To better serve our community: Introducing new Associate Editors and an expanded Editorial Advisory Board for 2015

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The Journal of General Physiology is beholden to no one but our editors, authors, reviewers, and readers. As a nonprofit journal run by scientists, for scientists, we are not governed by the board of a scientific society. We have no shareholders clamoring for revenue and no masters but the scientific community as a whole. JGP is what we make of it, limited only by our imaginations and the effort we are willing to invest. My vision for JGP is based on my view on the role of science in society and the role of journals in the scientific community.

Scientists work collectively to make the world a better place. Bringing new knowledge and understanding of ourselves and our world into being has the specific effect of improving human health and the general effect of elevating humanity with wisdom. The scientific process—in which evidence is at the center of every argument, every assumption can and should be questioned, and interpretations last only until new evidence is revealed—creates a shared system of values that is a model for civil societies. The core assumption, that we all strive to reach truth, creates a flat hierarchy of mutual respect and trust.

Journals are the primary medium by which scientists present evidence and argue interpretation. High quality peer review is, of course, essential to making sure evidence is sound and clearly presented. However, I see the role of JGP as something more than just reviewing and publishing manuscripts. As a journal run by scientists, for scientists, JGP can and should serve all the goals of its constituents. The challenges for me as editor are to make sure that JGP is welcoming and inclusive, responsive to the needs of our community, and faithfully reflects our collective voice. The physiology community has a proud tradition of applying quantitative, rigorous approaches to develop mechanistic insight into essential biological processes. I am committed to making sure JGP remains relevant, viable, and strong so that this tradition may continue.

As physiology as a field has evolved, JGP has evolved with it. Single-channel recording ushered in an era of single-molecule approaches for interrogating a variety of physiological processes. Advances in imaging technologies pushed quantitative measurement of cell dynamics further than we imagined possible. Structural and spectroscopic approaches have furthered our understanding of intra- and intermolecular mechanisms of signaling, drug action, and sensory transduction. New probes for lipid and soluble cargo have advanced the study of membrane trafficking and metabolism.

For 2015, I am pleased to announce a major expansion of the JGP Editorial Advisory Board. The Editorial Advisory Board of JGP has long reflected the interests of the physiology community. Because the size of the Board has remained fairly constant over the years, however, we have not always kept pace with new developments in the field. As you may read in the short bios of many of the 25 new Board members that follow, they are a truly amazing group. We welcome men and women from across the forefront of physiology, individuals who are pushing technology while insisting on rigor and care in their own work. Together with our continuing Board members, we hope to provide fair, constructive, and consistent reviews across the entire scope of physiology.

A look at our new masthead will show that we have three new Associate Editors, Merritt Maduke, Eduardo Rios, and Rick Aldrich, and we have bid goodbye to emeritus Associate Editors Chris Miller and Larry Palmer. To Chris and Larry: it has been an honor and pleasure to serve with you. To Merritt, Eduardo, and Rick: the weekly editors’ meetings with continuing Associate Editors Angus Nairn, Rick Moss, and Kenton Swartz, Executive Editor Elizabeth Adler, and Managing Editor David Greene will be richer because of your participation. At the end of my first year as JGP Editor-in-Chief, I thank our entire community of editors, authors, reviewers, and readers for your continued support of our journal. We are strong because of you.
Ed performed his PhD research at the University of Washington in Seattle, where he studied the neuronal growth cone protein, GAP-43, and the ubiquitous calcium sensor, calmodulin. He then packed up his pickup truck and headed east to Yale University, in New Haven, CT, for a postdoc in the laboratory of Reinhard Jahn, where he began his focus on neuronal exocytosis. In 1996 he started his own laboratory at the University of Wisconsin-Madison, where he is a Professor of Neuroscience and, since 2005, an Investigator of the Howard Hughes Medical Institute. His laboratory studies aspects of membrane traffic in neurons and neuroendocrine cells. Specific areas of interest include: the mechanisms by which calcium ions trigger membrane fusion, the structure and dynamics of fusion pores, and the mechanism of action of the clostridial neurotoxins (which block exocytosis). A more recent focus is on aspects of synaptic plasticity. His laboratory addresses these questions using a variety of approaches, ranging from reconstituted membrane fusion reactions to optical and electrophysiological studies of neurons cultured from genetically modified mice. In his spare time, he tries to lift heavy things.

Dr. Heping (Peace) Cheng

Dr. Heping (Peace) Cheng received his bachelor and master degrees in applied mathematics and mechanics and biomedical engineering, with physiology as his minor, from Peking University, China. Upon graduation, he served as a junior faculty member in the Department of Electrical Engineering at the same university before earning his PhD degree in physiology from University of Maryland at Baltimore. He then joined the National Institutes of Health (NIH) Intramural Research Program as a senior staff fellow in 1996, was selected as a tenure-track investigator in 1998, and became the Head of the Calcium Signaling Section in the Laboratory of Cardiovascular Science, National Institute of Aging, NIH. He was promoted to senior investigator in 2004. He is now a senior investigator heading the Laboratory of Calcium Signaling & Mitochondrial Biomedicine in the Institute of Molecular Medicine at Peking University. He was elected to the Chinese National Academy of Sciences in 2013.
“Ca²⁺ sparks” in 1993 and “mitochondrial superoxide flashes” in 2008, he strives to resolve elemental cell signals at the nanoscopic and millisecond resolution in the pursuit of principles of physiological signaling. In particular, his current focus is on the mechanism, regulation, and biology of mitochondrial flashes while continuing his research line on calcium signaling from the viewpoint of systems biology.

Cynthia Czajkowski
Born and raised in New York City, Cindy received a BA from New York University and a PhD from the State University of New York—Downstate Medical Center, where she studied GABA_A receptor trafficking under the guidance of Dr. David Farb. She then moved uptown to Columbia University to do a postdoc with Dr. Arthur Karlin, where she studied nicotinic acetylcholine receptors. Here, she was introduced to the use of photoaffinity labeling and protein chemistry as well as the substituted cysteine accessibility method to investigate structural mechanisms underlying nicotinic acetylcholine receptor function. In 1994, she joined the faculty at the University of Wisconsin-Madison and is currently a Professor in the Department of Neuroscience. She has continued to study pentameric ligand-gated ion channels with a focus on GABA_A receptors and recently, bacterial pentameric ligand-gated ion channel homologues. Her laboratory uses an array of approaches, including voltage- and patch-clamping recording, voltage-clamp fluorometry, and site-directed spin labeling electron paramagnetic resonance spectroscopy to elucidate structural mechanisms underlying how neurotransmitters activate pentameric ligand-gated ion channels and how allosteric drugs modulate their activity.

Ana Maria Gomez
After graduating at the Pharmacy School of University Complutense of Madrid (UCM), Ana received her PhD degree in 1994 from the same institution, performing her research work under the direction of Dr. Carmen Delgado, at CSIC (Spanish Research Council)-UCM institute. Her thesis work was focused on the alterations in ionic currents in cardiac hypertrophy, to get better insights into the cellular basis of arrhythmia. Ana extended this study during a postdoctoral stage at the University of Maryland, working in Jon Lederer’s laboratory (1995–1997). There, she specialized in intracellular calcium (Ca²⁺) homeostasis and analyzed Ca²⁺ sparks in heart failure models.

In 1998, Ana was awarded a competitive permanent research position from the CNRS (Centre National de la Recherche Scientifique) in France, and received the “Habilitation to direct Research” diploma from University of Montpellier 1, in 2001. In 2008 she was appointed a competitive permanent director of research position from Inserm (Institut National de la Santé et de la Recherche Médicale). Since 2011 she heads an Inserm team entitled “Calcium signaling and cardiac physiopathology” at the Pharmacy School of University of Paris Sud, and from January 2015 she will lead the Inserm Unit 1180 at the same location, entitled “Signaling and cardiovascular physiopathology.” She is interested in Ca²⁺ signaling in cardiomyocytes, and its alterations during cardiac hypertrophy, failure, and arrhythmia. She is currently focused in arrhythmia-related sudden cardiac death mechanism and excitation–contraction coupling in normal, hypertrophied, and failing hearts, to better elucidate the key role of Ca²⁺ in cardiac pathophysiology.

Katherine Henzler-Wildman
Katie received her BA in Chemistry from Cornell University in 1998 where she was first introduced to the field of NMR in the laboratory of Linda Nicholson. She then moved to the University of Michigan where she worked with A. Ramamoorthy for her PhD, using solid-state NMR methods to study the mechanism of lipid bilayer disruption by the human antimicrobial peptide LL-37. Putting her interest in membrane proteins on hold, Katie spent her postdoc in Dorothee Kern’s laboratory investigating the functional importance of protein dynamics on multiple timescales in the soluble enzyme, adenylate kinase. In 2008 she started her own laboratory at Washington University in St. Louis, combining her interests in membrane proteins and functional dynamics to study the transport mechanism of the small multidrug resistance transporter EmrE. Her group demonstrated that solution NMR could directly monitor the conformational exchange of EmrE between states accessible to opposite sides of the membrane, providing experimental evidence that the two monomers of the antiparallel EmrE homodimer swap conformations to transport substrate across the membrane. More recently, her laboratory has focused on questions of multidrug recognition and how EmrE achieves coupled antiport of drugs and protons. Katie’s research uses the power of NMR to quantitatively measure protein dynamics on multiple timescales with site-specific resolution to probe the relationship between structure, dynamics, and function in ion channels and transporters.
Vasanthi Jayaraman
Vasanthi Jayaraman received her BS from Madras University and MS in Chemistry from the Indian Institute of Technology, Chennai India. She completed her PhD in Chemistry in 1995 from Princeton University. Her doctoral work focused on investigating the allosteric mechanism of hemoglobin using time-resolved spectroscopy. She then received a Damon Runyon Walter Winchell postdoctoral fellowship and worked with Dr. George Hess at Cornell University until 1997. Her postdoctoral work focused on rapid kinetic investigations of ion channels using caged compounds. In 1997 she joined the Chemistry department at Marquette University and has since combined her spectroscopic and electrophysiological background to study structure–dynamics of ligand-gated ion channels. She is currently a Professor at the biochemistry department at University of Texas Health Science Center at Houston. PHOTO COURTESY OF V. JAYARAMAN

Andrea Meredith
Andrea did undergraduate studies at the University of Maryland before giving in to her love of the Wild West and returning to Texas to earn her PhD in Neuroscience from UT Southwestern in 2000. Continuing west, she headed off to California for postdoctoral studies with Rick Aldrich at Stanford and entered the ion channel field by generating a mouse knockout of BK, “The King.” In 2006, she moved to the University of Maryland School of Medicine in Baltimore, where she is currently an Associate Professor in the Department of Physiology. Andrea’s research on the physiological roles for the BK channel have taken her on a wayward journey through the brain, bladder, heart, and even the bucolic grasslands of New Zealand, where BK channels are a target of the fungal neurotoxin that causes Ryegrass Staggers. Using diverse techniques encompassing the creation of transgenic mice, patch-clamp electrophysiology, multi-electrode arrays, and telemetry, she and her laboratory team ultimately endeavor to identify specific biophysical properties of ion channels that can influence the physiology of the whole animal. Recent studies in the laboratory have focused on the brain’s circadian clock, where daily regulation of BK channel properties plays a unique role in the circadian patterning of excitability that drives physiological rhythmicity. PHOTO COURTESY OF A. MEREDITH

Mark T. Nelson
Mark received his BA in Mathematics and Biology with Honors from Tufts University in 1976, and his PhD in Neural Sciences from Washington University in St. Louis, Missouri, in 1980 under Professor Mordecai P. Blaustein. He went on to receive postdoctoral training with Professor Blaustein at the University of Maryland and with Professor P. Läuger at Universität Konstanz in West Germany. He is currently a University Distinguished Professor and Chair of the Pharmacology Department at the University of Vermont. He is also a Professor at the Institute of Cardiovascular Science at the Manchester University, Manchester, UK, and a Visiting Professor in the Department of Pharmacology at the University of Oxford, Oxford, UK. His overall research goal is to understand the control of smooth muscle and endothelial cell function by ion channels and calcium signaling. Areas of particular interest are the small blood vessels controlling cerebral blood flow (CBF) and neurovascular coupling; understanding how sympathetic nerves, smooth muscle cells, and endothelial cells communicate (vascular crosstalk) to control the function of resistance-sized peripheral arteries; and understand the roles of ion channels and calcium signaling in the control of urinary bladder function in health and disease. PHOTO COURTESY OF M.T. NELSON

Joseph Metzger
Joe Metzger is Professor and Chair of Integrative Biology and Physiology at the University of Minnesota’s School of Medicine. He is a member of The Lillehei Heart Institute and holds the Maurice B. Visscher Endowed Chair in Physiology. Joe received a Bachelor’s degree in Natural Science from Saint John’s University (1980), a Master’s degree in Biology and Exercise Physiology from Ball State University (1982), a PhD degree in Biology/Physiology under the mentorship of Dr. Robert Fitts from Marquette University (1985), and performed postdoctoral studies with Dr. Richard Moss at the University of Wisconsin (1991). His laboratory created a cardiac muscle–cell system that allows the transfer of genes into heart cells in order to assess the impact of those genes on the production of force and motion, the major function of cardiac muscle cells. The approach has the advantage of shedding light on the primary role of a normal or mutated gene in an otherwise normal muscle cell. Developing the technique, however, was a considerable task, as the technology to transfer genes and culture differentiated muscle cells was largely unavailable at the time. Joe’s research findings have opened the path to treatment for a variety of heart diseases. PHOTO COURTESY OF J. METZGER

Photo courtesy of M. B. Visscher Endowed Chair in Physiology
Keir C. Neuman
Keir, a native of Canada, received a BA in Physics and Applied Math from the University of California Berkeley in 1994. He remained at Berkeley for one year in the atomic physics laboratory of Roger Falcone. After attending a colloquium by Steven Block, he was captivated by biophysics and began a PhD in physics at Princeton University, eventually joining Steve Block’s laboratory to investigate transcription and transcriptional pausing using single-molecule optical trapping techniques. In 2004 he was awarded a Human Frontiers Fellowship in the laboratory of David Bensimon and Vincent Croquette at the Ecole Normale Supérieure in Paris, France, to investigate DNA topology and topoisomerases using magnetic tweezers. In 2007 he joined the Laboratory of Molecular Biophysics in the National Heart, Lung, and Blood Institute at the National Institutes of Health where his laboratory develops single-molecule manipulation and visualization tools to study DNA processing by topoisomerases and helicases, and collagen processing by matrix metalloproteinases. PHOTO COURTESY OF K.C. NEUMAN

Yasushi Okamura
Dr. Okamura received his MD and PhD degrees from the University of Tokyo. His PhD study addressed the changes of the properties of ion channels during early cell development of the ascidian embryo. He also learned molecular biology in the laboratory of Dr. Gail Mandel at Department of Neurobiology & Behavior, State University of New York at Stony Brook. He has been studying ion channel biophysics, and regulation of ion channel function and expression. He also has a background in developmental neurobiology of invertebrates. Dr. Okamura’s recent contribution is identification and characterization of two voltage sensor–containing proteins lacking authentic pore domains. One was the voltage-sensing phosphatase (VSP), which has the voltage-sensor domain connected with a phosphoinositide phosphatase. VSP operates as depolarization-activated phosphoinositide phosphatase with some unique properties of substrate specificity and regulation by voltage. VSP has proven a useful molecular tool for acute depletion of phosphoinositides in cells, and as a protein-based voltage probe that enables imaging of membrane potential, such as for individual action potentials in neurons. The other protein that Dr. Okamura and his colleagues have been studying is the voltage-sensor domain–only protein (VSP; now called Hv1, or its gene name is HVCN1), which turned out to be the molecular correlate for voltage-gated proton channel. Hv1 shows voltage-dependent gating and proton-selective permeation based only on four transmembrane segments corresponding to the voltage-sensor domain found in other voltage-gated ion channels. Hv1 is one of the minimal cation channels encoded in the mammalian genome. Along with its compact molecular size, ease of heterologous expression, and availability of recent structural information, it is appreciated for understanding many fundamental questions of ion channel biophysics, including gating, ion selectivity, coupling between gating and permeation, and cooperativity of gating by multimerization. Dr. Okamura is also interested in mechanisms of linkage between two aspects of biological membranes: the source of lipid signals, and the place of electrical signals. PHOTO COURTESY OF Y. OKAMURA

Geoffrey S. Pitt
Geoff received his BA degree in Classics from Yale. He completed MD–PhD training at Johns Hopkins, where he worked with Peter Devreotes studying adenylyl cyclase in Dictostelium. He then moved to Stanford for clinical training and a postdoctoral fellowship with Richard Tsien. At Stanford, he defined contributions of calmodulin to calcium-dependent inactivation of L-type calcium channels. He moved to Columbia in 2001 where he started his own laboratory, which studied calcium/calmodulin regulation of various ion channels. In 2007, he became the Director of the Ion Channel Research Unit at Duke University, where he is currently Professor of Medicine, Neurobiology, and Pharmacology & Cancer Biology. Geoff’s laboratory studies various ion channel disorders (channelopathies) associated with cardiac arrhythmias, epilepsies, ataxias, and autism using techniques that span from x-ray crystallography to animal models and patient studies. He currently also serves as an Associate Editor of The Journal of Clinical Investigation. PHOTO COURTESY OF G.S. PITT

Murali Prakriya
Murali obtained his BS in Electrical Engineering from the Indian Institute of Technology, Kharagpur, India, and an MS from the University of Miami in Biomedical Engineering. In 1997, he received his PhD in Neuroscience from Washington University, St. Louis, under the mentorship of Chris Lingle, working on the functional coupling of voltage-gated Ca\(^{2+}\) channels with...
K’ channels. After a brief postdoctoral fellowship at Washington University with Drs. Steve Mennerick and Charles Zorumski to study the role of Na’ channels in regulating neurotransmitter release, he joined the laboratory of Richard Lewis, Stanford University, as a postdoctoral fellow. Here, he gained expertise on the molecular biophysics of store-operated Ca²⁺ release–activated Ca²⁺ (CRAC) channels and developed a rigorous biophysical fingerprint of the channel that ultimately proved valuable for the identification of the Orai proteins as the molecules encoding CRAC channels. In 2005, Murali joined the faculty of Northwestern University where he has continued his work on investigating the molecular and cellular mechanisms by which CRAC channels are gated, and the mechanisms of their exquisite Ca²⁺ selectivity. His laboratory has also expanded its interests to study the physiological ways by which CRAC channels regulate gene expression, airway epithelial cell function, and the development of neural stem cells. Photo courtesy of M. Frakova.

Catherine Proenza

Cathy Proenza earned a BS in Zoology from the University of California, Davis, in 1984 and an MS in Physiology from the University of Illinois in 1986, where she studied intracellular pathways that control dark adaptation in rod photoreceptors. She then spent time examining alternative pathways in life. She lived at an intentional community in California and an off-the-grid cabin in Colorado, worked intermittently as a bicycle mechanic and wildland firefighter, and studied rock climbing and art. Eventually, she returned to the scientific fold, earning a PhD in Physiology from Colorado State University in 1999, where she studied excitation–contraction coupling in skeletal muscle with Kurt Beam. As a postdoc, Cathy studied HCN channel biophysics, first with Eric Accili at Simon Fraser University in Vancouver BC, and then with Gary Yellen at Harvard Medical School. After beginning her own laboratory at the University of Connecticut, Cathy moved back to Colorado in 2008 to join the Department of Physiology and Biophysics at the University of Colorado School of Medicine. Cathy’s research program focuses on the physiology of pacemaker myocytes from the sinoatrial node of the heart and on the peculiar biophysics of HCN4 ion channels, which are important for pacemaking. She employs whole-animal and single-cell electrophysiology, biochemistry, mass spectrometry, and immunofluorescent microscopy. More recent projects also incorporate FRET, mathematical modeling, and x-ray crystallography. Photo courtesy of C. Proenza.

Lucia Sivilotti

Lucia graduated in Pharmaceutical Chemistry from Ferrara University, Italy. Her final-year project (measuring acetylcholine release from neocortex) triggered her interest in basic research, but also inspired her to find and learn faster techniques that could directly monitor neuronal function. Inevitably, this led to electrophysiology, and she moved for her PhD to the London laboratory of Andrea Nistri, where she characterized the pharmacology of GABAC receptors. It was in her postdoctoral years in David Colquhoun’s laboratory at University College London that Lucia finally got to recording molecular function in real time, and used single-channel recording to investigate neuronal nicotinic and glycine channels. Lucia set up her own laboratory in the Pharmacology Department of the School of Pharmacy in 1997, and returned in 2003 to University College, where she holds the AJ Clark chair in Pharmacology. Her laboratory works on understanding the molecular mechanism of activation of channels in the nicotinic superfamily, and applies single-channel recording to the
Justin Taraska
Justin Taraska received his BA in biology from Reed College in 1999 and earned his PhD in cell biology from Oregon Health and Science University in 2004 in the laboratory of Wolfhard Almers. During his PhD, Justin investigated the processes of triggered exocytosis and endocytosis in neuroendocrine cells with high resolution microscopy methods. He conducted his postdoctoral research in the laboratory of William N. Zagotta at the University of Washington where he received a Jane Coffin Child Memorial Fellowship. During his postdoc, Justin developed and used novel fluorescence methods to study the structure of ion channels in biological membranes. In 2010, Dr. Taraska became an Investigator at the National Heart Lung and Blood Institute (NHLBI), National Institutes of Health (NIH). Dr. Taraska is a 2012 PECASE recipient. He is also the co-director of the analytical and quantitative light microscopy course (AQLM) at the Marine Biological Lab in Woods Hole, MA. Dr. Taraska’s laboratory studies the structural cell biology of exocytosis and endocytosis with advanced imaging methods including live cell microscopy, super-resolution fluorescence, and electron microscopy.

Dirk Slotboom
Dirk Slotboom received his BSc and MSc in chemistry at the VU University in Amsterdam in 1994. He then moved to Groningen, The Netherlands, for a PhD in microbiology in the laboratory of Wil Konings and Juke Lolkema (2001). With the aim to learn structural biology, he went to Cambridge, UK, as a postdoctoral fellow in the MRC unit headed by John Walker, in the laboratory of Edmund Kunji. Although his desire to determine high resolution structures of membrane proteins never materialized in Cambridge, it did so when he moved back to The Netherlands in 2004 as a tenure track/assistant professor in the Biochemistry department of the University of Groningen. Since 2014 he is full professor in biochemistry. Dirk Slotboom now uses a combination of biophysical (x-ray crystallography, spectroscopy), biochemical (membrane enzymology), and microbiological methods to study the mechanisms of solute transport across membranes. He currently focuses on vitamin transporters (both ATP-driven and facilitators) and ion-coupled glutamate and aspartate transporters found in prokaryotes.

Susan Treves
Susan received her BSc (1981) and MSc (1983) in Microbiology and Immunology at McGill University. She then decided to head back to Italy, join the group of Tullio Pozzan who was working with the first fluorescent calcium indicators developed by Richard Tsien, and start her PhD. During this time she acquired first-hand knowledge on the uses, misuses, and pitfalls of the newly developed calcium indicators. She then followed her husband Francesco Zorzato for 2 years in David MacLennan’s laboratory at the University of Toronto, where she acquired skills in biochemistry and cell and molecular biology. She returned to Italy and obtained her PhD in Molecular & Cellular Biology & Pathology from the University of Padova in 1990. She and her husband then decided to join forces and work together at the University of Ferrara on various aspects of skeletal muscle physiology, including the identification of novel proteins in the sarcoplasmic reticulum, the elucidation of how mutations in the gene encoding the ryanodine receptor 1 (RYR1) affect its function, and the identification of proteins interacting with and regulating the function of the ryanodine receptor. They then moved to Basel, Switzerland, in the Department of Biomedizin and Anesthesia where they continue to work on calcium homeostasis in skeletal muscle and on the role of ryanodine receptor 1 in nonskeletal muscle cells. Her research interests aim at characterizing the functional effect of mutations in the RYR1 gene associated with malignant hyperthermia and core myopathies due to dominant and recessive mutations. A unique aspect of her research is the fact that functional studies are performed on myotubes established from biopsies of affected patients or by exploiting the ectopic expression of skeletal muscle RyR1 in B lymphocytes. Her group has developed the latter experimental approach, which is now exploited in several laboratories worldwide.

Matthew Trudeau
Matt received a BS degree in Biochemistry and Molecular Biology in 1992 and a PhD in Physiology in 1998 from the University of Wisconsin-Madison. His thesis work with Dr. Gail Robertson was on the physiology of the human ether-a-go-go related gene (hERG) family of potassium channels. He was a postdoctoral fellow with Dr. William N. Zagotta at the University of Washington and Howard Hughes Medical Institute in Seattle from 1998 to 2004 where he focused on cyclic nucleotide-gated (CNG) channels. In 2004 Matt joined the
University of Maryland School of Medicine in Baltimore where he is now Associate Professor in the Department of Physiology. At Maryland, he uses a multidisciplinary approach (including electrophysiological, biochemical, and optical methods) to investigate gating mechanisms, regulation, and physiological role of hERG channels and the properties of mutant hERG channels in cardiac arrhythmias.

David Warshaw
David is Professor and Chair of Molecular Physiology & Biophysics at the University of Vermont. He received his BS in Electrical Engineering from Rutgers University and PhD in Physiology & Biophysics from the University of Vermont. He did postdoctoral work with Fredric Fay at UMass Medical, studying single smooth muscle cell mechanics. His present research focuses on the structure and function of cardiac muscle contractile proteins as well as nonmuscle molecular motors using single molecule biophysical techniques such as laser trapping and total internal reflectance microscopy. Most recently, his laboratory has characterized the molecular mechanism by which myosin binding protein-C modulates cardiac contractility using an in vitro model system of cardiac muscle. He is Principal Investigator of a National Institutes of Health (NIH) Program Project Grant that focuses on the molecular basis of genetic heart failure. He is an Established Investigator and Fellow of the American Heart Association and a Fellow of the Biophysical Society. He has organized numerous International Conferences and Symposia including the Gordon Conference on “Muscle Contractile Proteins” (1999, 2002) and was the program co-chair of the 2009 Biophysical Society annual meeting. He has served on numerous NIH review panels and was a member of the Scientific Advisory Panel for the NIH Nanomedicine Initiative.

Bill is a Professor of Physiology and Biophysics at the University of Washington School of Medicine. He received his BS degree in Biophysics from the University of California, Davis, and his PhD degree in Neuroscience from Stanford University working with Richard W. Aldrich. He continued his studies at Stanford as a Postdoctoral Fellow working with Richard W. Aldrich and Denis Baylor. His work is focused on understanding the molecular mechanisms of the opening and closing conformational changes in ion channels, and how these conformation changes are regulated. He has studied a family of channels that is regulated by the direct binding of cyclic nucleotides, cAMP and cGMP. These channels play a fundamental role in the initial generation of an electrical signal in sensory receptors such as photoreceptors and olfactory receptors, and in the control of the pacemaker activity in cardiac and neuronal cells. To study the mechanisms of gating by cyclic nucleotides, his laboratory employs a variety of approaches including electrophysiology, site-directed mutagenesis, protein chemistry, site-specific fluorescent labeling, EPR, and x-ray crystallography. By the combination of these approaches, he hopes to gain new insights into the molecular mechanisms for channel function.