell biologists and neuroscientists, to watch it all.

“It’s like a skeleton made of Legos that you keep putting together and taking apart,” says Vitriol. “You need to see it in order to really understand what is happening.”

They have evidence that what is happening in amyotrophic lateral sclerosis, or ALS, is disruption of this essential dynamic in the motor neurons that innervate our muscles so we can move.

They are going back to the actin building blocks to better understand how it’s done right, and what can go wrong.

“This might be a common mechanism that would help us better understand ALS,” Vitriol says.

The some half-dozen known “causes” of ALS don’t have apparent common ground, Read says. But the nearly 150 genes associated with ALS all interact with the actin cytoskeleton in some way. “If you could find common mechanisms between seemingly unrelated genes then you might find something that is causing ALS,” he says.

Disabling disconnections

In ALS, motor neurons die and you lose the ability to control your muscle cells, which also eventually die, Read says. “Lou Gehrig had some of the best motor neuron connections in modern history and what happens at some point in your life, you basically enter a rapid shutdown and system failure,” says Vitriol. Problems can start with weakness, spasticity and a hard time gripping things, and progress to where you cannot walk, breathe or swallow. But the neurons we think with most typically continue to work fine. Also, somewhat ironically, along the way, some patients develop inappropriate reflexes as nerves that are still alive start making some bad connections.

There are more than a dozen different types of motor neurons, some of which die early, others later. Read and Vitriol are working to understand what drives the degeneration of motor neurons in ALS, with the long term goal of designing targeted therapeutics to intervene.

Their focus includes, similar to those Legos, how actin molecules connect to form filaments, then later disconnect, and

how that essential, ongoing process can go bad, and what happens to motor neurons when it does, which is where ALS comes in.

They began connecting these potentially important dots when they discovered that a pathway in neurons important to their development and regeneration involves novel regulation of the actin cytoskeleton, and one of the genes important for that is the actin-binding protein profilin.

They reported in the journal *Current Biology* last year that profilin, which is also made by motor neurons, is a master regulator of these Lego pieces, both causing them to bind to form filaments when/where needed and preventing formation when/where they aren’t. In other words, profilin controls what types of filaments get made and the structure the cell takes. They have even shown that the amount of profilin controls the types of filaments assembled.

It’s a process that is happening quickly and constantly in busy motor neurons. “If you control the supply, you control the product,” Vitriol says, and profilin apparently is in control.

About that same time another group looking for more genes associated with ALS reported that mutations in profilin caused ALS in humans and that the mutations kept profilin from its natural binding to actin. The work provided more clues that the actin cytoskeleton held clues to motor neuron degeneration in the disease.

Now they want to know if structure or the function of actin or the polymerization, or stacking, of actin molecules is part of that and if it’s a factor in other, more common types of ALS, not just the familial type caused by profilin mutations.

“We know that profilin is messed up in some forms of ALS, that the cytoskeleton is messed up in several kinds of ALS, so realistically what the profilin mutants show you is that the cytoskeleton is probably important for disease progression,” Vitriol says.

“We are trying to figure out if actin regulation, if the way actin is regulated by

different proteins it interacts with or its structure, if any of that is dysregulated in ALS,” Read says. They already know that when profilin goes bad, so does actin.

Rare and precious access

A \$1.86 million Maximizing Investigators’ Research Award to Vitriol from the National Institutes of Health last summer, is enabling the sort of back to basics approach that will add insight into neurodegenerative diseases like ALS as well as heart disease, cancer and inflammatory disorders.

They have looked at some of the more common ALS associated genes and found a lot of them affect the actin cytoskeleton. Gene mutations that cause the hereditary form of ALS first enabled ALS studies in the lab, and scientists watched what happened to immortalized, commercially available cell lines when the mutants were expressed. But these don’t have a lot of

disease relevance, Vitriol says.

Their lab is one of the few that has been able to also culture spider-like adult motor neurons from mice. When examined at the end stages of ALS, they have seen the actin cytoskeleton is dramatically different, more evidence that actin is messed up in ALS, but still not enough, they say.

“ALS can run in families but 90% are spontaneous and you can’t recreate that in a mouse colony,” Vitriol says. Working with human samples can enable that exploration and is much of what brought the two to the Medical College of Georgia early in 2021 from the University of Florida.

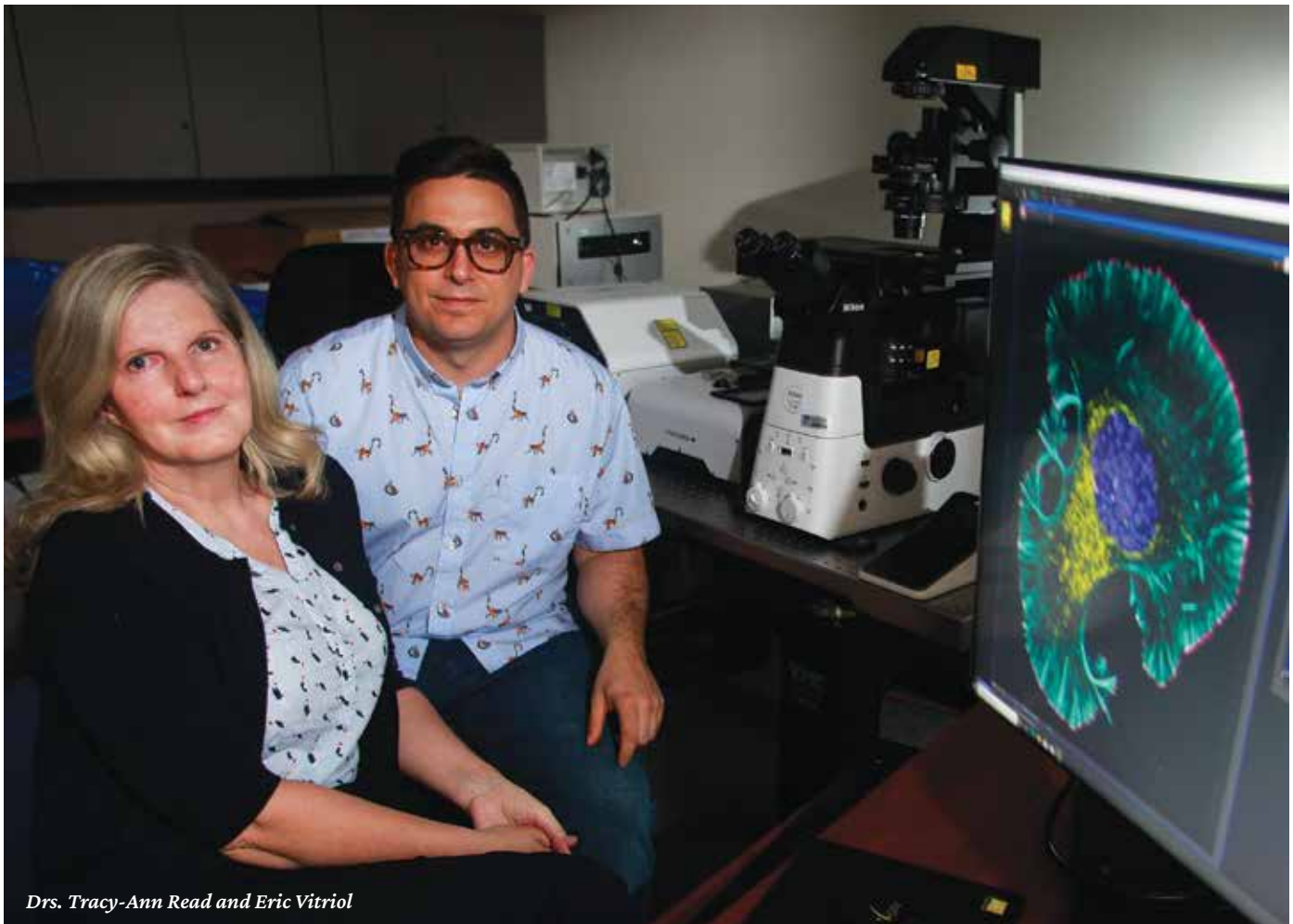
“If we really want to treat ALS properly, we need to know what the disease really is,” says Dr. Michael Rivner, medical director of the ALS Center at MCG’s affiliated AU Health System, an ALS Association certified treatment center of

excellence in Augusta. Rivner has been taking care of patients with ALS about 30 years and notes that his slide on the causes of ALS in his also now 30-year-old, although regularly updated, ALS presentation really has not changed much.

The treatment options have not either, with two approved drugs, riluzole and edaravone providing some slowing down of the disease, says Dr. Ben Barnes, neuromuscular neurologist who joined Rivner at MCG late last summer.

“The bottom line comes back to, we don’t know the cause(s), which is why besides having a clinical research component, we need to have a strong basic science component,” Rivner says. So he and Barnes worked hard with Department of Neuroscience and Regenerative Medicine chair Dr. Xin-Yun Lu and others to recruit Vitriol and Read.

At MCG, the new recruits have what they consider “rare and precious” access



Drs. Tracy-Ann Read and Eric Vitriol

to patient tissue and to the physicians who care for patients.

To develop studies of the more common, spontaneous ALS, the scientists now will be able to use the skin or blood of patients to make human motor neurons, as well as muscle samples to look at their muscle cells. They will even use these valuable human materials to rebuild the point of interaction between motor neuron and muscle cell, a fine point called the neuromuscular junction, which enables us to move and which the cytoskeleton enables.

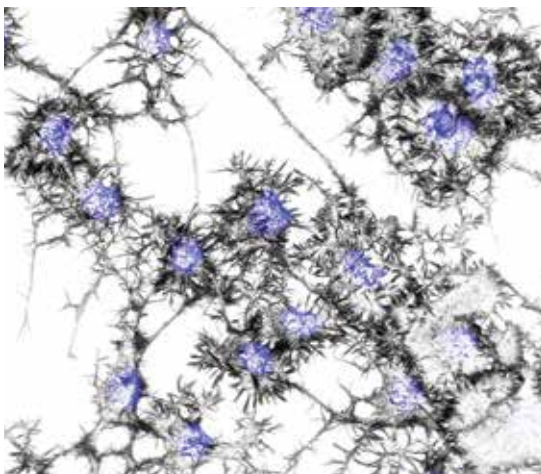
Vitriol says that connection will happen if you just co-culture this natural cell pairing. While in ALS, dying motor neurons retract their connections with their partner, Read says.

While there is no definitive test for ALS, part of the evaluation includes a nerve biopsy to ensure it is a nerve problem, Barnes says. He and Rivner are confident most of their patients will also readily agree to a small, safe punch biopsy to provide the other tissue needed to move science forward. Their families likely will happily supply the normal control samples needed.

The always-pragmatic Rivner says he knows he and Barnes are making patients' lives better through the care they offer at the bimonthly ALS clinic. To enhance offerings they are recruiting a third subspecialist and seeking center status for the ALS clinic at Atrium Health Navicent in Macon, Georgia, which Rivner also directs. He and Barnes, along with Brandy Quarles, research operations coordinator, also conduct clinical trials, currently exploring the potential of three new immune modulators that should reduce damaging inflammation around motor neurons.

But Rivner readily admits he wants the tools to do more. "It's hard to tell patients the diagnosis; it's hard to see the disease spread." 🧡

Actin filaments (black) and DNA (blue) in differentiated neuronal cells.



Read was born in the United Kingdom, moved to Norway while still a child — dad was English, mom Norwegian — a country which was beautiful and which she loved. Her early plan was to become a lawyer, but she was pulled by the dynamics of science, particularly neuroscience. "I think what I like most about science is that it is forever changing. Nothing becomes stagnant," she says. "You learn something then you learn something else then you have to modify your first opinion," she says. It was the pursuit of science that took her from a country of 5 million to the United States after she earned her PhD at the Department of Anatomy and Cell Biology at the University of Bergen. She completed her postdoctoral training in the Department of Pathology at Duke University and Department of Neurosurgery at Johns Hopkins University, before becoming a senior research associate at Duke University and the University of Bergen. She was on the faculty of Emory University School of Medicine and the University of Florida College of Medicine before coming to MCG in early 2021. She has served as director of the Pediatric Neuro-oncology Laboratory at Children's Healthcare of Atlanta and as a member of Emory's Winship Cancer Institute.

Vitriol earned his PhD in cell and developmental biology at the University of North Carolina at Chapel Hill, did his postdoctoral studies at Emory University and joined the University of Florida College of Medicine in 2014, where he was a member of the Center for Translational Research In Neurodegenerative Disease and McKnight Brain Institute. Vitriol, who fortunately does not appear to live up to the literal definition of his name, didn't know what he wanted to do until he enrolled at a community college in south Florida and met science teacher Dr. Charlie Ray. Dr. Ray taught every class — zoology, organic chemistry, introduction to biology — without notes, had two PhDs and gave tests that were impossible to finish on time but he took the time to give his students, who were willing to work hard, the opportunity to keep working on the tests until they got them right. Like Read, (and Ray's testing techniques) Vitriol began to see the magic of science's fluidity. Vitriol would transfer to the University of North Carolina to finish his undergraduate degree in biology (with honors) but is certain that if he had never met Dr. Ray, there is no telling what he would have become. He did like music and saw the rock band Phish 103 times, but there was the small problem that he had no musical talent. When he met Dr. Kerry Bloom, now chair of biology at UNC, and then an outstanding cell biologist and microscopist, the deal was sealed on research, on visualizing and better understanding the incredible, complex dynamic of even a single living cell as they tagged a chromosome, made a break in the DNA, then watched what it did to the chromosome's integrity. When he was doing his last interview for graduate school, Dr. Richard Cheney at UNC Chapel Hill showed him a movie of a cytoskeletal protein moving up and down the cytoskeleton. It looked like fireworks to Vitriol and imploded his plan to study DNA damage and repair and cancer at Duke University.

The couple in the lab and in life met while he was in graduate school and she was doing her postdoc in North Carolina.

Today he still finds the cytoskeleton a beautiful, and unbelievably dynamic structure and he, Read and their research team are helping define how it is far more than a cell framework. 🧡

Driving On

Neuro-oncology director Dr. John Henson is accelerating MCG's program for brain and central nervous system cancers.

BY DANIELLE WONG MOORES

Monday through Friday, neuro-oncologist Dr. John Henson climbs into his car, buckling in for the drive home from Augusta to Lake Oconee. Today, it's a podcast, tomorrow it may be an audiobook. He hits play, breathes and settles in for the ride.

After 30 years in the field of neuro-oncology, Henson still appreciates that ride, too, and is just as driven and engaged in caring for patients with a form of cancer that's often unsurvivable. He came to the Medical College of Georgia in January 2021 from Seattle, where he was medical director of the Ivy Center for Advanced Brain Tumor Treatment at the Swedish Neuroscience Institute, and director of the Swedish Neurofibromatosis Center. Prior to that, he worked for more than 20 years in academic medicine, including serving as an associate professor of neurology at Harvard Medical School and Massachusetts General Hospital and a clinical associate professor of neurology at the University of Washington School of Medicine.

Now, as director of neuro-oncology at MCG and at the Georgia Cancer Center at Augusta University, Henson is actively building a neuro-oncology program that's currently the only one in the state outside of Atlanta. He's focused on launching new clinics, advancing new clinical research and sharing an important opportunity: "to provide something of value," he says, for families throughout the region impacted by tumors and cancers of the central nervous system.

Developments

It's March — only about seven weeks into his time at MCG — and Henson shuts the door to his office, sends an email

through the AmWell program and, a couple of minutes later, is talking to and seeing his first teleneuro-oncology patient; it's a "big development, given the large area we cover in Georgia and South Carolina," he says, and one that he plans to grow.

Two times a week, he has already been holding joint multidisciplinary clinics: a neuro-oncology clinic with surgical neuro-oncologist Dr. Martin Rutkowski at the Georgia Cancer Center and a complex brain metastasis clinic with radiation oncologist and interim chair of the Department of Radiation Oncology Dr. John Barrett at the Georgia Radiation Therapy Center. "There are few patients we see who don't need to talk to two or three other doctors," he says, typically a surgical neuro-oncologist and radiation oncologist. Then there are the side effects of brain cancer and treatment that must be managed, such as seizures, mood changes, risk of infection and more. With a host of other neurological issues that always need to be addressed, the multidisciplinary format of the clinics — which includes Sheila Hall, BSN, nurse navigator, who provides patient coordination — eases that process significantly.

In May, he launched the state's first clinic for both adult and pediatric patients with neurofibromatosis, a rare and disfiguring genetic disorder that causes tumors to grow on nerve cells.

He's also establishing a Hereditary Cancer Clinic, which will offer multidisciplinary risk assessment, genetic testing and counseling, personalized surveillance and care coordination to patients and families with known or potential hereditary cancers, like breast

and ovarian; Henson co-authored the first medical textbooks in this area.

He also recently had a virtual meeting with a longtime colleague in Orlando to talk about the possibility of developing a southeastern brain tumor study group, which, he says, "if it takes off, could be massively exciting."

In March alone, the neuro-oncology program also worked to open three new clinical trials: one focused on immunotherapy for brain tumors, another testing a device developed by oncology company Novocure that uses electric fields tuned to specific frequencies to disrupt cell division and inhibit tumor growth, and still another studying a new imaging molecule to better identify brain tumors during scans.


The program's focus on clinical research is a differentiator, says Henson. What a robust clinical trials program can do is change not only the lives of current patients, but also the future of brain cancer care. "That's significant," he says.

Necessary Components

Most of the necessary components for a robust neuro-oncology program were already here at MCG and working — medical oncology, surgical neuro-oncology, radiation therapy, nursing, and social work — but "we didn't have the subspecialty neuro-oncologist to drive program development, and now we do," he says.

As a result, patient volumes, he says, have been "skyrocketing."

"If you look at cancer, it's one of the most feared diagnoses a person can get from a doctor, but within cancer, brain cancer is the worst because it's a tumor that affects your humanness," says



Henson's wife, Dr. Lily Henson, is a neurologist who moved into leadership and administration and is the CEO of Piedmont Henry Hospital in Stockbridge, where she served as chief medical officer in 2016. Prior to that, she was also chief of neurology at Piedmont Healthcare; Dr. John Henson also once oversaw Piedmont's cancer program.

Every day, she and Henson drive roughly an hour and 30 minutes to meet each other at their home at Lake Oconee.

Henson. “And so what we find is there’s a very, very urgent, deep need to get a diagnosis, formulate a treatment plan, and get the patient on something that can help them move forward with what is an emotionally devastating diagnosis.”

At the same time, with more than 100 different types of brain tumors currently classified, the five-year relative survival rate for certain cancers such as ependymoma or anaplastic ependymoma — which arise in the ependymal cells in the brain and spinal cord — can be as high as 92%, according to the American Cancer Society. That’s good news. That same rate for common and aggressive brain cancers such as glioblastoma — what Henson calls brain cancer’s “bad actor” — sits at 22%, but compare that to less than 1%, which is what it was 15 years ago.

Overall survival rates have increased dramatically within the last decade thanks to the addition of temozolomide to the chemotherapy regimen, says Henson. While that still leaves about 80% of patients with glioblastoma with a sobering diagnosis, certain patients also have targetable mutations that respond to small molecule inhibitors or immunotherapy. “There’s [also] a whole host of less common brain tumors, some of which are compatible with a normal life span,” he says. “For lymphoma in the brain only, we can cure two-thirds of patients. That’s cure with a capital C — it’s a big deal.”

Still, it remains a challenging field, and there can be no cookie-cutter treatments with brain cancer because of the delicate organ it invades: “Your brain is not only the control center of a lot of function, but it’s also what makes you human,” he says.

The Need

Brain tumors make up only 1% of all cancers, so historically, as the field of oncology began to subspecialize, the focus was on more common breast, lung and colon cancers. Neuro-oncology is relatively new, started only about 30 or 40 years ago, just as Henson was beginning his training. Memorial Sloan Kettering formed one of the nation’s first fellowships, which is where Henson went.

While the subspecialty is still uncommon — the U.S. trains only about 10 to 12 neuro-oncologists annually, says

Henson — “every hospital of any good size, particularly in academics, nowadays really wants a neuro-oncologist.”

“It turns out that even though [brain cancer] is only 1% of cancers, in a metropolitan area, enough patients need that care, so an organization can support a neuro-oncologist.”

Here at MCG, Henson is helping to train 12 neurology residents and eight medical oncology fellows, including one senior neurology resident he hopes will rejoin the institution following his fellowship in neuro-oncology.

The Beginning and the End

Their interest in the subspecialty likely mirrors his own. “I talk to trainees a lot about this,” he says. “During your training, you’re learning some facts, but what you’re really doing is you’re dating subject areas to see if one fits you. If you’re lucky, you find something that just fits your intellectual and your personal and your emotional interests extremely well.”

When he “met” neuro-oncology during his residency at Vanderbilt University, he says that it suited him.

“There was the science, [and] there was the ability to actually make measurements about your treatments,” he says. “In our area, things get bigger or smaller — you can actually see them [and] it’s very concrete.”

“I also appreciate the emotional aspects of taking care of brain tumor patients...It’s remarkable the emotional connections you can make with people facing problems like this.”

It’s a wonderful feeling to be able to help, he says, and he cherishes those relationships. At the same time, it’s also what has made the field harder as he has grown older and, in many ways, more aware of his own mortality.

Seeing his patients — many of whom he counts as his friends — suffer and experience life-shortening illnesses gets more difficult all the time.

So, every day, he’s driven to do more and to ensure that patients feel that value. “It’s like anything, the more focused you become, the more complicated things get...the fact that we understand that complexity and can help patients navigate it, they understand that right away.”



Suicide is a public health crisis that experts at the Medical College of Georgia are trying to change, whether it be through better education and prevention efforts or through the search for a biomarker that would indicate who is most at risk.



Broken Bonds

BY JENNIFER HILLIARD SCOTT

He was my ditto

Identical twins share an undeniable bond.

Pragmatically speaking, it could be because they share the same DNA. Anecdotally, identical twins have reported feeling each other's pain and joy and even being able to complete each other's sentences.

The bond began at birth for identical twins Ron and Don Graham. It was shattered on a hot middle Georgia afternoon when Ron committed suicide.

Arriving two months premature in 1958, at a little over two pounds, Don Graham was the "sick and puny" twin while Ron was stronger. "I didn't start thriving until they put me in the incubator with Ron," Don says.

Their bond endured, even through their teenage years — they shared a car as 16-year-olds and went on double dates. As

adults, they still spoke on the phone every day and even decided to pursue the same career as firefighters/paramedics — Don still works as fire chief and emergency management director for Jones County, Georgia.

"He was my ditto," Don says. "We just didn't do anything without each other. We were so much alike it was almost scary."

The morning of Aug. 8, 2015, Don was busy going about his Saturday ritual of cleaning the house, changing all the bedsheets, when his ditto called like he always did. Ron noticed his brother was out of breath and Don laughed and explained that he was exhausted from changing 11 pillowcases. Ron joked that he only had eight pillows and the two chatted some more about a hockey game Ron was excited to watch on television that afternoon. When they hung up, Don

texted his brother a picture of those 11 pillows piled on the bed.

Don still has that photo saved on his phone six years later. It was the last time he was able to text his twin brother.

Later that afternoon, Ron texted a friend about coming over to watch the game at his house in Cochran, Georgia. Sometime later, he placed his shoes beside his recliner, leaned the chair back, covered up with a blanket and shot himself in the head.

Back in Jones County, Don was in the grocery store when a friend called to tell him he'd heard a rumor that Ron had committed suicide. Don left his cart in the middle of an aisle and walked out of the grocery store in a daze. By the time he reached his car, the sheriff called confirming what he knew in his heart to be true.

His brother was gone.

"I had so many questions. I just didn't understand," Don says. "He had no history of mental illness or drug or alcohol use." He can only speculate that maybe all Ron had seen as a paramedic and firefighter took a cumulative toll and that letting his emotions pile up for so long eventually caused something in him to break, or maybe it was something else he didn't see.

"He absorbed everything and dispersed it in his own way. This was a way out. If he'd just given me the chance to do something," he trails off. "He knew there wasn't a thing I wouldn't do for him."

Don says one thing that helped ease some of the grief of losing Ron has been throwing himself into finding ways to ensure this doesn't happen to other people.

"Anything, I didn't care what it was, if it had something to do with suicide or people trying to hurt themselves, I wanted to be involved. I can relate to (people) through the simple story of my brother, through the love I have for my brother. Helping others is therapy," he says.

Broadening her focus

A little more than 100 miles away at the Medical College of Georgia, Dr. Martha Tingen has searched for the same type of therapy.

In her more than three decades as a nurse researcher, Tingen, Charles W. Linder, MD, Endowed Chair in Pediatrics and professor of medicine at MCG, has had a laser focus on prevention. She has helped prevent people from becoming obese, helped them stop smoking and exposing children to passive smoke. She has helped children and families develop healthier behaviors, like exercising and eating better, and helped ensure better cancer prevention education and increased access to screenings, particularly for the underserved.

Three years ago her focus broadened in a way she could have never expected.

On January, 17, 2018, Tingen's oldest son Nathan drove his truck to the Forks Area Trail System near Clarks Hill, South Carolina, and ended his own life at the age of 33.

It shook her to her core. "My life's work has been prevention and I couldn't prevent this. I couldn't save my son," she says tearfully.

Nathan's life had been the definition of success. He had All-American good looks. He had been a tennis champion in high school. He was a phenomenal recreational poker player who had won thousands of dollars. Like his mom, he worked as a nurse and had dreams of furthering his education and becoming a nurse

*The Tingen Family
(from left) Nathan,
Joseph, Martha and Jim*



anesthetist. “He didn’t have an enemy, he befriended everyone,” Tingen says. “He is one of the most tenderhearted people I have ever known, a true gentle giant.”

And then he was gone.

Tingen knows, she says, Nathan was experiencing a deep heartbrokenness and felt suicide was the only solution. “I feel like half of me died when he died,” she says. “He was my soulmate. I’ll never get over losing him.”

Her grief consumed her for months, and admittedly sometimes it still does. But Tingen, whose work, family and faith have always driven her, set her sights again on prevention. “I believe suicide is 100% preventable, but I also think people have to feel like they can reach out and call someone and be connected with resources ahead of the time they’re in crisis.”

She knew she had to find a way to prevent suicide from devastating other families.

The State of Georgia knew too.

The Georgia Department of Behavioral Health and Developmental Disabilities reached out to Tingen and asked her to apply for a suicide prevention grant.

She named the application Choose Life.

The first sentence on the application read “Prevention is so key to decreasing morbidity and mortality in so many diseases. However, when a suicide occurs, there is no longer an option for prevention.”

She received \$113,000 in funding to help address the suicide rates in Richmond County, which based on data from coroner’s reports had high rates of suicide. But the state had a catch. Tingen and her team also had to work with another high-risk county of the state’s choice.

The state’s choice was Jones County.

A problem they didn’t know existed

Located in middle Georgia, sandwiched between Macon and Milledgeville, Gray is the county seat of the largely rural Jones County. It spans around 395 miles and around 29,000 people live there.

Tingen knew of the town — she used to drive Nathan and his younger brother Joseph through on the way to Macon for tennis tournaments. What she didn’t know was that it had the 10th highest suicide rate among the state’s 159 counties. Among the 33 counties in the

Georgia Department of Behavioral Health and Developmental Disabilities’ Region 2, which also includes Richmond County, it had the highest.

Tingen and her team poured over all the data they could get their hands on — coroner’s reports and answers to the Georgia Student Health Survey, which every student takes annually starting in sixth grade. It asks questions like “Have you ever thought of harming yourself?” and “Have you harmed yourself in the last 30 days?” They couldn’t believe what they read. “The state had told us we would be shocked at the number of (suicides) there, and we were.”

Tingen and her team needed a partner — someone or some group in Jones County that could help them reach the community. They found that in Joy Carr, executive director of the Jones County Family Connection. Family Connections are state-funded collaboratives found across Georgia that are charged with bringing community partners together to develop, implement and evaluate plans to address issues that affect children and their families.

In her initial phone call to Carr, Tingen was her usual matter of fact self. “We want to figure out a way to get the community engaged and help develop strategies to prevent suicide,” she told Carr.

“I hadn’t realized how big the problem was,” Carr says. “We knew that we had a general lack of access to mental health services. We had, at the time, two therapy offices in town. We had been working through that and started by adding counseling services in all the schools, but when we got the report from DBHDD and looked at our suicide rates, that was a real eye opener.”

Carr began reaching out to community stakeholders and invited Tingen to present at an initial meeting with them in July 2019. Tingen told the group that day that her favorite job had always been being a mother and about the absolute devastation and trauma of losing her first-born to suicide. People began to cry. And then she told them about the similar devastation families in Jones County must be experiencing. “This is a real crisis,” she said. “We want to help.”



“I hadn’t realized how big the problem was. We knew that we had a general lack of access to mental health services. We had, at the time, two therapy offices in town. We had been working through that and started by adding counseling services in all the schools, but when we got the report and looked at our suicide rates, that was a real eye opener.”

– Joy Carr, executive director of the Jones County Family Connection

After the meeting, Tingen was approached by a man in tears — a man whose twin brother had committed suicide. He told her he'd never get over it and that he wanted to help however he could.

That man was Don Graham. He was the first person to respond to Carr's initial request for community stakeholders.

A new coalition

After that meeting, Tingen says it was clear community leaders were on board. Her phone and email started blowing up. The coroner was sending every suicide report and at one point there were five suicides in six weeks. "It was becoming more and more clear something needed to be done," she says.

Throughout her career, Tingen and her research team have followed the tried and true strategic prevention framework when making a plan to address an identified issue. This time was no different.

There are five steps: Assessment, planning, building capacity, implementing the work and evaluation.

The first step in Jones County was a community readiness assessment. On a scale of 0-9 with 9 meaning the

community was fully aware of a problem, had resources in place to address it and was making strides, Jones County scored a 2.

"Most people weren't aware there was a problem, but the good news was that most people there had faith that when they did become aware that community leaders would try to help solve it," Tingen says.

Next came planning and building capacity — reaching out across the community and figuring out the best ways to reach people. That effort birthed the Jones County Suicide Prevention Coalition with leaders from the school system to the coroner's office, from EMTs to the mayor's office. It was formed with three goals in mind: prevention by decreasing the stigma of reaching out for help; intervention to ensure that someone who has attempted suicide doesn't try again; and postvention — an organized response in the aftermath of a suicide to help family and friends start to heal and to prevent them from attempting suicide since they are at greater risk themselves. Implementation came in many forms, but with a simple goal — raising awareness and destigmatizing reaching out for help.

Volunteers stuffed bags with suicide awareness bracelets, pens and cards with

information on what to do and who to call if you or someone you know is suicidal. They gave them out at parades and other community events.

They sent 45,000 mailers to educate people about what to do if they were concerned about someone.

They hung banners at ballfields.

They hosted online chats with health professionals and survivors of suicide.

Teachers took part in Trauma Informed Care training to help them recognize at-risk students.

More than 160 people in the county were trained in QPR, which stands for Question, Persuade and Refer, a widely-used framework known as the CPR for suicide prevention.

Don Graham trained all of Jones County's paramedics.

The work has paid off.

Don Graham and late twin brother Ron (right)



Every month Tingen's team is required to provide a monthly progress report to the state, which includes the number of suicides in Jones County. From September 2020 through February of 2021, there were zero. At press time, there had been 1 since the end of February.

"We went from five in six weeks, to none in as many months. That was like a miracle to me," Tingen says, noting there is always more work to be done. "My (motto) is that one is too many."

Changing hats

Dr. Vaughn McCall, chair of the MCG Department of Psychiatry and Health Behavior and an expert in depression, insomnia and suicide, would agree.

"This is a major public health problem in this country," he says. "We've made such progress with so many other diseases, but suicide rates just continue to climb. Mortality rates (from suicide) have only increased over the last 20 years. Why?"

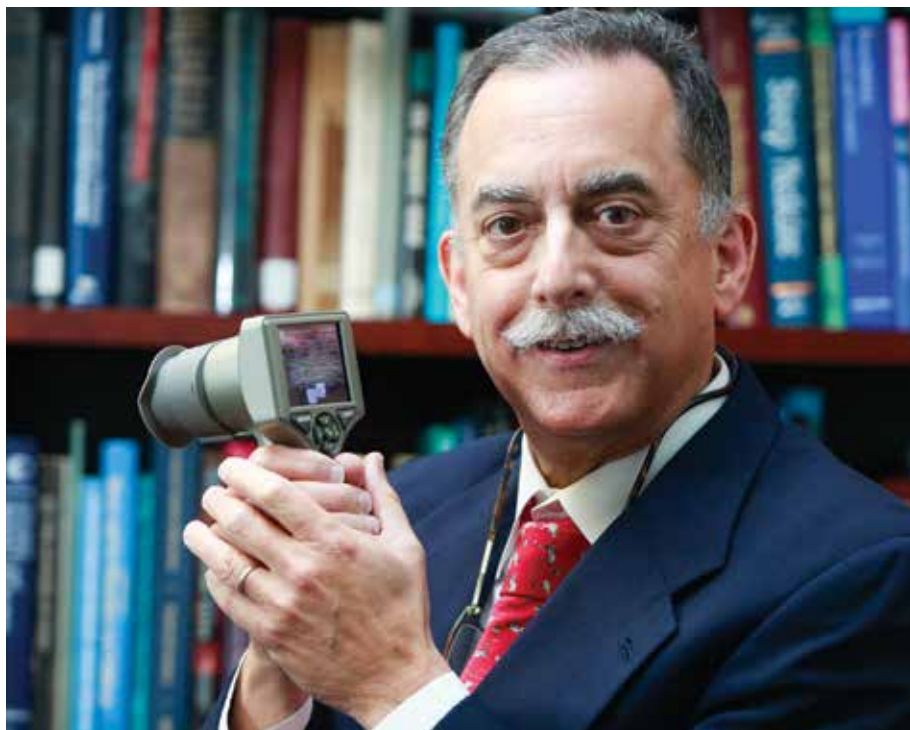
That's anyone's best guess.

According to a report from the American Psychological Association, there is hardly ever one single identifiable reason that someone commits suicide, making it harder to predict which people are most at risk. The tipping point is different for everyone — from depression or substance abuse for some; stressful life events like relationship problems or unemployment for others; to other factors like previous suicide attempts or childhood abuse.

Many physicians report being uncomfortable bringing up the subject to their patients.

Still another reason, and one McCall and his colleagues have identified as a major driver for suicide is insomnia. "If you have a patient who complains that their sleep has taken a turn for the worse then there is reason to open the door to a question about suicide," he says. "Insomnia is a risk factor for suicidal thinking, suicidal behavior and suicide death. That is universally true. It cannot be explained away, even controlling for other mental illness like depression and schizophrenia."

In the 2019 Reducing Suicidal Ideation Through Insomnia Treatment — or REST-IT — study, the first clinical trial looking



Dr. Vaughn McCall with the pupillometer

at whether targeted insomnia treatment reduced suicide risk, McCall and his colleagues found that people with severe insomnia could safely benefit from taking a sedative, in turn reducing suicidal thoughts.

"But then the question became 'why did that happen,'" McCall says. "Why is insomnia a risk factor for suicide? Why should treating insomnia mitigate suicidal thinking? For that, I had to take off my psychiatry hat and put on my sleep hat."

Always on go

Around 25 years ago in sleep research a new theory began to circulate. Many physicians treating patients with insomnia began to suspect that many cases weren't due to a sleep system that was broken, rather an "awake system" that was in overdrive. "It squashes the sleep system so it can't function," McCall says. "This hyperarousal theory describes patients that are always on go."

Many scientists began to hypothesize that if hyperarousal leads to insomnia and insomnia can lead to suicide, then maybe

hyperarousal could help explain suicide. This potential biomarker may offer one of the most promising pathways to predicting whose more at risk for suicide, he says.

The problem is how to find out who is always on go and who isn't.

Insomniacs "run hotter" than other people, so a rectal thermometer with an ambulatory monitor worn for 24 hours could tell us.

Insomniac's brain rhythms run a little faster, so an EEG could tell us, but not everyone has access to the necessary equipment.

There is a bundle of sympathetic nerves near the groin and impaling those could tell us how quickly the traffic is moving — it's faster in insomniacs.

"But there is no way any of those will ever have a practical application in a clinical setting," McCall says. "These are all physiological evidence that indicates insomniacs are in a state of hyperarousal, but you're not going to go into a psychiatry clinic and use any of them. It's great science, but you can't use it."

Knowing there had to be a better way to measure, McCall and his colleagues have turned their sights to the autonomic nervous system, a control system that acts largely unconsciously and regulates bodily functions like heart rate, digestion, respiratory rate and pupillary response, and is in charge of the body's fight-or-flight response. They've developed a handheld device called a pupillometer that measures and records how the pupil responds to a flash of light and think that will help them learn who has an overzealous autonomic response and, in turn, are in a state of hyperarousal — those whose pupils constrict and dilate quicker.

In an early study of 30 patients — 21 patients with suicidal ideations and nine healthy controls — they were able to show that the device was more powerful in separating the suicidal from the non-suicidal than just asking questions like “what’s your appetite been like?” and “how’s your mood been?”


“Can you can imagine if you were in a public mental health clinic, seeing a different patient every 20 minutes and you could walk around with this device and predict whose most at risk,” McCall asks.

We’ve asked all the questions

There are still more questions to be answered. Among them, if someone is in a state of hyperarousal and that leads to insomnia, will that uniformly lead to suicide? Not necessarily, he says.

“Think of it like atherosclerosis. In some people that will present as coronary artery disease. Some will present with a stroke. Still more with peripheral vascular disease. It’s the same condition but presents in different ways. The same is true with hyperarousal — it could present as insomnia, as suicide, as other things. There will be overlap with other types of mental illness.”

In fact, McCall and another MCG psychiatrist Dr. Brian Miller, who studies schizophrenia, are looking at the pupils of patients with schizophrenia to see if they’re in a state of hyperarousal and whether that makes them more at risk for suicide.

If the device did nothing more than help determine which patients are most at risk and may, for instance, need to be seen more frequently in the clinic, “that would be useful information,” McCall says. “Is it truly possible to prevent suicide? Well, how successful have we been at preventing myocardial infarctions? You just do the best you can. But, we need something that is going to help while patients are actually in clinic. We’ve focused for too long on asking people questions. We’ve asked all the questions. We need a biomarker.” 

Ranking 10th among the leading causes of death in the United States, suicide rates only continue to climb. They increased 35% over the 20-year period from 1998-2018, according to the National Institutes of Health.

In 2019, nearly 47,500 people died by suicide, 1 every 11 minutes; 54% did not have a known mental health condition.

SUICIDE NUMBERS for Georgia in 2019:

- **2nd leading cause of death for ages 25-34**
- **3rd leading cause of death for ages 15-24**
- **4th leading cause of death for ages 35-44**
- **10th leading cause of death for all Georgians and in the U.S.**

Georgia’s average age-adjusted suicide death rate for 2018 was 14.5 per 100,000 people. Rural counties have a higher suicide rate per capita, so although 79% of suicide deaths in 2018 occurred in non-rural counties, the rates in rural counties are actually higher due to the smaller population sizes.

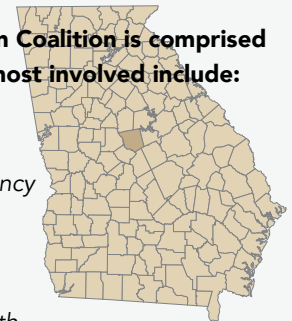
As of Feb. 1, 2021, the state has increased Choose Life grant funding to \$226,000 annually and the team is expanding their work into Columbia County, a neighbor of Richmond County, where work similar to that in Jones County has also been taking place.

Sources:

American Foundation for Suicide Prevention
Centers for Disease Control and Prevention
Georgia Department of Behavioral Health and Developmental Disabilities
Georgia Department of Public Health

While the Jones County Suicide Prevention Coalition is comprised of nearly 80 community members, those most involved include:

- Joy Carr**, *Family Connection*
- Travis Killen**, *Board of Education*
- Don Graham**, *Emergency Management Agency*
- Scott Walston**, *Gray Memorial Chapel*
- Donald Black**, *Family Connection*
- Debbie Lurie-Smith**, *Jones County News*
- Jordann Cohen**, *River Edge Behavioral Health*
- Debra Daniel-Purnell**, *Retired from Department of Juvenile Justice*
- Brandi Cunningham**, *Division of Family and Children Services*
- Shana Johnson**, *Operation Early Intervention*
- Patty Gibbs**, *Family Counseling Center of Central Georgia*
- Dianna Gonzalez**, *Bibb County Department of Community Supervision*
- Sandra Hammack**, *First Baptist Church of Gray*
- Matthew Jarratt**, *Deputy Coroner and Gray Memorial Chapel*
- Kerisia Wasztyl**, *Navicent Health*



If you or a loved one are experiencing a suicidal crisis, go to the nearest emergency room. The MCG Department of Psychiatry and Health Behavior can be reached at 706-721-6597.



Dr. Turner Wayne Rentz, '72
President, Medical College of Georgia Alumni Association
Campus Associate Dean, MCG Southeast Campus
Surgeon, Southeast Georgia Health System



My personal journey with the Medical College of Georgia began in Augusta at the old University Hospital, long before MCG's current teaching hospitals were ever conceived.

My father was a junior medical student at MCG when the car he was driving, with my mother in the passenger seat, was t-boned by a car full of nuns, who I am sure bore my mother no ill will.

Fortunately, no one was seriously injured, but my mother went into labor later in the day and I was born in University Hospital a few weeks premature.

After my dad graduated from MCG and did a rotating general internship in Greenville, South Carolina, he moved back to his hometown, and like many others right after WWII, hung up his shingle. He joined an older physician and they taught each other as they took care of the people in the community. MCG's legacy was a familiar part of my childhood — I grew up hearing about the medical school, seeing that unique skull and crossbones ring on his hand and watching him practice his art.

When it came time for me to apply to medical school, I, of course, followed his lead and applied to MCG — best move I ever made. After medical school I trained in surgery at the Naval Regional Medical Center at Portsmouth, Virginia, with residents, students and faculty from all over the country. I felt like my MCG education prepared me better than many and certainly better prepared me clinically. That was MCG's legacy at work in me.


As campus associate dean at our medical school's Southeast Campus in Savannah and Brunswick, I am now privileged to be a part of instilling that legacy in future generations of MCG-trained physicians.

As president of the 130-year-old MCG alumni association, I was honored to speak to our newest colleagues at this spring's Hooding Ceremony. I told the medical school's 185th graduating class that they were now the bearers of that nearly 200-year-old legacy.

They will be mentors and role models to many who will follow their lead as they grow and mature. I know they will lead wisely, as so many of you have and continue to do.

They are the word of mouth ambassadors and representatives of our medical school. Our reputation depends on them. I know that their knowledge, experiences and input to this association will influence the course for many more generations to come. Their contributions, like yours, will be invaluable to our medical school's growth.

I hope that they, and you, will continue to stay engaged with your medical school and help us ensure that every medical student continues to benefit from the same support and superior education that you received.

My best to you always. 

Zooming Career

BY JENNIFER HILLIARD SCOTT

Drs. Drew and Kaylar Howard, '92, had already established a scholarship to attract the best and brightest students to the Medical College of Georgia.

Dr. Anil Puri, '05, had named a learning community, a quiet place for medical students to study and gather with classmates, in the J. Harold Harrison, M.D., Education Commons.

The Howards, Tifton, Georgia OB/GYNs, and Puri, a Milledgeville, Georgia, internist who specializes in sleep and pulmonary medicine, also both teach MCG students in their respective practices.

Now these MCG alums are finding a new way to give back to their alma mater — through the next generation.

The Howards and Puri are part of a group of alums who have been participating in virtual career advisory panel discussions with first- and second-year MCG students.

“Choosing a career is one of the single most important decisions that an individual will make, short of choosing a life partner,” says Dr. Jennifer Tucker, '97, a pediatric emergency medicine physician who serves as MCG’s assistant dean for career advising and fourth-year class dean. “In our traditional four-year curriculum, students have to choose what they will do for the rest of their careers after only 2-and-a-half years of medical school — that’s even earlier now for students in our 3+ (Primary Care) pathway. That choice often comes without direct exposure to the

Dr. Kaylar Howard



Dr. Anil Puri



wide range of specialties available to them or the breadth of practice options — in an academic versus community setting; or inpatient versus outpatient, for example.”

While students do have access to “high touch” career advising, early and often, and an advisor assigned to them throughout their four years at MCG, the panel discussions are designed to be another way to allow students to sort of “pick the brains” of those that have gone before them.

“We’re looking for any contact we can get with physicians, so we really appreciate this. It’s really helpful,” explained MCG Class of 2024 student Natalie Zink, who served as moderator for several panels, including the late March Panel featuring Drs. Howard and Puri and others.

The discussions have given students a space to ask questions about anything and everything — from how they chose their career to what they love and hate about it; from questions about work/life balance to examples of how their career has changed over the years.

Examples like the one Dr. Kaylar Howard gave them about how OB/GYN has changed from when she was a resident — when she was one of three first-year residents and it seemed she was on shift or on call 24/7, leaving her deliriously questioning whether she’d made the right career choice after all.

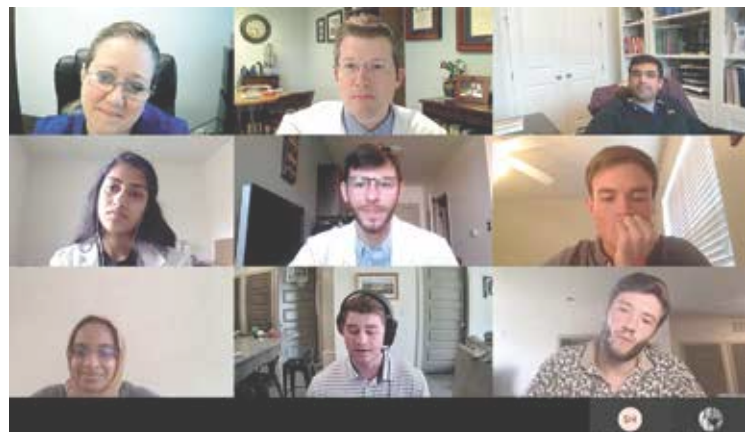
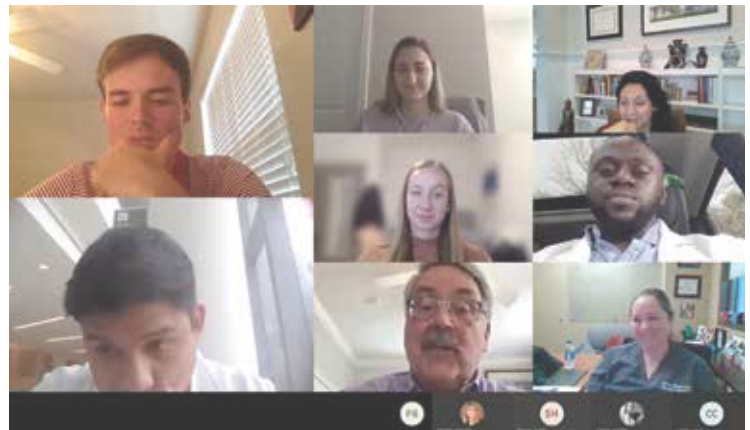
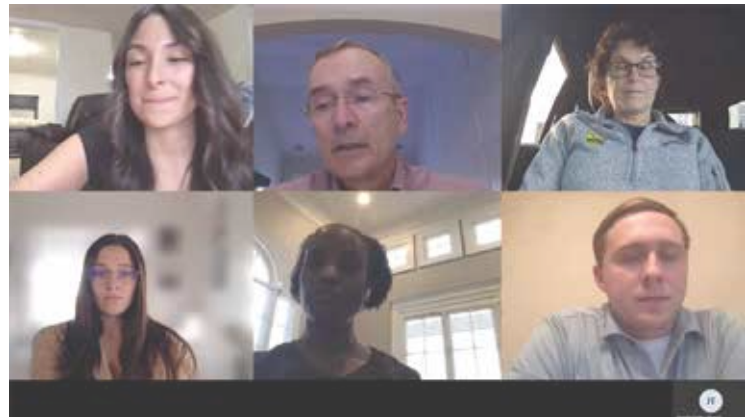
These days, the Howards — owners of the Howard Center in Tifton and a new office in Covington — have been just one of many practices who have instead developed a system that divides obstetrical care from gynecological care, and allows physicians to choose how they want to practice. Some people in their practice work 48 hours straight delivering babies and then they’re off. Others work a more 8-5 shift, taking care of gynecological needs.

“I think that’s the way the field is shifting,” she told students. “We have a system here where you can do one or the other or both, just on different days. You can still have a life. In the old days you worked until you were nearly dead and then quit OB, and it’s just not like that anymore.”

Puri says it’s that real-world seasoned perspective that he hopes he can offer as well.

“When I was a student we didn’t get a lot of career advice, unfortunately,” he says. Although the regional campus model that sees MCG students learning in private practices and community hospitals across the state has changed things, back then “If you were in an academic setting, like MCG, from start to finish, your mentors were (mostly) in academics. I want to be able to help provide that private practice perspective. I see this as an opportunity. I think it’s just another really cool way to be able to give back.” 🍀

Advisory panel discussions via Zoom



Never Taken for Granted

John West, '85, was saved thanks to the friendship of two MCG graduates

BY DANIELLE WONG MOORES

The story begins more than 120 years ago, when Robert M. West Sr. graduated from the Medical College of Virginia in May 1900. Or maybe it begins when Robert M. West Jr. — Bob — earned his degree from the Medical College of Georgia in June 1955.

Or maybe not.

John — Bob's son and Robert's grandson, who graduated from MCG in 1985 — keeps both of their diplomas on his wall.

John knows that his grandfather was a general practitioner in Salisbury, North Carolina — he has his original office ledger of daily office visits, with charges and payments including cash and also chickens, eggs and vegetables. John knows that Bob, once a professional saxophone player until he decided to follow in Robert's footsteps, tried for four years to get into MCG; it took another seven years of part-time study for him to graduate as a general practitioner.

John West also knows this — and this is actually the real story: Had his grandfather not become a doctor, and had his father not persevered, he would not be here.

A first-born son

It was December 1955, and Dr. Bob West and his wife, Mary Alyce, were preparing for the Christmas holidays. Life was not perfect, but it was good: Bob was completing an internship at Atlanta's Grady Memorial Hospital — there was no residency requirement at that time — and the couple would soon be moving to Forest Park, Georgia, to start his practice.

Then came a call from Dr. Jack Raulston, '53, Bob's former MCG cadaver lab partner and good friend. Raulston was an OB/GYN practicing in Palm Beach, Florida, and a pregnant woman with five other children had come into his office with a desperate request: Although abortion was illegal at the time, she

wanted his help to end this unplanned pregnancy.

Raulston knew the Wests weren't able to have children. So, he asked the mother if she would consider adoption; she said yes, and Raulston immediately put in a call.

Six months later, Mary Alyce flew to Palm Beach alone, and came back to Atlanta with a newborn baby, the Wests' first-born son, John.

Coincidental Circumstances

West grew up knowing his story, and so did his friends — “So my adoption never became a weapon used to tease, and if they tried it, I had one great comeback: ‘My parents had to go get me, so they really wanted me.’ ... it was never thought to be a shameful thing.”

West also knew all the other family stories. Bob used to ride with his own father in a horse and buggy to call on patients. After playing saxophone with big

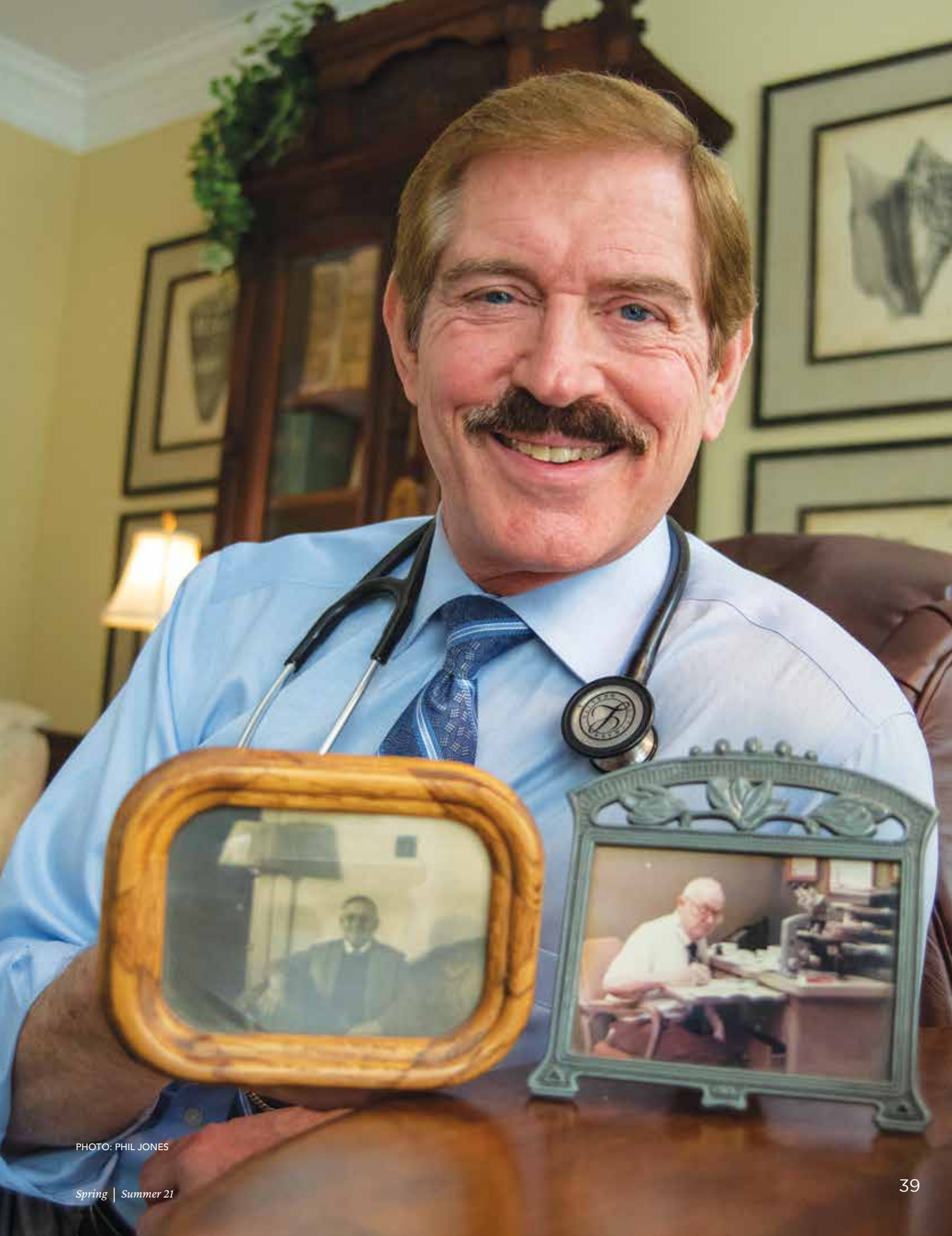


PHOTO: PHIL JONES

bands for a time, Bob started studying to be a doctor, but had to start his dream all over again when the medical college at Atlanta's Oglethorpe University was discredited and closed. Then, his MCG application kept getting blocked by one faculty member who didn't believe he had the aptitude for medicine.

Bob and Mary Alyce worked to save money — Bob as a research assistant for Dr. Robert Greenblatt, internationally known for his work in reproductive endocrinology, while Mary Alyce was a secretary for Dr. Hoke Wammock, '28, who established MCG's then Department of Oncology — while he kept applying, until he finally was accepted. Then, over the next seven years as Bob attended MCG part-time, he was shoulder to shoulder with the giants — Greenblatt and Wammock, along with G. Lombard Kelly, president of MCG from 1950-53; Edgar Pund, chair of the Department of

Pathology who was named president in 1953; and others.

West drank it all in, including the fact that Bob graduated with honors and was a member of the Alpha Omega Alpha Honor Medical Society. So, it didn't seem at all surprising for him at age 5 to declare to his Pop, "I want to be a doctor just like you."

Three years later, West was going on house calls with Bob, just as Bob had done with his father. At 10, he was helping in the office, whether that was handing him instruments, sterilizing equipment, or wrapping sutures. Bob would even often wake West when he had an emergency call as the company doctor for Georgia Power, American Can and Crown, Cork, and Seal, or in response to a car accident or other situations. The scenes could be traumatic, "but those situations helped shape who I would become," says West. "And at that young age, I knew that each incident was a learning experience."

After high school, West decided to attend the University of Georgia — the fact that he was dating his future wife, Lee, his high-school sweetheart, had more than a little to do with it. The choice of medical school was even easier: "Pop had a lot to do with that," he says. "I don't recall applying anywhere else [but MCG]."

He was finishing up his last requirements for graduating UGA and had completed an interview for MCG when the first rejection letter came. He wasn't going to give up, but in the meantime, joined the Georgia Department of Human Resources in downtown Atlanta as a senior laboratory scientist in the virology lab. Every day after work, he'd stroll two blocks to Georgia State University, where he was taking graduate-level courses at night to stay academically active.

He and Lee married that fall, then a month later he walked up to the sign-in table at UGA to retake the MCAT when he

Photos of Mary Alyce & Bob with Bob's 1955 MCG class ring, as well as his father's pocketwatch



PHOTO: PHIL JONES

stopped and walked right back out. “I knew with everything going on and us getting married, I hadn’t spent any additional time trying to study or to improve,” he says. “At that moment, I had the awareness that if I took the MCAT again and did worse, it would worsen my chances.”

He applied again based on his original MCAT scores — then was rejected outright, without even an interview.

Remembering how Bob hadn’t given up, West took the day off from work to drive the nearly three hours to Augusta. He walked into the student center and into the office of Dr. Jim Puryear, then director of student affairs, and asked to speak with him.

As West had no appointment, Puryear’s secretary said, “You’ll have to wait.” “So I sat down in his outer office and waited,” says West. “My knowledge of Pop’s experience and perseverance to become a physician had taught me that you have to work for what you want and not to let

So West responded, “OK, I’ll get them and I’ll be back.”

The meeting took all of 10 minutes. But West had his marching orders. For the next eight months, he studied for 2.5 hours a day — during breaks at the lab, with permission from his manager — while continuing to work full-time and take night classes. He retook the MCATs and raised all of his scores, including doubling his physics score, and reapplied with high hopes.

Then it was February, and friends were getting acceptances, but not West. Finally, the invitation for an interview came, but in June, another letter: He was an alternate.

Lee was worried, so as a backup plan, West applied and was accepted to UGA’s landscape architect school. It wasn’t as out of left field as it might seem: He enjoyed building and nature, and his nextdoor neighbor growing up was a landscape architect.

Then late summer, after he came home, Lee greeted him with shining eyes. In her

Three Lifetimes

“To me, it was a lifetime of preparing [to be a doctor],” says West. Or, even three lifetimes, thinking back to not just his father but also his grandfather, three generations of physicians.

It was also due to MCG, which connected Bob to Jack Raulston and saved West.

What he appreciates most about his time at MCG is the medical school’s training philosophy: “The people who trained and taught me always did so in a way that I always felt they were there to help me be the best,” he says. “They wanted you to go out with confidence.” After graduating and completing his internship and family medicine residency at MCG, West opened a private practice in family medicine in 1988; today, he’s still seeing patients, having just opened a new practice with Atlanta’s Northside Gwinnett Hospital. In addition to family medicine, West has also been board certified in clinical lipidology since 2008, and his practice now involves adult medicine with an emphasis on adult primary care and prevention, focusing particularly on atherosclerotic coronary artery disease prevention and aggressive treatment of lipid and lipoprotein disorders. In more than three decades of medical care, West has received multiple awards, including being named 2017 Physician of the Year by the Gwinnett County Chamber of Commerce for his work in lipidology for coronary artery disease prevention and treatment.

“I was saved,” repeats West. “I never took that for granted.” And now, with 30-plus years in practice, “There are people I’ve seen, whom I know as a physician, whose lives I’ve saved. Had I not been here to begin with, that might not have happened. I have tried to give something back every day to honor the fact that I am here. If not for two MCG grads, I would not be here today.” 🍀

“I was saved. I never took that for granted.

There are [patients] I’ve seen whose lives I’ve saved.

Had I not been here to begin with, that might not have happened. I have tried to give something back every day to honor the fact that I am here. If not for two MCG grads, I would not be here today.”

—Dr. John West

anyone else tell you that you were not good enough to do it. I was not going to leave until I had talked to this person.”

He waited from 10 a.m. to 4 p.m. — the secretary took pity on him and gave him a lunch ticket to the downstairs cafeteria — until Puryear finally was available. West had one question ready: “What do I need to do to get into MCG?”

There was more waiting as Puryear pulled out a file on West and flipped through it. “I remember him as a matter-of-fact, but kind person,” says West. “He looked over everything, he didn’t dissuade me...but he matter-of-factly said, ‘You need better numbers.’”

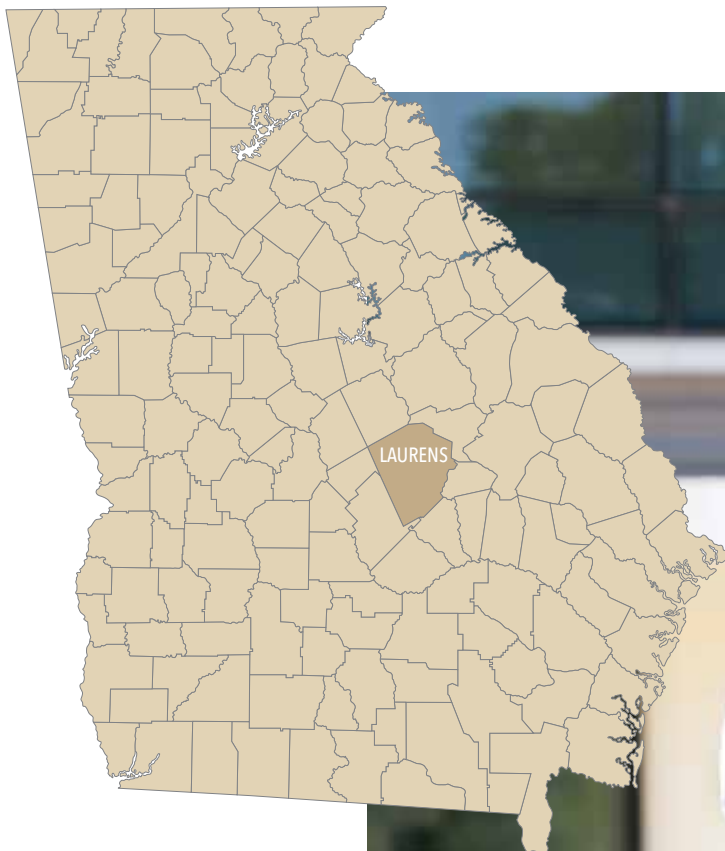
hands was a registered letter. “Back then, you knew that you were going to be accepted because the letter came by registered mail,” West says. It was signed by Dr. Puryear.

He and Lee immediately drove the 15 miles to Forest Park, where Bob and Mary Alyce still lived. “I walked in and handed him the letter, and he got all teary,” says West. Bob passed away in 1997 at age 82, an MCG-trained physician for 41 years. But in West’s mind’s eye, “I can still see him... He didn’t say anything, just hugged me and cried.”

Critical Shortage

As one of the fastest growing states with some of the worst health outcomes in the country, Georgia's supply of frontline primary care physicians is reaching a critical shortage. With a recent gift from Peach State Health Plan, and matching funds from the State of Georgia, the state's only public medical school is working to incentivize students to pursue primary care careers in rural and underserved areas.

BY JENNIFER HILLIARD SCOTT



Scotty Hall, part of the first group of students to commit to the 3+ Primary Care Pathway

L Laurens County, near middle Georgia, is one of the state's largely rural counties. Also one of the biggest, it spans 818 square miles from end to end.

While the county seat of Dublin is home to two mid-size hospitals and an adequate number of physicians, Laurens County native and Medical College of Georgia student Scotty Hall says there are stretches of the county that go on for miles and miles where there's absolutely nothing.

That includes physicians.

Growing up in Dexter, a town with a population of less than 600 people that sits about 12 miles northeast of Dublin, Hall says he clearly remembers at an early age noting the stark differences those miles could make when it came to accessing health care.

"There are no doctors, none, that I know of in Laurens County that exclusively practice outside of Dublin," he says. "That fact made me understand the differences you can see (in access to care), really within a small physical area."

It's a scenario that's often repeated across Georgia.

Despite consistently ranking among the top 10 in terms of population growth, the state consistently ranks among the 10th worst for many of the country's most prevalent medical conditions, averaging some of the



highest rates of stroke, heart disease and cancer in the nation in the face of a shortage of primary care physicians and lack of access to basic health care services.

Nine counties in Georgia have no physician; 60 no pediatrician; 76 no OB/GYN; 18 no family medicine physician; 32 no internists; and 74 no general surgeon.

With help from a recent \$5.2 million gift from Peach State Health Plan, and recently announced matching funds from the State of Georgia, Hall and some of his classmates will soon get the chance to help change those numbers.

He and seven other second-year students are the first cohort of MCG students to commit to the medical school's new 3+ Primary Care Pathway. Part of one of the most comprehensive redesigns of MCG's curriculum, shortening the core MD curriculum from four years to three for all students, the 3+ Primary Care Pathway would see a percentage of each medical school class commit to primary care practice, then graduate in three years and immediately enter a residency in Georgia — either in emergency medicine, family medicine, general surgery, internal medicine, OB/GYN, pediatrics or psychiatry.

In exchange for service to a rural or underserved area of the state, those students will receive a scholarship — provided in part by the funds from the Peach State Health Plan gift and the matching state funds.

“In October 2020, Peach State Health Plan launched the Office of Rural Health and Strategic Initiatives to develop programs and partnerships with organizations like the Medical College of Georgia at Augusta University,” says Wade Rakes, CEO of Peach State Health Plan. “By partnering with the state's only public medical school, we are doing our part to ensure that all Georgians have access to highly trained physicians and quality health care.”

“As the state's only public medical school, it is our duty to ensure we help produce the physicians we need to keep Georgia healthy,” adds Dr. David Hess, MCG dean. “The physician shortage, especially in rural and underserved areas, is a huge problem that will only worsen over time. Primary care physicians help improve a community's overall health by managing chronic conditions on an outpatient basis. They also bring more jobs, attract new businesses and

strengthen the economy. By placing more of them in underserved areas, we'll help ensure a healthy future for every corner of the state.”

Ensuring that healthy future and improving access to care for people who live in places like his tiny hometown has always been part of the plan for Hall, who says he hopes to pursue a career in general surgery.

“Going back home to practice or to a similar area — with a similar need for physicians — has always been my goal. That's what I grew up knowing and I feel like there's an acute need that sometimes gets overlooked,” he says. “MCG does an excellent job prioritizing the needs of the people across the state and especially people in rural areas of the state, but outside of here, many people just don't realize how big the need is. Outside of Atlanta, it's easy to find rural and underserved areas.”



“In October 2020, Peach State Health Plan launched the Office of Rural Health and Strategic Initiatives to develop programs and partnerships with organizations like the Medical College of Georgia at Augusta University. By partnering with the state's only public medical school, we are doing our part to ensure that all Georgians have access to highly trained physicians and quality health care.”

– Wade Rakes, CEO



To donate to MCG's 3+ Primary Care initiative, visit mcfoundation.org/moremcgdoctors



By Mary L. McCormack,
Associate Vice President for Gift and
Estate Planning for the Medical College
of Georgia and Augusta University

**Important Extended Provision
of the original CARES Act
(Coronavirus Aid, Relief, and
Economic Security)**

Donors who itemize their deductions can now give more cash to charity before reaching their adjusted gross income (AGI) limitation. Formerly set at 60%, the limitation for cash contributions to certain public charities has now been raised to 100% of an individual's AGI for both 2020 and 2021. Any giving beyond this 100% limitation may be carried over and used in the next five years.

Leaving a Legacy and Inspiring Others

When Dr. David Parks graduated from the Medical College of Georgia in 1984 and went on to become an otolaryngology resident at his alma mater, he had no idea his enthusiasm for his beloved medical school would result in a \$1 million endowed chair 35 years later.

It was the mid-1980s when BioLab founder and known philanthropist Leon Bloom approached his friend and attorney Sydney Parks about making a meaningful gift to a medical school through his estate plans. Knowing of his son David's fondness for MCG, Parks convinced Bloom to tour the campus and meet then-president Dr. Jesse Steinfeld to discuss the possibility of a major gift. One stop on that tour was the neonatal intensive care unit, where then-Chief of Neonatology Dr. William Kanto showed them around.

"My dad, Mr. Bloom and I went to dinner in Augusta that night and it was clear that the NICU in particular had just made such an impression on Mr. Bloom," remembers Dr. Parks, now an Atlanta head and neck surgeon. "He (Mr. Bloom) grew up during the Great Depression with nothing and here he is giving \$1 million."

Bloom founded his successful business, BioLab, in his home basement in Decatur, Georgia, in 1955, where he created an algacide useful to the rapidly growing pool industry as well as other innovations, earning the moniker "godfather of modern pool care."

After the influential visit to MCG, and with his attorney's assistance, Bloom decided to leave his legacy through a designation in his trust to create the Leon and Dorothy Bloom Chair in Medical Research at the Medical College of Georgia, which now honors the late Mr. and Mrs. Bloom. MCG received the estate gift of \$1 million in late 2020.

Named chairs and professorships like the one named for the Blooms allow

MCG to recruit top-tier faculty members, continue to provide excellent educational opportunities to medical students, and conduct cutting-edge research. The establishment of the Leon and Dorothy Bloom Chair in Medical Research will allow this focus on research to continue in perpetuity.

With the simple act of sharing his passion for MCG with his father, Dr. Parks created a ripple effect that will impact not only future MCG students but will serve the health and wellness of the citizens of our state and beyond for generations.

When MCG alumni and supporters show enthusiasm for MCG to others, such as advisors who help guide clients in estate planning, or close friends or colleagues, they are also inspiring others to reflect on how their legacy and their values align.

In its most personal meaning, a legacy is how one lives on after passing – how to ensure that one's values carry forward and have lasting meaning.

Whether inspiring others, thinking about how you want others to remember you, or how you want to honor others, there are many ways to leave a legacy in support of MCG. Gifts through wills and trusts are most common, but gifts designated from IRAs or retirement funds may be the most advantageous for your loved ones and are also the easiest to put into place. Gifts of assets, such as appreciated stock, real estate and artwork may also be good options for you to consider. We are here to help you explore options to make a meaningful difference for the institution that you love and we are here to help you consider different strategies to talk through with your advisors.

We are grateful to be part of continuing the legacies of Mr. and Mrs. Bloom. ♡

For more information on creating a legacy, contact Mary,
706-721-5027 or Mmccormack1@augusta.edu.

A Seed That's Bloomed

Betty and Lovick Corn's extraordinary endowment 20 years ago to MCG pediatric cancer researcher Dr. David Munn continues to bear fruit

BY DANIELLE WONG MOORES



It was 2002, and Dr. David Munn, '84, a pediatric cancer researcher at the Medical College of Georgia, walked into the President's Conference Room in the G. Lombard Kelly Administration building for a meeting with Betty and Lovick Corn of Columbus, Georgia. The older Southern couple was gracious and lovely, recalls Munn. They were also asking him whipsmart questions about the research he was hoping to conduct to identify more effective and less toxic treatments for children with cancer.

He must have done something right. He left that day with a commitment for a \$1.5 million gift from the Corns, which with subsequent giving has turned into \$4 million to date.

On that extraordinary day, that gift by Betty and Lovick Corn was the seed, allowing Munn to pursue the research he most wanted to do. Now, just shy of two decades later — a blip in the timeline of most translational research — Munn's pediatric cancer bench science has translated into clinical trials, which have helped extend life or provide improved quality of life for children from around the world with the worst types of brain cancer. And he's not stopping.

Funding hope

A photo of Betty and Lovick graces the office of Teddie Ussery, founder of Family Office Matters, who has helped manage the Corn family's BELOCO Foundation and philanthropy for the past 40 years. Betty smiles graciously, while Lovick stands protectively over his wife.

Yet, if you look closer, there's a sense of restlessness. The Corns — as anyone who knows them can surely imagine — appear to be pausing just for a moment, ready and eager to take on the next thing.

PHOTOS: COURTESY OF THE CORN FAMILY

Lovick grew up in Macon, and Betty in Columbus. She was a Turner but also part of the W.C. Bradley family, which owns W.C. Bradley Co., still a manufacturing company and a major investor in The Coca-Cola Co. She also graduated from Wesleyan College and was determined to make a difference in her work as a community volunteer. He was the son of Emory-trained urologist Dr. Ernest Corn (one of the first in the field in Georgia), graduated from the University of North Carolina-Chapel Hill, and served in the U.S. Navy during World War II in the Southwest Pacific Theater and the Philippines Campaign as commanding officer of the USS Apc42.

The two met on Sea Island after the war while their parents were vacationing there. Betty met Lovick's brother Tom first, but it was Lovick who captured her heart. After their wedding in Columbus in 1949, they lived briefly in New York City while Lovick worked in

sales for Bibb Manufacturing Company, but when Betty became pregnant with daughter Elizabeth, they came home, where Lovick joined the family company.

He would rise to become vice chairman at the W.C. Bradley Co. and of the associated Bradley-Turner Foundation and was also founder and chairman of the foundation he and Betty formed, the BELOCO Foundation. It was an inside joke, that name, whose letters were taken from BETty and LOvick CORn.

Both Betty and Lovick grew up with a strong family philosophy that they would pass on to their five daughters — “that we’ve all been tremendously blessed and we owe it to give back,” says Elizabeth Corn Ogie, who herself has served on the MCG Foundation. “We were raised, from the time I was little, [to believe that] you volunteer, you help in the community, you serve on boards, you give back. And that’s the broad family as a whole, the 100-plus of us all were raised that way” — and not only to give back but to do so with humility.

Originally, Betty and Lovick’s giving was generally focused on the arts and Methodist education. Pediatric cancer became a more specific interest thanks to Dr. Denny Hammond, a fraternity brother and close friend of Lovick’s, who headed the Division of Hematology/Oncology at Children’s Hospital Los Angeles, served as group chairman of the Children’s Cancer Study Group, and was president and CEO of the National Childhood Cancer Foundation, now known as CureSearch for Children’s Cancer. The Corns often traveled to California to visit Hammond and his wife, which is when Hammond and Lovick would no doubt swap stories of their work.

“Back in the day, pediatric hospitals each did their own treatment for different childhood cancers,” explains Elizabeth — meaning that a child in Minnesota might not receive the same advanced treatment a child in, say, California might. In the late ‘90s, Hammond was among the new thinkers at the time who wanted to ensure no children with cancer would needlessly

Elizabeth Corn Ogie





Betty and Lovick Corn with daughters Elizabeth and Polly

suffer or die simply because a new treatment hadn't yet been communicated to their region of the country. "Mama and Daddy bought into it, for sure," says Elizabeth. "They had five kids and grandkids — and it just makes so much sense that the best protocol ought to be shared throughout the pediatric hospital networks."

Then, their second daughter, Polly, was diagnosed with Hodgkin's lymphoma at age 30.

Polly's son, Gilbert Miller, was only 3 at the time. Although the cancer was even then highly treatable, Gilbert, now a father himself, can imagine what his grandparents went through knowing that

their child — even their adult child — had been diagnosed with cancer. "I think when you start to look at the spectrum of this, she was young enough where you go, 'Wait, she's too young to get cancer.' Then you find out, 'Well, yeah, there's kids that are two weeks old that have cancer.'

"My grandfather never talked at length about anything," Gilbert says, "but what strikes me is this sense of justice in him and how he just felt like certain things were unjust. And I think they could see a mechanism to say, 'We can help bring some justice to this by funding this and accelerating these studies so these parents and these kids have hope.'"

All about the research

It was another family friend, Dr. Cecil Whitaker, '62, who led the Corns to Munn at MCG. Much of the Corns' giving was already locally focused, so supporting pediatric cancer research in their home state likely appealed. Whitaker, an OB/GYN living and practicing in Columbus, not only served as a member and chair of the MCG Foundation (and remains a chair emeritus), but he also is a member of the MCG Advisory Board.

Ussery believes that the uniqueness of Munn's research also caught their attention. Immunotherapy was in its infancy at the time, and "the idea that it was a different strategy that had been thought about...it was a new idea ... that would bring hope to this disease," she says. "It intrigued them in that they wanted to see where this could go, and it was targeting pediatrics. Those two were the keys, I think."

The meeting with the Corns couldn't have come at a better time for Munn. Although he was and is a pediatric cancer researcher, he was then conducting research into cancer immunotherapy focused entirely on adult tumors, both melanoma and breast cancer models, funded by the National Institutes of Health. It's a difficult fact, says Munn, but "the problem with developing new treatments for kids with cancer is that there's not really a funding constituency for basic research on kids with cancer because children don't constitute a large market."

About 16,000 new cases of pediatric cancer occurred in the U.S. in 2020, compared to more than 1.8 million new adult cases overall, according to the American Cancer Society. It can also be assumed that families of children with cancer tend to be younger, with less wealth accumulated and less ability to direct earnings into a foundation or other vehicle to support cancer research. "I think [my grandparents] saw in that a real opportunity to make a difference where there was not a perceived gap, but a real gap in funding relative to the need," says Gilbert.

That need was to find better treatments. In Munn's case, unlike chemotherapy, which must be quite different for adults than for children due to potential toxicities on growing bodies, the immune system works similarly in both adults and children. "So, the case we made to Mr. and Mrs. Corn was to put some funding toward the pediatric aspect and earmark it specifically just for developing pediatric approaches; then we could use the research we already were doing in adult cancers," says Munn.

That research was for the immunotherapy drug indoximod, an enzyme and natural immune suppressor that Munn would develop to inhibit a specific pathway called IDO (indoleamine 2,3-dioxygenase). Munn believed that using the drug in combination with chemotherapy and radiation would enable the immune system to better recognize tumors and aid the body in fighting the cancer and slowing tumor growth. The Corns' gift was the turning point that allowed him and his team to, at last, laser their focus onto pediatric cancer.

Betty and Lovick also laid the foundation that has helped attract a broader base of donors for pediatric cancer research at MCG, including from Alex's Lemonade Stand Foundation for Childhood Cancer and the Press On Fund, among others. After Lovick passed away in 2013, Betty also continued to personally support Munn's work. Two years later, she saw the seed they planted together bloom: MCG's Dr. Ted Johnson, MD/PhD, '04, '07, a pediatric hematologist/oncologist, opened the first clinical trial of indoximod for children with relapsed brain cancer or a difficult-to-treat cancer called diffuse intrinsic pontine glioma or DIPG.

Gilbert still remembers the BELOCO Foundation meeting when the trial was announced. "We'd moved beyond thinking about something theoretically to something that actually might change and/or save lives," he says. "While I can't tell you what year that was, I remember it now.

It's always been one of those things where you want to use that story as the reason you do what you do, not the ones



Dr. David Munn

"Their legacy is one that will continue to live on by the very nature of what they chose to give to, not because they built a building, not because they put a plaque in the ground by a tree. They are literally helping people like Dr. Munn give life and hope to other people. I cannot imagine a better legacy to steward than that."

– Gilbert Miller

where it doesn't work out because there are way more of those ... But when you hit on something like that, that's where it becomes the magic for both your long-term commitment, as well as for these families."

And Munn keeps going back to this: The Corns' support is about much more than the clinical trial work.

"The Corns remain unique," he says, "in that they have always seen the value of the discovery research. That's not as common. There's such an urgent need to

try and fix the kids who have the cancer ... and the donors tend to want to support at the clinical trials stage. That is very logical ... But the problem is that if you only support at the clinical trials stage, then you don't make new discoveries that drive the next generation of clinical trials and make things better ... The Corns have always seen [that]."

Now, he says, MCG is planning to open additional clinical trials in children based on the discoveries funded by the Corns. Those trials, with an anticipated start

date of Summer 2022, will layer additional immunotherapy agents with indoximod to further activate the immune system and create a synergy that hopefully will further slow and shrink tumor growth. While Munn is unable to discuss the specifics of the two drugs, “this will be the first time this combination is tried anywhere, and it’s going to be in the pediatric population.”

And that is the power of the Corns’ ongoing giving, because what Munn is describing would have been unheard of even a decade ago: “It’s the first time ever — and it’s in kids.”

Legacy work

Lovick has been gone for more than seven years, and at 94, Betty is no longer as active as she once was. But Elizabeth remembers how thrilled they were to be a part of this important research “at the beginning”: “I can remember them saying very clearly that the reason they cared so much about pediatric treatment and research is you can treat an adult and buy them 5, 10, 15 years, but a child — if you treat and cure them, they have a lifetime. They thought that was a no-brainer.”

In March, Betty and Lovick’s family — the fourth and fifth generation, as they call themselves — gathered on Zoom to continue the work of the BELOCO Foundation and its founders, including their ongoing support of Munn’s pediatric cancer research. It’s exactly what Betty and Lovick would want.

“In many ways, I think my grandparents have left us a legacy of doing the work,” says Gilbert. “It’s almost leaving something ahead for us. We are now responsible for their legacy.”

“Their legacy is one that will continue to live on by the very nature of what they chose to give to, not because they built a building, not because they put a plaque in the ground by a tree. They are literally helping people like Dr. Munn give life and hope to other people. I cannot imagine a better legacy to steward than that.” 🌱



Gilbert Miller, wife Jamee, and children William, Benjamin and Juliette

MCG PEDIATRIC IMMUNOTHERAPY PROGRAM DONORS

- **BELOCO Foundation and Betty and Lovick Corn**
- **Alex’s Lemonade Stand Foundation**
- **Hyundai Hope on Wheels**
- **Cannonball Kids’ Cancer Foundation**
- **Press On Fund**
- **CAMFUND**
- **Northern Nevada Children’s Cancer Foundation**
- **Eli’s Block Party Childhood Cancer Foundation**
- **Gracie’s Hope**
- **Rally Foundation**
- **Miriam L. Halsey Foundation**
- **Other grateful patients and associated companies and foundations**

For more information on giving to MCG’s pediatric immunotherapy program, contact Eileen Brandon at **706-721-2515** or **ebrandon@augusta.edu** or visit **augusta.edu/giving/neversayno**

Dr. Kimford “Kim” Meador, ’76, a professor of neurology and neurosciences at Stanford University, and clinical director of the Stanford Comprehensive Epilepsy Center, had an article “Changes in Seizure Frequency and Antiepileptic Therapy during Pregnancy,” published in the Dec. 2020 issue of the *New England Journal of Medicine*.

Retired U.S. Navy Captain Dr. Frank K. Butler Jr., ’80, received the American College of Surgeons Distinguished Military Lifetime Achievement Award. Butler is only the second recipient of the Distinguished Military Lifetime Achievement Award.

Dr. Steven Kitchen, ’85, retired as CMO of Phoebe Putney Memorial Hospital in Albany, Georgia, which serves as the clinical hub for MCG’s Southwest Campus. **Dr. Kathy Hudson, ’98**, is Phoebe’s new CMO.

Dr. Sharon Beall, ’90, has joined MCG and the Children’s Hospital of Georgia as medical director of the pediatric palliative care and hospice program.

Dr. Nestor Edward Dourron, ’90, is leading Inception Fertility’s new network of clinics, Pathways Fertility in Atlanta.

Dr. William Hancock, ’92, is president and chief of staff with Tiftarea Cardiology in Tifton, Georgia.

Dr. Jay Goldstein, ’95, had his career highlighted in the *Savannah Morning News* regarding the impact of the COVID-19 pandemic.

Dr. Tracy Lovell, ’00, was named a 2020 Top Doctor in Rheumatology by *Atlanta Magazine* for the fifth year in a row.

Dr. Daniel Karolyi, ’04, was named chair of the Department of Radiology at the Virginia Tech Carilion School of Medicine and Carilion Clinic.

Dr. Destin Hill, ’07, joined the Emory Orthopaedics and Sports Medicine team as a team physician for the Atlanta Falcons and will work primarily out of the Flowery Branch, Georgia, clinic connected to the Falcons training facility.

Dr. Dustin Calhoun, ’08, is medical director of Emergency Management for UC Health in Cincinnati.

Dr. Ami DeWaters, ’12, assistant professor of internal medicine and assistant director of Health Systems Sciences Education at Penn State University, was featured in the September “Exceptional Moments in Teaching” program, which recognizes exceptional faculty, residents and fellows.

Dr. Krista Lim-Hing, ’13, has been appointed director of the Linda and John Bohlsen Neurosciences Intensive Care Unit at South Shore University Hospital in Bay Shore, New York.

Dr. Maharsh Patel, ’14, has joined OrthoGeorgia and is now practicing full-time in the Macon office.

Dr. Sung Lee, ’15, has joined Northeast Georgia Medical Center in Gainesville, Georgia, and will perform critical stroke intervention procedures in NGMC Gainesville’s new Neurointerventional Lab.

Dr. Jared Dowdy, ’16, has joined Sterling Center Women’s Health in Moultrie, Georgia.

Dr. Rebecca-Lyn Sokolove, ’16, a pediatrician with Yakima Neighborhood Health Services in Yakima, Washington, has been named a Person to Watch by the *Yakima Herald*. 🍀

Dr. James Wynford Pate, the last surviving member of the MCG Class of 1950, died January 31.

After graduating from MCG, Pate served during the Korean War and completed two general surgery residency programs, the first at the National Naval Medical Center in Bethesda, Maryland; and a second at the University of Alabama at Birmingham. He also completed a thoracic surgery residency at the Veterans Administration Teaching Group in Memphis, Tennessee.

During his Bethesda residency, Pate headed the Division of Experimental Surgery, where he helped conceive and develop the freeze-dried process for preserving arteries to use as grafts, proving the practice was practical and could be used on soldiers wounded in combat. He also was the co-discoverer of the bioelectrical causes of blood clots in arteries and veins.

Among his many accomplishments over his decades-long career he:

- Was a team leader for the first animal and human transplantation of long-term stored arteries and bone;
- Co-developed the world's first tissue bank in Bethesda, and was invited to present his findings at the first International Conference on Transplantation in London in 1953;
- Established the first tissue banks at the University of Alabama, the University of Tennessee and the Veteran's Administration;
- Established open-heart surgery programs at University of Tennessee's John Gaston Hospital, Baptist Memorial Hospital, and St. Joseph's East, among others;
- Co-developed the first intensive care unit with remote electronic monitoring of physiologic variables in 1960;
- Established the Heart Transplant Program at University of Tennessee (now UT-Baptist) in 1985; and
- Co-led establishment of Tennessee's first statewide Level 1 Trauma System, which included the Elvis Presley Regional Trauma Center and the Firefighters Regional Burn Center.



During the Vietnam War, Pate spent several months serving as a civilian surgeon teaching Vietnamese physicians modern operating techniques. During this time, he performed the first heart operation in Vietnam and was awarded the Republic of South Vietnam's "Contributions to Surgery Award."

In 1978, he was one of four surgeons invited to introduce pacemakers to the People's Republic of China. Eleven years later, Pate received a Certificate of Appreciation from President George H.W. Bush for "Service to United States Armed Forces" and was named Distinguished University Professor by the University of Tennessee. 🍀



Dr. Norman Pursley Sr., a 1948 MCG graduate and longtime superintendent of Gracewood State School and Hospital, died June 14.

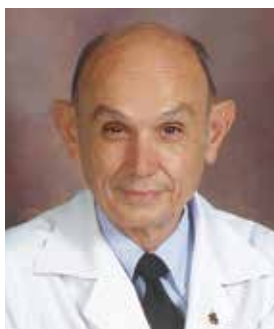
Pursley began his medical career in general practice in Hiawassee, Georgia, before accepting a position at Central State Hospital as senior physician in psychiatry in 1949. He began his career at Gracewood in 1951 and served as superintendent there

until 1985. Upon his arrival, Gracewood only provided minimal custodial care. By the mid-1960s, the facility had grown to approximately 1,900 residents with about 1,200 employees that required an increase in the annual budget from \$7 to \$10 million. By the 1970s, a full range

of programs were offered in support of not only the care, but the training of the residents.

He “opened up” Gracewood, inviting visitors especially parents and parent groups. This helped parents see that there were more possibilities for their children and form the Georgia Association for the Mentally Retarded. The Gracewood training program for medical students educated hundreds of physicians and prepared them to meet the medical needs of the intellectually disabled.

Pursley served on the first President’s Committee for People with Intellectual Disabilities and was an early proponent of community services that targeted the needs of the individual. He was a consultant and advisor on a national effort that ultimately established the Special Olympics and was appointed by President John F. Kennedy to help develop a national plan to combat mental retardation. He also served as president of the Georgia Public Health Association and the National Association of Superintendents of Public Residential Facilities for the Mentally Retarded. 🌱



Dr. Joseph Bailey Jr., a 1955 MCG graduate, rheumatologist and longtime faculty member, died June 19.

After graduating from MCG, the Augusta native completed an internship at the Medical College of Virginia before returning to his alma mater to finish his residency and fellowship.

From 1957-59 Bailey was an active duty captain in the United States Army Medical Corps, assigned as the post

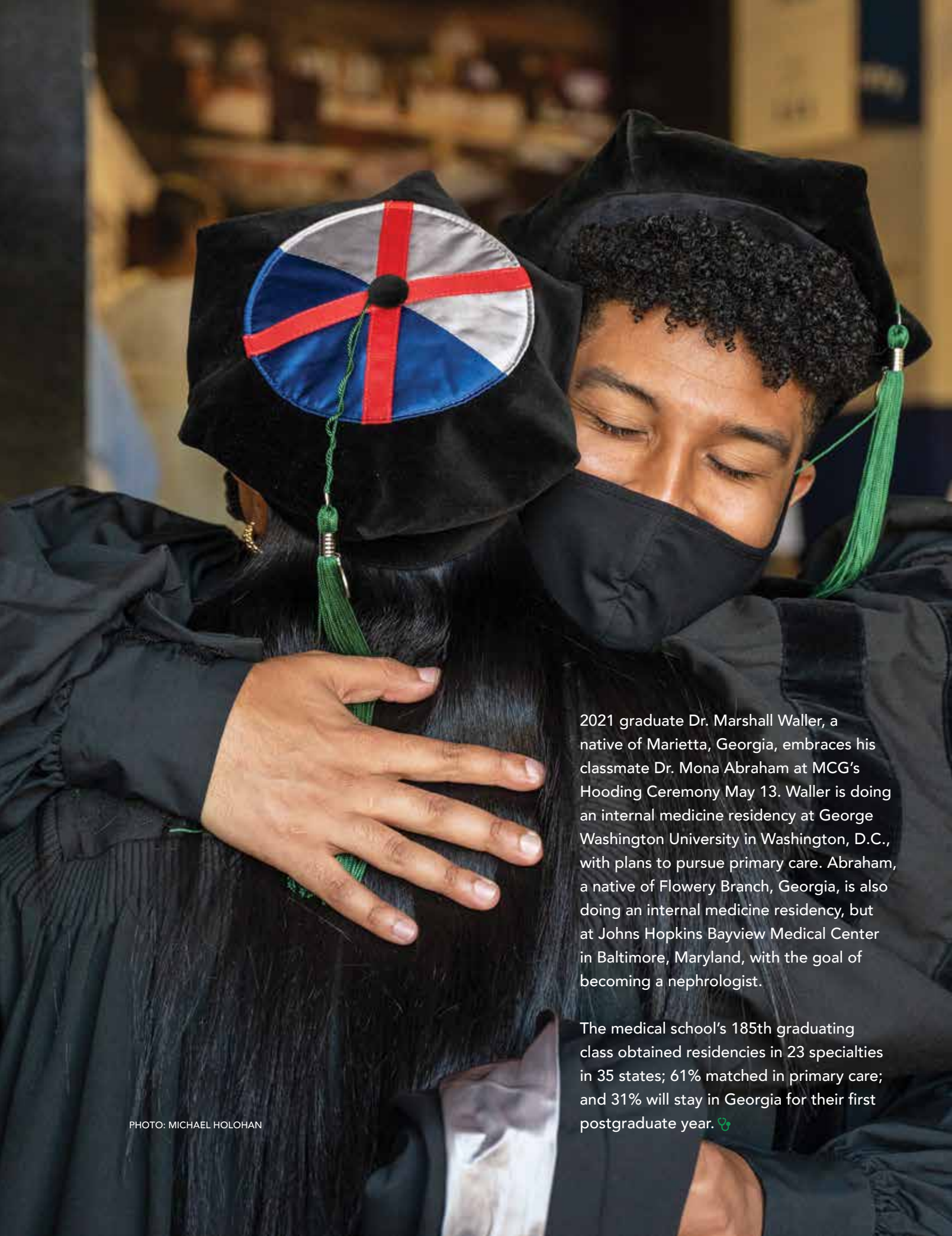
surgeon to Fort Buchanan in San Juan, Puerto Rico.

He returned to his medical school’s faculty in 1961 and in 1967 established the Section of Rheumatology, and became its first chief, a position he held until his retirement from MCG in 2000. He was named associate dean for clinical sciences in 1972 and Charbonnier Professor of Medicine in 1984, and later held emeritus titles honoring both those posts.

Bailey worked with numerous professional groups, including serving as president of the Richmond County Medical Society, the Georgia Society of Rheumatology and the Medical Association of Georgia. He also served on the Georgia delegation for the American Medical Association from 1989-2014.

Over the years, he was honored for his work, both in and out of medicine. In 1990 he received the Distinguished Service Award from the Medical Association of Georgia, an award the association later renamed in his honor. In 2014, the Medical Association of Georgia awarded him their Legacy Award and he received the Lifetime Distinguished Service award from the American Medical Association in 2015. 🌱

- Dr. David B. Byck*, '86, Nov. 21
- Dr. William J. Hardman*, '59, Dec. 30
- Dr. Bill M. Bailey*, '55, Jan. 1
- Dr. William D. Tumlin*, '67, Jan. 5
- Dr. Thomas S. Branigan*, '60, Jan. 14
- Dr. Alva H. Faulkner*, '45, Jan. 18
- Dr. Joseph A. Snitzer*, '63, Jan. 18
- Dr. Tammy J. Robinson*, '90, Jan. 19
- Dr. William Sidney Smith*, '64, Jan. 28
- Dr. Alva L. Mayes*, '56, Feb. 3
- Dr. Donna M. Wall*, '80, Feb. 3
- Dr. Elaine Hajosy*, '60, Feb. 17
- Dr. John A. Crowe*, '67, Feb. 18
- Dr. William H. Kilpatrick*, '59, March 4
- Dr. Thomas C. Johnson*, '91, March 6
- Dr. Thomas H. Lowe*, '71, March 6
- Dr. McCoy L. Moretz*, '82, March 10
- Dr. Roy G. Duncan*, '53, March 13
- Dr. Ralph A. Tillman*, '57, March 16
- Dr. Charles R. Hatcher*, '54, March 27
- Dr. Dennis H. Jones*, '78, April 1
- Dr. James L. Bland*, '60, April 13
- Dr. Lowell J. Kepp*, '57, May 7
- Dr. Paul G. McDonough*, '66, May 8
- Dr. Robert S. Thornton*, '68, May 13
- Dr. Bowen Asserson Jr.*, '70, May 25
- Dr. Chris Smith*, '78, May 29
- Dr. Danielle McFarland-Neloms*, '02, May 30
- Dr. Murray Arkin*, '53, June 6
- Dr. William Ogden*, '65, June 15 🌱



2021 graduate Dr. Marshall Waller, a native of Marietta, Georgia, embraces his classmate Dr. Mona Abraham at MCG's Hooding Ceremony May 13. Waller is doing an internal medicine residency at George Washington University in Washington, D.C., with plans to pursue primary care. Abraham, a native of Flowery Branch, Georgia, is also doing an internal medicine residency, but at Johns Hopkins Bayview Medical Center in Baltimore, Maryland, with the goal of becoming a nephrologist.

The medical school's 185th graduating class obtained residencies in 23 specialties in 35 states; 61% matched in primary care; and 31% will stay in Georgia for their first postgraduate year. 🌱



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neuron noun

neu·ron | \ nü-rän , nyü-; nür-än, nyür-\

a grayish or reddish granular cell that is the fundamental functional unit of nervous tissue transmitting and receiving nerve impulses and having cytoplasmic processes which are highly differentiated frequently as multiple dendrites or usually as solitary axons which conduct impulses to and away from the cell body.

Merriam Webster

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