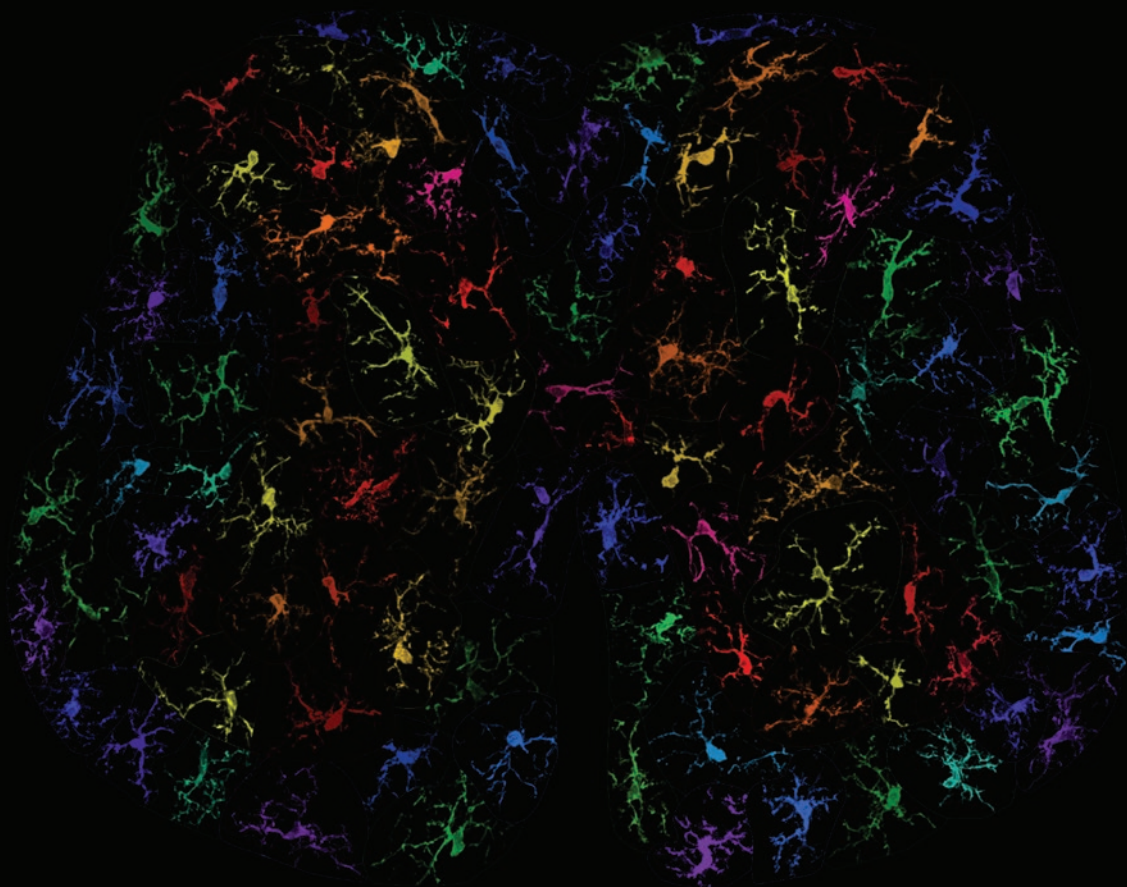


The Rita Allen Foundation

# AWARD IN PAIN SCHOLARS: ADVANCING BASIC PAIN RESEARCH



COVER: Tuan Trang, a 2014 Rita Allen Foundation Scholar, has investigated mechanisms of opioid tolerance and withdrawal. Trang and his research group have found that immune cells in the central nervous system, known as microglia, play a key role in the development of morphine tolerance in an animal model. This image, a compilation of spinal microglia forming a cross section of the lumbar spinal cord, appeared on the cover of the October 18, 2017, issue of *The Journal of Neuroscience* in conjunction with the research article "Site-Specific Regulation of P2X7 Receptor Function in Microglia Gates Morphine Analgesic Tolerance." (Image by Heather Leduc-Pessah, Trang Laboratory)

# A NETWORK OF HOPE



Elizabeth G. Christopherson  
*President and  
Chief Executive Officer  
Rita Allen Foundation*

Created in 2009 to expand the reach of the Rita Allen Foundation Scholars program, the Award in Pain has now supported 29 pioneering early-career Pain Scholars. Each year, as we welcome our newest class of Scholars, we reflect on the accomplishments of this growing community of researchers and the profound questions that drive them forward to new discoveries.

These scientists are leading efforts to understand the complex neurobiological mechanisms that underlie pain—including mapping the neural circuits of chronic pain, defining the roles of immune signals, and examining the connections between pain and itch. Their findings point to approaches for combating opioid tolerance and withdrawal, interventions to interrupt the transition from acute to chronic pain after injury, and targets for completely novel pain therapies with the

potential to improve safety and specificity.

They are part of teams creating and combining new tools and knowledge, brought together by a deep commitment to seeing this field into the future.

Fueled by that commitment, we also are working to rally more support for a future where pain is better understood and managed. Pain research was identified by our original benefactor, Rita Allen, as among a handful of priority areas requiring support to relieve human suffering. Her vision, combined with insight from pioneers in public health and biomedical research, led to the Rita Allen Foundation Scholars program—which included pain research in its areas of focus when it began supporting high-promise, innovative research 45 years ago.

Since her first days as a Rita Allen Foundation Scholar, Kathleen Foley, has advanced pain research and treatment globally and now supports new generations of Scholars as the Foundation's Medical Adviser. Together, we are developing partnerships to support research and collaboration to confront the scope of pain in America—including identifying and supporting early-career scientists opening exciting new horizons of research with transformative potential.

In addition to celebrating the efforts of the Rita Allen Foundation Award in Pain Scholars, we express gratitude to the scientists—many of whom also are Rita Allen Scholars—who have advised our work and served on selection committees over the years.

The message we are sharing about pain research is one of great urgency to meet a growing global need as well as promise. The scientists on the pages that follow represent a vibrant,



Dr. Kathleen Foley  
*Rita Allen Foundation  
Medical Adviser*

growing community of pain researchers. Together—whether by way of neuroscience, genetics, cell biology, or clinical practice—pain scientists represent, in the face of great challenges, a network of hope.

Their work grows more urgent as we face an aging population and widespread experiences of pain for cancer survivors and others. Chronic pain affects more than 20 percent of adults in the United States, and 20 million Americans experience high-impact pain that significantly impairs their life or work, at the cost of hundreds of billions of dollars and suffering that is impossible to quantify. One of our society's challenging epidemics is drug overdose deaths—with nearly as many Americans now dying from opioid overdoses as from car accidents and gun homicides combined. Investing in the research necessary to lay the groundwork for safer, more effective pain treatments is a vital priority as part of a longer-term solution.

In addition to the widespread experiences of pain and challenges with drug abuse and overdose, the current coronavirus pandemic poses new concern that many COVID-19 patients have experiences with pain. Rita Allen Pain Scholars and others in the field have started to explore the connection between pain and COVID-19—exploring questions on the connections between coronavirus infection, inflammation, and neurons—to develop a deeper knowledge of the biology of this new disease as well as targets for therapeutics. Breakthroughs from this new intersection of study could even have applications for developing more effective pain treatments and understanding other widespread experiences of pain.

The field of pain research is full of seeds of innovation to advance medicine and ultimately change the lives of individuals affected by a widespread and complicated disease. Investing in biomedical research, especially pain research, brings hope for a new future for patients and families living through this silent epidemic.

With profound appreciation for the outstanding scientists who are dedicated to making essential discoveries,

A handwritten signature in black ink, reading "Elizabeth G. Christopherson". The script is fluid and cursive, with the first letters of the first and last names being capitalized and prominent.

Elizabeth G. Christopherson  
President and Chief Executive Officer  
Rita Allen Foundation



## STEVE PRESCOTT, M.D., PH.D.

Senior Scientist, Neurosciences and Mental Health  
The Hospital for Sick Children, Toronto  
Professor, Physiology and Biomedical Engineering  
University of Toronto

*Project: Pain Processing by Neural Networks - A Critical Link between Molecular and Perceptual Changes Associated with Neuropathic Pain*

Steve Prescott obtained his M.D. and Ph.D. degrees from McGill University and did postdoctoral training in computational neuroscience at the Salk Institute for Biological Studies. In 2008, he set up his own lab at the University of Pittsburgh, combining experiments and simulations to investigate pain processing. In 2012 his lab moved to The Hospital for Sick Children in Toronto. Prescott was named a Mallinckrodt Scholar and has also received a Canadian Institutes of Health Research New Investigator Award and an Ontario Early Researcher Award. He has received funding from the NIH, the Canadian Institutes of Health Research, the Natural Sciences and Engineering Research Council of Canada, and the Canadian Foundation for Innovation. In 2019 he secured a prestigious CIHR Foundation Grant, which provides operating funds for seven years.

Prescott's lab synergistically combines computational methods with diverse experimental techniques including electrophysiology, optogenetics, and calcium imaging to uncover how pathological changes in neuronal excitability and synaptic transmission disrupt pain processing. His team has demonstrated how the same pathological change in neuronal excitability can arise through distinct molecular mechanisms; this so-called degeneracy has important implications for choosing effective drug targets and may help explain why neuropathic pain is so difficult to treat. His team studies several additional topics, from homeostatic regulation of excitability to spinal cord stimulation, and the basis for and consequences of synchronized spiking.



## THEODORE PRICE, PH.D.

Eugene McDermott Professor, School of Behavioral and Brain Science  
Director, Center for Advanced Pain Studies  
The University of Texas at Dallas

*Project: Translation Regulation as a Novel Paradigm for Understanding Nociceptor Sensitization and Developing Analgesic Targets*

Theodore Price completed his Ph.D. at the University of Texas Health Science Center in San Antonio and a postdoctoral fellowship at McGill University. He took his first faculty position at the University of Arizona in 2007 and moved to UT Dallas and the Center for Advanced Pain Studies in 2014. Funded by multiple National Institutes of Health research grants and contracts, Price's laboratory focuses on molecular mechanisms involved in the development of chronic pain, with the goal of creating the first therapeutics that can reverse chronic pain states in humans. He has received early-career awards from the American Pain Society and the International Association for the Study of Pain. Price is a member of the Neurobiology of Pain and Itch Study Section of the NIH and serves on the editorial boards of numerous journals, including *PAIN*, where he is the neurobiology section editor. His group has published more than 150 peer-reviewed articles in leading journals. He also has an entrepreneurial focus and has started several companies developing pain therapies and biomarkers—these include CerSci Therapeutics, 4E Therapeutics, and Doloromics.

Price's work has focused on understanding the phenotypic changes that occur after injury and cause pain-sensing neurons to become hyperactive, driving chronic pain. His work has focused on how translation control signaling governs these phenotypic changes (mRNAs are translated in an activity-dependent fashion in these neurons) and how targeting kinases that regulate translation of mRNAs can lead to disease modification resulting in long-lasting relief of chronic pain. His lab uses preclinical models—often working with human neurons from organ donors, or surgical patients—with a focus on generating molecular profiles of single nociceptors, including nociceptors from chronic pain patients. Insights from these studies can dramatically improve our ability to generate next-generation pain medicines.





## SEENA AJIT, PH.D.

Associate Professor, Department of Pharmacology and Physiology  
Drexel University College of Medicine

*Project: MicroRNA Regulation and Its Utility for Biomarkers in Neuropathic Pain*

Seena Ajit received her Ph.D. in molecular biology from Rutgers University, and was a scientist in Neuroscience Discovery at Wyeth Research from 2001 to 2009, when she became an assistant professor at Drexel University College of Medicine.

Epigenetics is now recognized as an important aspect of gene regulation, and epigenetic analysis is likely to play an increasingly important role in the diagnosis, prognosis and treatment of diseases. Ajit is interested in pursuing various aspects of epigenetics, including DNA methylation, histone modifications and RNA-mediated gene silencing, all aimed at understanding the molecular mechanisms underlying pain. Currently her group is studying the role of noncoding RNAs in mediating pain, and their utility for biomarkers. They have identified differentially expressed circulating miRNAs in whole blood from patients with complex regional pain syndrome, relative to controls. Additionally, studies using blood samples from good and poor responders to ketamine treatment have identified differential miRNA signatures both before and after therapy. A major focus of the Ajit lab now is to understand the significance of miRNA alterations in regulating gene expression leading to inflammation and pain.

Circulating miRNAs in bodily fluids are delivered to recipient cells via exosomes. Intercellular communication once thought to be limited to secreted signals can now be attributed to exosomes. Exosome contents, including miRNAs and mRNAs, are capable of modulating gene expression in the recipient cells. Ajit's lab is investigating the mechanistic basis of pro- and anti-nociceptive roles of exosomes from various sources, including patients and rodent models of pain.



## DIANA BAUTISTA, PH.D.

Professor, Cell and Developmental Biology and Neurobiology  
Department of Molecular and Cell Biology, Helen Wills Neuroscience Institute  
University of California, Berkeley

*Project: Molecular Mechanisms of Somatosensory Mechanotransduction*

Diana Bautista earned her B.S. in biology from the University of Oregon and her Ph.D. in neuroscience from Stanford University, where she worked in the laboratory of Richard Lewis. She pursued a postdoctoral fellowship at the University of California, San Francisco, with David Julius. Bautista joined the Berkeley faculty in 2008. In addition to a Rita Allen Foundation Scholar award, she received the 2014 Society for Neuroscience Young Investigator Award, a Howard Hughes Medical Institute Faculty Scholar Award, a 2019 NIH Director's Transformative Research Award, and a 2021 Weill Neurohub Research Award.

Bautista's lab aims to understand the molecular mechanisms underlying the sensations of itch, touch, and pain. Humans rely on these senses for a broad range of essential behaviors. For example, acute pain acts as a warning signal that alerts us to noxious mechanical, chemical and thermal stimuli, which are potentially tissue damaging. Likewise, itch sensations trigger reflexes that may protect us from disease-carrying insects. Despite these essential protective functions, itch and pain can outlast their usefulness and become chronic. Bautista's lab uses cellular physiology, molecular biology, molecular genetics, and behavioral studies to elucidate the molecular mechanisms underlying itch and pain transduction under normal and pathophysiological conditions. The Bautista lab's recent work has focused on the neuroimmune interactions that drive chronic pain and itch. Her findings not only highlight the importance of the nervous system in driving inflammation and directing the immune response, but have also revealed the numerous ways peripheral immune cells can affect neuronal activity, innervation and plasticity in both the peripheral and central nervous system.



## E. ALFONSO ROMERO-SANDOVAL, M.D., PH.D.

Associate Professor, Anesthesiology  
Wake Forest School of Medicine

*Project: Spinal Cord Mechanisms in the Resolution of Postoperative Pain*

Alfonso Romero-Sandoval is originally from Guatemala, and received his M.D. degree from Universidad de San Carlos de Guatemala in 1999 and his Ph.D. degree in neuroscience from Universidad de Alcalá de Henares in Spain, in 2003. He did postdoctoral training at Wake Forest University and at Geisel Dartmouth Medical School. He has served on the faculty at Geisel Dartmouth Medical School and Presbyterian College School of Pharmacy. Romero-Sandoval is currently an associate professor of anesthesiology at Wake Forest School of Medicine and is studying the molecular mechanisms of macrophages and their role in wound healing in the transition from acute to chronic pain, and the use of nanotechnology to promote surgical wound healing and prevent the development of chronic postoperative pain. His other projects include the role of cannabinoid receptor activation in skin cells for induction of analgesia, the role of endocannabinoids in postoperative pain, and the function of phosphatases and kinases in the spinal cord and periphery in the context of pain.

Romero-Sandoval's lab uses a clinically relevant cell target gene therapy method applying nanotechnology (successfully used in HIV-positive patients for gene induction) in human macrophages. The final goal of this project is to target macrophages to induce a cellular phenotype that drives the efficient resolution of inflammation and prevents the development of chronic pain following major surgeries. His team uses nanoparticles to specifically target macrophages. Romero-Sandoval proposes to induce specific genes that are involved in the resolution of inflammation by driving an M2 macrophage phenotype.



## YUAN-XIANG TAO, M.D., PH.D.

Professor and Vice Chair of Research, Department of Anesthesiology  
Rutgers University, New Jersey Medical School

*Project: Discovery of a Large Non-Coding RNA and Its Regulation by Peripheral Nerve Injury*

Yuan-Xiang Tao earned B.Sc., M.Sc. and M.D. degrees from Nanjing Medical University, and holds a Ph.D. in neuroscience from the Shanghai Brain Research Institute of the Chinese Academy of Sciences. He completed a postdoctoral fellowship at the University of Virginia Health Sciences Center, where he studied the role of postsynaptic density proteins in chronic pain and anesthesia. Tao was a faculty member in the Department of Anesthesiology and Critical Care Medicine at Johns Hopkins University School of Medicine from 1999 to 2013, when he became a full professor with tenure at Rutgers New Jersey Medical School and Vice Chair of Research in the Department of Anesthesiology. Tao has received funding from the National Institutes of Health, the Blaustein Pain Research Fund, the David H. Koch Prostate Cancer Research Fund, and the Johns Hopkins Medicine Brain Science Institute. He was selected for the Rutgers New Jersey Medical School's Faculty of the Year Award, as well as an Excellence in Research Award from the New Jersey Health Foundation in 2017, and the Rutgers Board of Trustees Award for Excellence in Research in 2020. Tao is the Co-Editor-in-Chief of Translational Perioperative and Pain Medicine. He has published more than 155 scientific articles and invited reviews, and has been featured in top-rated scientific journals, such as *Nature Neuroscience*, *Advanced Science*, *Neuron*, *The Journal of Clinical Investigation*, *Nature Communications*, and *Cell Research*. Tao also currently holds five patents.

Tao's research focuses on understanding the molecular and cellular mechanisms that underlie chronic pain and opioid-associated disorders. Research projects in his laboratory have attempted to address questions regarding different aspects of chronic pain and opioid analgesia: how protein translation is regulated under the conditions of chronic opioid tolerance and hyperalgesia; how epigenetic and epitranscriptomic modifications participate in the development and maintenance of chronic pain; and how acute vaso-occlusive episodes induce and aggravate sickle cell disease-associated pain.



## MICHAEL JANKOWSKI, PH.D.

Theodore W. Striker, MD Chair in Anesthesia Research  
Director of Research and Associate Professor, Department of Anesthesia  
Cincinnati Children's Hospital Medical Center, Department of Pediatrics  
University of Cincinnati College of Medicine

*Project: Molecular Mechanisms of Musculoskeletal Pain after Ischemic Tissue Injury*

Michael Jankowski earned an M.S. in neuroscience and a Ph.D. in neurobiology from the University of Pittsburgh, where he also conducted postdoctoral research. He joined the faculty of the Cincinnati Children's Hospital Medical Center in 2011. Dr. Jankowski has received several Department of Defense and National Institutes of Health grants in addition to industry contracts. He also is a recipient of an International Association for the Study of Pain Early Career Research Grant Award, and was named a Cincinnati Children's Trustee Scholar in addition to being a Rita Allen Award in Pain recipient. He serves on numerous national and international grant review panels and journal review boards, and is an associate editor for the journal *PAIN* and reviewing editor for *Molecular Pain*. He is the current Associate Director of Admissions and Recruitment for the Medical Scientist Training Program and Associate Director of Basic Science Research for the Center for Understanding Pediatric Pain.

Dr. Jankowski's lab is interested in the peripheral mechanisms of pain development under unique injury conditions. Pain is a significant health issue that affects a large number of people worldwide. The mechanisms by which pain develops in children may be distinct from adults and influenced by non-neuronal communication. While we know a great deal of information about the role of nociceptors in the development of pain states, we do not have a comprehensive understanding of how distinct subtypes of sensory fibers modulate pain across the lifespan under different injury conditions.

Some of his lab's discoveries include finding that peripheral growth hormone signaling to neurons not only modulates normal sensory development, but exogenous GH may also be used as a potential therapy for pediatric pain. The lab also found that distinct growth factor signaling pathways can modulate both muscle pain and cardiovascular reflexes after ischemic injury to the periphery. These studies will hopefully lead to the development of treatments for adverse changes in cardiovascular reflexes or musculoskeletal pain and generate novel information on the development of both nociceptive and non-nociceptive afferents in relation to how they communicate with non-neuronal cells.



## SARAH ROSS, PH.D.

Associate Professor, Department of Neurobiology  
Pittsburgh Center for Pain Research  
University of Pittsburgh

*Project: Investigating the Neural Circuits of Itch and Pain*

Sarah Ross earned her B.Sc. from the University of Western Ontario and her Ph.D. from the University of Michigan, both in physiology. She was a postdoctoral fellow in the Department of Neurobiology at Harvard Medical School, where she worked with Michael Greenberg. She joined the faculty at the University of Pittsburgh in 2011. In addition to the Rita Allen Foundation, she has received support for her work from the National Institutes of Health.

The spinal cord plays a critical role in processing somatosensory information. To study these spinal microcircuits, the Ross lab has developed a novel somatosensory preparation that allows recording from the output neurons (via retrograde labeling of spinal projection neurons) while controlling somatosensory input (via natural stimulation of the skin) and simultaneously manipulating activity of specific populations of spinal interneurons (via the combination of Cre alleles and optogenetics). Using this physiological preparation, the Ross lab is addressing long-standing questions in the field of somatosensation, such as: how is itch distinguished from pain; why does scratching relieve itch; and how does pain become abnormally amplified upon injury.





## REBECCA SEAL, PH.D.

Associate Professor, Neurobiology  
Pittsburgh Center for Pain Research  
University of Pittsburgh

*Project: Mechanical Pain Circuits in the Dorsal Horn*

Rebecca Seal earned her B.S. in chemistry and psychology from the University of Oregon and her Ph.D. in neuroscience from Oregon Health and Science University. Her graduate studies with Susan Amara focused on the structure and function of plasma membrane glutamate transporters. As a postdoctoral scholar at the University of California, San Francisco, she studied the vesicular glutamate transporter 3 in hearing and pain with Robert Edwards. She has received a NARSAD Young Investigator Grant from the Brain and Behavior Research Foundation, an Innovation Award from the American Diabetes Association, a Japanese Society for the Promotion of Science Fellowship, and a Whitehall Foundation Research Award.

Seal's laboratory focuses on defining the neural circuitry underlying a wide range of nervous system functions in health and disease, including persistent pain and motor symptoms of Parkinson's disease and audition. A major impediment to identifying new pain treatments is incomplete understanding of the neural networks and mechanisms that underlie pain. Her team's work, using multiple approaches ranging from cellular and molecular to physiological and behavioral, centers on elucidating the neural circuits and mechanisms that underlie a particular form of persistent pain in which touch becomes painful in the setting of injury, termed mechanical allodynia. The spinal cord dorsal horn is a major site for the integration of somatosensory information and is vital for the induction and maintenance of this form of pain. Their work thus far suggests that the pain is encoded by distinct microcircuits in the dorsal horn, depending on the nature of the injury. This concept not only has important implications for understanding at a basic level how the nervous system encodes mechanical allodynia, but also highlights the need to consider etiology in the design and implementation of therapeutic strategies.



## REZA SHARIF-NAEINI, PH.D.

Associate Professor  
Department of Physiology and Cell Information Systems Group  
Director, Alan Edwards Centre for Research on Pain (McGill Pain Center)  
McGill University

*Project: Role of Parvalbumin Neurons in Dorsal Horn Pain Circuits*

Reza Sharif-Naeini earned his Ph.D. in physiology from McGill University in 2007, and returned there to join the faculty in 2012. In the interim, he was a postdoctoral fellow at Institut Pharmacologie Moléculaire et Cellulaire in Nice, France, and in the Department of Anatomy at the University of California, San Francisco. He has received fellowships from the Canadian Institutes of Health Research, the International Association for the Study of Pain, and the Human Frontier Science Program. He has also received the Peter and Patricia Gruber International Research Award in Neuroscience from the Society for Neuroscience, and in 2021 the Canadian Pain Society Early Career Award. Since starting his independent position, Dr. Sharif has published over 20 articles in top tier journals including *Cell*, *Neuron*, *Nature Communications*, and *Cell Reports*. His research is supported by grants from the Fonds de Recherche du Québec, the Natural Sciences and Engineering Research Council of Canada, the Canadian Institutes of Health Research, as well as several philanthropic foundations.

Dr. Sharif currently leads a pain research laboratory focusing on identifying novel pain sensors, discovering the mechanisms of chronic pain, and translating these lab discoveries into effective therapies for patients. He is the new Director of the Alan Edwards Center for Research on Pain (also known as The McGill Pain Center), Director of the Strategic Initiative on Neuronal Mapping of the Quebec Pain Research Network, a member of the Pain Scholars Leadership Committee of the Rita Allen Foundation, and Associate Editor for the journal *PAIN*. Recently, he co-founded PteroTech, a Canadian company that developed an ointment that blocks the pain caused by lionfish and jellyfish stings.



## GREGORY SCHERRER, PH.D.

Associate Professor, Department of Cell Biology and Physiology  
Department of Pharmacology  
University of North Carolina at Chapel Hill

**Project:** *Molecular Mechanisms of Opioid-induced Analgesia, Tolerance and Hyperalgesia*

Gregory Scherrer earned his Ph.D. in cellular and molecular biology from the University of Strasbourg, France. He completed postdoctoral training at the University of California, San Francisco, and at Columbia University. He joined the faculty at the Stanford University School of Medicine in 2012. In addition to a Rita Allen Foundation Award in Pain, he has received an International Association for the Study of Pain Postdoctoral Fellowship, National Institutes of Health/National Institute on Drug Abuse K99/R00 Pathway to Independence and R01 Awards, a Department of Defense Neurosensory Research Award, and an International Narcotics Research Conference Young Investigator Award, and most recently was named a New York Stem Cell Foundation – Robertson Neuroscience Investigator.

The members of the Scherrer laboratory investigate how the nervous system generates the sensory and affective dimensions of pain experience and opioid analgesia to discover novel analgesic therapies. They aim to identify the pathological changes that occur within neural circuits when chronic pain develops, at the neural network, cellular and molecular levels. One of their approaches is to gain understanding of how our endogenous opioid system modulates pain thresholds. Opioid receptors mediate the effects of opioid painkillers, such as morphine. By determining how opioids generate analgesia and detrimental side effects (e.g., tolerance, addiction, respiratory depression), Scherrer and his team hope to develop more efficient and safer analgesics for the treatment of chronic pain. These studies will also identify novel approaches to counteract opioid side effects and battle the current opioid epidemic. To reach these goals, Scherrer's research combines a variety of experimental approaches, including molecular and cellular biology, neuroanatomy, electrophysiology, opto- and pharmacogenetics, *in vivo* calcium imaging and behavior.



## TUAN TRANG, PH.D.

Associate Professor, Comparative Biology and Experimental Medicine,  
Physiology and Pharmacology  
University of Calgary

**Project:** *Alleviating Opioid Withdrawal by Blocking Pannexin-1 Channels*

Tuan Trang completed a Ph.D. in pharmacology and toxicology at Queen's University, and pursued postdoctoral training as a Canadian Institutes of Health Research (CIHR) Fellow in the laboratory of Dr. Michael Salter at the Hospital for Sick Children in Toronto. He has received a CIHR New Investigator Award, as well as young investigator awards from the Canadian Association for Neuroscience and Canadian Society for Pharmacology and Therapeutics. His research has been supported by grants from the Rita Allen Foundation, the CIHR, the Natural Sciences and Engineering Research Council of Canada, the University of Calgary, and the Canada Foundation for Innovation.

Opioids are among the most powerful and widely prescribed drugs for treating pain. However, a major problem in terminating opioid pain therapy is the debilitating withdrawal syndrome that can plague chronic opioid users. The mechanisms involved in opioid withdrawal are poorly understood, and the limited clinical strategies for treating withdrawal are ineffective. Trang and his collaborators have identified the pannexin-1 channel as a novel therapeutic target for treating morphine withdrawal. They discovered that morphine treatment induces synaptic plasticity in spinal lamina I/II neurons, which manifests as long-term synaptic facilitation upon naloxone-precipitated morphine withdrawal. This synaptic facilitation is critically gated by activation of Panx1 channels expressed on microglia. Pharmacologically blocking Panx1, or genetically ablating this channel specifically from microglia, blocked spinal synaptic facilitation and alleviated the behavioral sequelae of morphine withdrawal. Trang and his team are now moving these discoveries into the clinic.



## ROBERT SORGE, PH.D.

Associate Professor, Department of Psychology  
The University of Alabama at Birmingham

*Project: Immune System Modulation of Pain via Diet*

Robert Sorge earned his Honors B.Sc. in psychology from McMaster University in Hamilton, Ontario, his M.A. in experimental psychology from Wilfrid Laurier University in Waterloo, Ontario, and his Ph.D. in psychology from Concordia University in Montreal, Quebec. He was a postdoctoral fellow at McGill University before joining the faculty at The University of Alabama at Birmingham in 2012. In addition to being named a Rita Allen Foundation Pain Scholar, he has received a Young Investigator Award from the Sex, Gender and Pain Special Interest Group of the International Association for the Study of Pain. He also has received postdoctoral fellowships from the Alan Edwards Center for Research on Pain at McGill and from the National Sciences and Engineering Research Council of Canada.

Obesity in America is reaching epidemic proportions, with more than one-third of the population classified as obese, and even more as overweight. In addition to the increased risk for metabolic syndromes and cardiovascular disease, obesity is comorbid with chronic pain for a significant number of patients. It is known that adipose tissue and components of the American diet can contribute to a chronic proinflammatory state that may predispose individuals to significant negative health effects. Sorge and his collaborators believe that this state is the result of heightened activity of the immune system. Their previous work has shown that consumption of a Western diet results in changes in acute sensitivity to stimuli, increased systemic inflammation and prolonged recovery from injury. These effects are believed to be the result of chronic immune cell activation in the peripheral and central nervous system. Current work is underway to investigate the temporal profile of immune cell activation following differential exposure to the American diet in rodents. Through examination of the immune-related impact of diet, it may be possible to formulate treatments that will reduce the negative effects of the American diet with respect to pain and other related inflammatory conditions.



## YI YE, PH.D.

Assistant Professor  
Associate Director of Clinical Research Operations  
Bluestone Center for Clinical Research  
College of Dentistry, New York University

*Project: The Role of Schwann Cells in Neural Invasion and Associated Cancer Pain*

Yi Ye holds a Ph.D. in neuroscience from the University of Wyoming, a Master's degree in clinical research from New York University and an M.B.A. from NYU's Stern School of Business. She was a research fellow in the Department of Oral and Maxillofacial Surgery in the College of Dentistry at the University of California, San Francisco, and joined the Bluestone Center as an associate research scientist in 2010. She has been in her current position since 2015. She has received a Travel Award and an Early Career Research Grant Award from the International Association for the Study of Pain, and was awarded both a National Institutes of Health-NYU-Clinical and Translational Science Institute Scholarship and an NYU Whitehead Fellowship in 2015.

Ye's research aims to understand the neurobiological basis of cancer pain, with an additional focus on carcinogenesis and tumor progression in head and neck cancer. The ultimate goal of her research is to develop novel therapies that can be used for both cancer and pain treatment by targeting shared mechanisms. In progression toward this goal, she directs a translational research program that uses multiple approaches including *in vitro* cell culture, animal models and human studies.



## STEVE DAVIDSON, PH.D.

Assistant Professor, Department of Anesthesiology  
University of Cincinnati College of Medicine

*Project: Thalamo-limbic Circuit Control of Pain*

Steve Davidson earned a B.S. in psychology from the University of New Orleans and a Ph.D. in neuroscience from the University of Minnesota. He was a postdoctoral scholar in the Department of Anesthesiology at Washington University School of Medicine in St. Louis from 2009 to 2014, and in 2015 he joined the faculty of the University of Cincinnati College of Medicine. In 2010, Davidson received a Future Leader in Pain Research award from the American Pain Society.

The ability to tolerate pain is widely recognized to differ between people, by age, and even in the same person across situations. This plasticity represents an underutilized feature of the neural capacity to control pain. The Davidson lab seeks to identify and manipulate the neural circuits responsible for regulating pain tolerance. Specifically, Davidson's research tests the main hypothesis that pain control can be achieved by manipulating neural activity in a thalamo-limbic pathway to reduce suffering from pain.

The Davidson lab has also pioneered the study of human dorsal root ganglia with the goal of improving the translation of pain relieving treatments developed in animal models to people. The Davidson lab recovers live neural tissues from organ donors to use physiological and genetic tools to understand the nature of these first cells in the pain pathway. This research has opened many exciting collaborations with other pain and itch scientists using animal models seeking to confirm the potential of therapeutics in humans. Recent efforts are aimed at tissue engineering an innervated, layered skin graft *in vitro* to remove barriers to directly examining the interaction of skin and nerves during pain, itch, wound healing, and skin disease.



## KATHERINE HANLON, PH.D.

Assistant Professor of Pharmaceutical Sciences  
Director of Research  
Presbyterian College School of Pharmacy

*Project: Dorsal Root Ganglion Macrophages - Function and Impact on Nociception*

Katherine Hanlon earned a B.S. in biochemistry and molecular biophysics and a Ph.D. in pharmacology from the University of Arizona, where she worked with Todd Vanderah. She went on to complete a postdoctoral fellowship in tumor immunology at the Mount Sinai Medical Center with Joshua Brody and Peter Heeger. Hanlon's primary research interests include macrophage-neuron communication in pain processing and the role of tumor-associated macrophages in cancer development. She also studies the mechanisms of dysregulation of cannabinoid receptor signaling in tumor and immune cells in metastatic disease.

Studies in the Hanlon lab are carried out using multiple *in vitro* and *in vivo* models, including leukocytes and neurons isolated from dorsal root ganglia; leukocytes and tumor cells isolated from murine mammary tumors; leukocytes harvested from post-surgical peritoneal adhesions; and human blood monocyte primary cultures. With the support of the Rita Allen Foundation and the American Pain Society, the lab is able to explore the communication that occurs between sensory neurons and macrophages (innate immune cells that are critical in injury response) in dorsal root ganglia. Macrophages in the DRG are a unique population of cells that bear some resemblance to brain microglia, but are functionally distinct and exhibit specific phenotype differences. In response to peripheral injury, DRG macrophages respond to activity in the ascending pain pathways and may alter pain perception. By evaluating the phenotype and function of this unique population, Hanlon hopes to isolate novel and exploitable mechanisms that may be used to develop non-opioid therapeutics for the treatment of persistent pain.



## KYLE BAUMBAUER, PH.D.

Assistant Professor, Anatomy and Cell Biology  
University of Kansas Medical Center

*Project: Targeting ASIC3 for Disruption of Nociceptor Sensitization Following Spinal Cord Injury*

Kyle Baumbauer earned a B.S. in psychology and a B.A. in sociology from the University of Central Florida. He holds an M.A. and Ph.D. in experimental psychology from Kent State University, where he studied molecular mechanisms that allow neurons in the spinal cord to mediate learning and adapt to the environment. This research contributed to an emerging view of the spinal cord not merely as a channel for signals traveling to and from the brain, but as a dynamic group of nerves with important effects on behavior. Baumbauer continued this area of research while a postdoctoral fellow at Texas A&M University, and explored how painful stimulation impacts spinal cord function to understand how the presence of pain affects the recovery of function after spinal cord injury. Baumbauer then did a second fellowship at the University of Pittsburgh, where he began examining the impact of injury and inflammation on peripheral sensory neuron function.

In 2014, Baumbauer joined the faculty at the University of Connecticut School of Nursing, where his research focuses on unraveling the relationship between alterations in gene expression and sensory neuron function, and how these processes contribute to chronic pain following spinal cord injury. Through these investigations, Baumbauer and his team aim to make advances that aid in the treatment of pathological pain. In addition to the Rita Allen Foundation Baumbauer's research is supported by the National Institute of Neurological Disorders and Stroke. He is also a recipient of a Mary Lawrence Research Development Award from the UConn School of Nursing and has been honored as a Sigma Theta Tau Friend of Nursing.



## ARKADY KHOUTORSKY, D.V.M., PH.D.

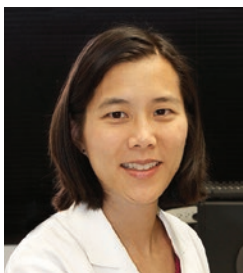
Assistant Professor, Department of Anesthesia  
Alan Edwards Centre for Research on Pain  
McGill University

*Project: Extracellular Matrix-Mediated Spinal Cord Plasticity in Neuropathic Pain*

Arkady Khoutorsky earned a B.Sc. in biology and an M.Sc. in neurobiology, as well as D.V.M. and Ph.D. degrees, from the Hebrew University of Jerusalem. During a postdoctoral fellowship at McGill University, Khoutorsky investigated how regulation of protein synthesis controls neuronal plasticity in the brain and in the pain pathway. He joined McGill's Alan Edwards Centre for Research on Pain in 2016. In addition to the Rita Allen Foundation, Khoutorsky's work is supported by the Canadian Institute of Health Research, the Simons Foundation Autism Research Initiative, an ERA-NET NEURON Grant, and a Louise and Alan Edwards Foundation Grant in Chronic Pain Research.

Khoutorsky's lab is examining how neuronal circuits in the spinal cord are remodeled to promote sensitivity to pain. He is interested in the extracellular matrix, a network of proteins that surrounds neurons. In the brain, this matrix appears to restrict the ability of neurons to form the new structures necessary for learning and memory. Enzymes that degrade the matrix are activated in some chronic pain conditions. Khoutorsky and his team are investigating how such degradation impacts spinal cord neurons that normally inhibit pain signals. They aim to determine how changes in the extracellular matrix might enable the neurons to become "hyperexcitable" and inappropriately propagate pain.





## HELEN LAI, PH.D.

Assistant Professor, Department of Neuroscience,  
Anesthesiology and Pain Management  
The University of Texas Southwestern Medical Center

*Project: Understanding the Molecular and Developmental Basis of Painlessness*

Helen Lai earned a B.A. in chemistry at Cornell University and a Ph.D. in biophysics at the University of California, San Francisco, where she worked with Lily Jan to investigate the structural mechanisms of voltage-gated potassium channels, which help to define the signaling characteristics of neurons. As a postdoctoral fellow in Jane Johnson's lab at the University of Texas Southwestern Medical Center, Lai studied transcriptional mechanisms of differentiation and cell type specification in spinal cord and cerebellar neurons. She was a Sara and Frank McKnight Fellow at UT Southwestern from 2012 to 2015, when she became an assistant professor.

The Lai lab is interested in understanding how somatosensory systems develop and are wired, with a focus on proprioception and pain. With support from the Rita Allen Foundation, Lai's lab has studied the role of a gene implicated in congenital insensitivity to pain—the chromatin-modifying factor PRDM12. Her lab has found that while PRDM12 plays a critical role in the embryonic development of nociceptive sensing neurons, it appears to play a completely different and unknown role in the adult mouse. Her lab's findings point to a critical role of PRDM12 in the generation of nociceptors whose molecular mechanisms might give insight into pathways needed to regenerate nociceptors upon sensory neuron degeneration, or injury. Her lab is generally interested in the genetics underlying pain sensation and has begun a forward genetic screen in mice to identify new genetic players involved in nociception.



## CANDICE PAULSEN, PH.D.

Assistant Professor, Department of Molecular Biophysics and Biochemistry  
Yale University

*Project: Uncovering the Regulation of TRPA1 by Irritants,  
Cofactors and Proteins*

Candice Paulsen earned a B.S. in genetic biology at Purdue University and a Ph.D. in chemical biology at the University of Michigan. As a graduate student with Kate Carroll at the University of Michigan and Scripps Research Institute, Paulsen studied redox regulation of signal transduction cascades important to cancer. As a postdoctoral fellow with David Julius at the University of California, San Francisco, she used cryo-electron microscopy to determine the structure of the TRPA1 ion channel, an important detector of noxious chemical agents also known as the “wasabi receptor.” Paulsen joined the Yale University faculty in 2018.

Nociception is a process by which distinct, potentially harmful thermal, mechanical or chemical stimuli activate specialized sensory neurons to initiate pain signals and neurogenic inflammation. The wasabi receptor, TRPA1, is expressed in chemosensory neurons innervating the periphery and visceral organs, and is activated by a chemically diverse panel of environmental toxins. TRPA1 is also activated by endogenous electrophiles produced during inflammation, which contribute to chronic pain associated with arthritis, asthma and colitis. The Paulsen laboratory is taking a multidisciplinary approach to understand how TRPA1 is regulated by small molecules, cofactors and proteins, with an eye toward uncovering novel avenues for analgesic and anti-inflammatory agent development that directly target initiation of pain and neurogenic inflammatory signals, reducing or eliminating the side effects and risks of addiction. Initial work in Paulsen's lab will focus on unresolved questions about how TRPA1 is regulated: determining the structure of TRPA1 in its open, active conformation; identifying endogenous cofactor(s) that stabilize functional TRPA1; and finding proteins that interact with TRPA1 in its basal and activated states.



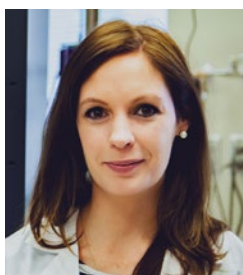
## MICHAEL BURTON, PH.D.

Assistant Professor, Neuroscience  
University of Texas at Dallas

*Project: Novel Models to Assess Sufficiency of Single Cell Paradigms in CB1R-Analgesia*

Michael D. Burton earned his B.S. and Ph.D. from the University of Illinois at Urbana-Champaign. His introduction into the pain field was quite serendipitous. During his first postdoc at the University of Texas Southwestern with Joel Elmquist, he was working on a project that assayed peripheral sensory neurons in the context of metabolism. As he conducted experiments, it became clear that the mice in his experiments were different—they did not seem to exhibit pain in a similar way throughout their lives, and he was intrigued to find out why. Luckily Elmquist was supportive—he allowed Burton to seek a second postdoc mentor in the field of pain and use these animals for a novel pain research project. In 2015, Burton found his second postdoc mentor, Ted Price (2009 Pain Scholar) at UT Dallas, and he began working on the biology of pain. Burton then joined the University of Texas at Dallas faculty in 2017.

Recent initiatives in the Burton lab have revealed the importance of how different cell types recognize and respond to pain stimuli. Mastering these single-cell paradigms will lead to a better understanding of mechanisms in pain plasticity and evolve new therapeutics. Burton's work sits at an important interface between the immune and nervous system and strives to understand the complex nature of their interconnectivity. The lab has three areas of focus: inflammation, aging, and cannabinoid signaling. Burton hopes to understand how factors like age, sex, and endogenous cannabinoid signaling modulate pain outcomes.



## MEAGHAN CREED, PH.D.

Assistant Professor, Anesthesiology  
Washington University in St. Louis

*Project: Synaptic Adaptations Underlying Affective Symptoms of Chronic Pain*

Meaghan Creed received her B.S. from the University of Toronto Scarborough and her Ph.D. from the University of Toronto. She completed a post-doctoral fellowship with Christian Lüscher at the University of Geneva, Department of Basic Neuroscience, and joined the faculty of Washington University in St. Louis in 2018.

The Creed Lab focuses on synaptic plasticity and neuromodulation within defined neural circuits in the ventral basal ganglia, a collection of brain structures involved in reward learning and selection of flexible behavior. Specifically, Creed asks how chronic pain, addictive drugs, or genetic mutations alter the function of these neural circuits, and how circuit dysfunction contributes to symptoms of chronic pain, and substance use disorders. Her ultimate goal is to leverage insight from circuit studies to develop novel neuromodulation for these disorders, including deep brain stimulation and focused drug delivery. By first determining how neuronal and circuit adaptations drive specific behavioral symptoms of disease, she can establish a strategy for targeted circuit manipulation in a disease state. Creed then rationally designs neuromodulation paradigms and validates them in model systems to provide novel strategies to treat symptoms at the interface of chronic pain, mood, and substance use disorders.



## PETER GRACE, PH.D.

Assistant Professor, Symptom Research  
University of Texas MD Anderson Cancer Center

**Project: Antibody Receptor Signaling via Astrocytes - A New Pathway for Neuropathic Pain**

*In conjunction with Open Philanthropy*

Peter Grace earned his B.S. and Ph.D. from the University of Adelaide. Grace joined the faculty at University of Texas, MD Anderson Cancer Center in 2017. He studies pain that becomes chronic and outlasts the period of healing, which is a major medical challenge. The Grace lab investigates the neuroimmune interactions that drive chronic pain. After injury to sensory nerves, glial cells, such as microglia and astrocytes, are activated throughout the central nervous system. These activated glia secrete neurotransmitters and cytokines that increase the excitability of neurons in pain pathways.

Grace is focused on the open question of how spinal cord astrocytes are persistently activated in such remote regions. This line of research is also being expanded to determine how activated glia in the brain also contribute to common comorbidities of chronic pain, including depression, anxiety, and cognitive impairments. The ultimate goal of this work is to find new ways to treat chronic pain. The Grace lab is investigating the function of a completely unexplored receptor that is expressed by astrocytes, which may provide insight into why these cells are persistently activated after remote nerve injury.



## JORDAN MCCALL, M.P.H., PH.D.

Assistant Professor, Anesthesiology  
Washington University in St. Louis

**Project: Using Persistent Homology to Model and Predict Spontaneous Pain Behavior**

*In conjunction with Open Philanthropy*

Jordan McCall received his B.S./B.A. and M.P.H. from the University of Missouri Columbia, and his Ph.D. from the Washington University in St. Louis. McCall leads a multidisciplinary research program aimed at understanding the neural mechanisms underlying the emotional distress associated with stress, chronic pain, and addiction. The long-term goal of the laboratory is to use a neural circuit-level understanding of the brain systems that are disrupted in anxiety and chronic pain to reverse these conditions. If these questions become intractable, the laboratory plans to develop new wireless technology to interface with the nervous system and explore new approaches to data analysis, to access the most information from collected data.

One of the biggest challenges McCall and his lab face in using rodents as models of complicated human conditions is that one cannot ask the animals how they feel. A simple question, but a very difficult one to answer. McCall will be working to overcome this barrier by extracting detailed information from videos of the animal's behavior to determine whether they are in pain, or distress. He will use new types of data analysis from mathematics to essentially ask the mice that simple question, "How do you feel?" McCall and his lab aim to make strides in identifying stress and pain in animals without having to disturb their daily routine. This approach will hopefully enable new strategies for understanding neural circuit function and therapeutic development.



## VIVIANNE TAWFIK, M.D., PH.D.

Assistant Professor, Anesthesiology, Perioperative, and Pain Medicine  
Stanford University Medical Center

**Project: Engaging Pro-resolution Microglia to Block the Transition to Chronic Pain**

*In conjunction with Open Philanthropy*

Vivianne Tawfik graduated from McGill University with a B.Sc., and received her M.D./Ph.D. degrees from Dartmouth College. After completing her anesthesiology residency and Pain Medicine fellowship at Stanford, Tawfik joined the lab of Gregory Scherrer (2014 Pain Scholar). In Scherrer's

lab, she focused on the expression of delta and mu opioid receptors in the peripheral and central nervous system, and established that spinal cord microglia do not express the mu opioid receptor. During a second postdoc with Dave Clark, also at Stanford, Tawfik focused on microglial modulation in post-injury pain using mouse models. She subsequently launched her own clinically-informed basic science lab and joined the faculty of Stanford University in 2017.

The mission of the Tawfik lab is to advance our understanding of neuroimmune contributions to chronic pain in a thoughtful manner, with our patients always in mind. Tawfik is particularly interested in understanding the unique underpinnings of various types of chronic pain and how central nervous system glial cells (astrocytes and microglia) contribute to the transition from acute to chronic pain. Tawfik and her lab will dive more deeply into the contributions of spinal cord microglia, using transgenic manipulations and microglial transcriptome analyses in a mouse model of complex regional pain syndrome—a disease that affects the limbs after minor fracture, or surgery. She expects findings in this model will also extend to other forms of chronic pain and allow for the development of more specific glial-targeted therapeutics.

## SCHOLARS 2020



### ISHMAIL ABDUS-SABOOR, PH.D.

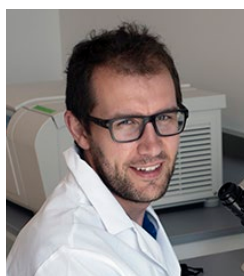
Principal Investigator, Zuckerman Mind Brain Behavior Institute  
Assistant Professor, Biological Sciences  
Columbia University

*Project: Discovering Behavioral Signatures of Pain at Millisecond Timescales*

*In conjunction with Open Philanthropy*

Ishmail Abdus-Saboore earned his B.S. from North Carolina A&T University and received his Ph.D. from the University of Pennsylvania. He completed two postdoctoral fellowships at Weill Cornell Medicine and the University of Pennsylvania. Abdus-Saboore joined the faculty at the University of Pennsylvania in 2018, and in 2021 his lab moved to the Zuckerman Mind Brain Behavior Institute and Department of Biological Sciences at Columbia University. In 2021, he also received the Sloan Research Fellowship award.

The Abdus-Saboore lab focuses on a long-standing question—how does the nervous system encode a soft gentle caress versus a harsh painful stimulus? To accomplish this, Abdus-Saboore uses neurobiology, computational biology, and mathematics to objectively measure pain—a sensory experience that is inherently subjective. Traditionally, researchers have applied sensory stimuli to the rodent paw and tried to infer the animal's pain state based on the singular readout of whether the animal moved its paw or not. The problem is, animals will lift their paw to both innocuous and noxious stimuli; and with that sole measurement parameter, there is a high likelihood of incorrectly assigning the animal's sensory experience. An innovation in Abdus-Saboore's work is to use videography to take thousands of images per second to measure sub-second pain behaviors, and coupled with statistical modeling and machine learning to develop rodent “pain scales.”



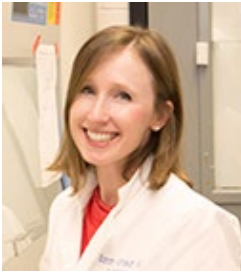
### GEOFFROY LAUMET, PH.D.

Assistant Professor, Physiology  
Michigan State University

*Project: Cellular and Molecular Mechanisms Underlying Remission and Relapse of Pain*

Geoffroy Laumet graduated with a Ph.D. from Lille University School of Medicine in France. Laumet joined the faculty of Michigan State University in 2019.

The Laumet lab is interested in understanding why pain becomes chronic and how can one stop it. While it is obvious that neurons convey pain signaling throughout the body, neurons do not work in isolation and are constantly communicating with and getting influenced by other cells. Laumet is particularly interested in the contribution of non-neuronal cells to chronic pain. He thinks that impaired communication between “pain-sensing” neurons and their surrounding cells may result in chronic pain. For example, he has discovered that anti-inflammatory molecules secreted by cells from the immune system prevent pain-sensing neurons from becoming persistently activated—this constant activity is the cellular basis of chronic pain. Laumet hopes that a better understanding of neuron/non-neuronal cell communication will lead to the development of new and better analgesics.



## SARAH LINNSTAEDT, PH.D.

Assistant Professor, Anesthesiology  
University of North Carolina at Chapel Hill

*Project: FKBP51 Inhibition to Prevent Chronic Pain Following Traumatic Stress*

*In conjunction with Open Philanthropy*

Sarah Linnstaedt received her B.S. from Virginia Tech and her Ph.D. from Georgetown University. She completed a post-doctoral fellowship at Duke University, studying the role of small regulatory RNAs in the pathogenesis of B cell lymphomas. She joined the faculty of the University of North Carolina at Chapel Hill in 2012.

Almost all individuals will experience at least one traumatic injury in their lifetime. While the majority of these individuals recover, a substantial subset develop chronic post-traumatic pain. Linnstaedt is interested in better understanding how to predict which individuals will recover versus develop CPTP following trauma, and how to prevent CPTP in those at high risk. To address this goal, the Linnstaedt lab uses translational research approaches to discover genetic and molecular vulnerability factors that predict CPTP and to identify promising targets for therapeutic preventative interventions.

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## ANDREW J. SHEPHERD, PH.D.

Assistant Professor, Department of Symptom Research  
The University of Texas, MD Anderson Cancer Center

*Project: Neuro-immune Interactions in Pain Associated with Cancer and Chemotherapy*

Andrew J. Shepherd graduated with a B.S. and Ph.D. from the University of Manchester Institute of Science and Technology in the United Kingdom. He completed post-doctoral fellowships at the University of Iowa and Washington University in St. Louis. Shepherd joined the faculty at the University of Texas in 2019.

The Shepherd lab focuses on how injury, inflammation, and cancer interact with the nervous system to cause pain. Shepherd is particularly interested in how chronic illnesses disrupt the immune system, thereby increasing pain risk. Macrophages, a type of immune cell, are important contributors to pain. Macrophages infiltrate damaged tissue to clear debris and infection. Ordinarily, this process eventually resolves, promoting healing. He hypothesizes that chronic pain often stems from macrophages failing to make this transition from a “damage response” to a “pro-repair” state. In such cases, macrophages continually sustain inflammation, causing nearby nerves to remain hyper-excitable and drive chronic pain. Our knowledge of these mechanisms is surprisingly limited, a problem that is set to become more widespread as chronic illnesses and cancer survivorship improve. The Shepherd Lab hopes that improving our understanding of these neuro-immune interactions, will identify novel therapeutic targets and facilitate the development of safe and effective analgesics.



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92 Nassau Street, 3rd Floor  
Princeton, New Jersey 08542  
609-683-8010  
[www.ritaallen.org](http://www.ritaallen.org)