JGP Journal of General Physiology

EDITORIAL

JGP in 2023

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The start of the year is an appropriate time to reflect on *JGP*, particularly as the associate editors and I are beginning our second 3-year term. For many of us, life is now more normal than when I wrote my two previous start-of-year editorials. While still causing infection, COVID-19 has been tamed by effective vaccination programs in many countries. One can only hope that all will be in this position sooner rather than later. The development of COVID-19 vaccines has been quite properly lauded. It is important to remember and, in particular, remind, both politicians and the general public that the ability to respond to this and future pandemics relies on the vitality and therefore the funding of basic science which can then be translated into clinical benefit.

Undoubtedly, the major event which has cast a shadow over the world in 2022 is the war in Ukraine. *JGP* is proud of its authors, reviewers, and readers in that country and saddened by the conditions in which they and their compatriots now live. Membrane physiology in Ukraine was world-leading, contributing enormously to the knowledge of such important areas as the properties of calcium currents (Kostyuk et al., 1979) and ATP-sensitive currents (Krishtal et al., 1988). The history of Ukrainian electrophysiology has been summarized recently in the context of a discussion of what the response of the international community should be to this war (Petersen and Verkhratsky, 2022).

JGP has had another successful year, publishing novel, mechanistic, quantitative molecular and cellular physiology writ large and in Special Issues encompassing: Mechanotransduction by Membrane Proteins; and Excitation–Contraction Coupling in Cardiac, Skeletal and Smooth Muscle (Dirksen et al., 2022). Another Special Issue entitled Channels in Context mirrors the focus of the 2022 meeting of the Society of General Physiologists. We are grateful to the following for acting as Guest Editors: Bob Dirksen, Miriam Goodman, Elizabeth Haswell, Cathy Proenza, Karin Sipido, Matt Trudeau, and Valeria Vásquez.

Readers will be aware of discussions relating to Open Access. This was initially prompted by a concern that when a paper is published, it was only available (at least immediately) to those working in an institution with a subscription to the journal in which it is published. This is a consequence of the traditional model of financing of scientific journals by subscription charges in which the reader pays the costs. Given that the majority of research is funded by either public or charitable funds, there was a widespread feeling that it should be publicly available. Open Access publication makes the article immediately free to all readers and, instead, charges the authors.

Over time, the challenges of Open Access publishing have become apparent. Many business models shifted the burden to authors, simply moving us from one unfair system to another. The hybrid business model, in which authors can elect to pay a fee to make their articles immediately Open Access, has the danger of charging both the reader and author through a subscription and publishing fee.

To address these challenges, *JGP* has introduced three initiatives. The "No Fee or Low Fee" initiative offers free publishing to authors who do not require Open Access until after a 6-month embargo. Those wanting immediate Open Access can now pay a reduced $2,000 fee. The "Transparent Pricing" initiative ensures that *JGP* is not receiving subscription revenue from articles published as Open Access by adjusting the list subscription price based on the number of articles that require subscription access. The "Read-and-Publish" initiative allows free and unlimited Open Access publishing to corresponding authors based at subscribing institutions who have entered into an agreement with *JGP*'s publisher, Rockefeller University Press.

Finally, the success of *JGP* depends on the service of all of you who give up your time to referee manuscripts. Many of our most hard-working reviewers are members of the Editorial Advisory Board (EAB). We have appointed three new members: László Csanády, Ivy E. Dick, and Theanne Griffith. Brief descriptions of their backgrounds can be found below. I would also like to thank those members of the EAB who are stepping down at the end of their terms: Douglas Tobias, Rikard Blunck, Robert French, Jochen Hub, Timothy Jegla, Régis Pomès, Timothy Ryan, Avner Schlessinger, Sarah Veatch, Hailin Zhang, and Jie Zheng. It is particularly fitting to make special mention of and thank the following members who have served for periods of between 17 and 35 years: Richard Aldrich, Francisco Bezanilla, Michael Cahalan, Michael Fill, H. Criss Hartzell, Anna Menini, and Angus Nairn.

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New EAB members

László Csanády

László Csanády is a Professor in the Department of Biochemistry at Semmelweis University, Budapest, Hungary. After obtaining an MD at Semmelweis, he joined the laboratory of David Gadsby at Rockefeller University, where he earned his PhD studying structure and function of the CFTR anion channel. He conducted postdoctoral research at Semmelweis University under the guidance of Veronika Adam-Vizi, focusing on gating of native Ca^{2+}- and voltage-activated nonselective cation channels in brain endothelial cells. His group, established with the help of an HHMI International Early Career Scientist Award, combines electrophysiology and biochemical approaches to investigate structure and mechanism of two ion channel enzymes, CFTR and the nonselective cation channel TRPM2. His group has contributed to the understanding of CFTR channel gating by ATP binding and hydrolysis, CFTR channel activation by protein kinase A, and the molecular pathology of some cystic fibrosis–associated CFTR mutations. The group has also contributed insight into TRPM2 channel activation through ADP ribose and Ca^{2+}, and discovered that the evolutionary appearance of TRPM2 pore-inactivation was correlated with the loss of its ADPR-hydrolase activity. They have also dissected the biophysical mechanism that allows TRPM2 to function as an exquisitely sensitive deep-brain temperature-sensor. Photo courtesy of László Csanády.

Ivy E. Dick

Ivy E. Dick is an Assistant Professor in the Department of Physiology at the University of Maryland School of Medicine. She began her career under the direction of Drs. Charles Cohen and Owen McManus at Merck Research Labs, and subsequently earned her doctoral degree under the direction of Dr. David Yue in the Biomedical Engineering department at Johns Hopkins University, where she studied the mechanisms underlying calmodulin regulation of voltage-gated calcium channels. Dr. Dick's current research focuses on gaining mechanistic understanding of the regulatory processes of voltage-gated Ca^{2+} channels, and applying those findings to gain new insight into the pathogenesis and treatment options for Ca^{2+} channelopathies and related diseases. Her recent work focused on elucidating the impact of mutations in L-type Ca^{2+} channels, which underlie severe cardiac and neuronal disorders such as Timothy syndrome (TS), a severe multisystem disorder featuring both cardiac and neurological phenotypes. By studying the biophysical effects of multiple TS mutations on $Ca_V1.2$, Dr. Dick's recent publication in *JGP* demonstrated the diverse impact mutations may have on channel gating and provided mechanistic understanding of how alterations in channel function lead to distinct patient phenotypes. Moreover, Dr. Dick continues to probe the impact of these mutations on therapy, offering new insights into treatment options for channelopathy patients. Photo courtesy of Ivy E. Dick.

Theanne Griffith

Dr. Theanne Griffith received her undergraduate degrees in neuroscience and Spanish from Smith College and earned her doctorate in neuroscience from Northwestern University. As a graduate student, she combined electrophysiology and molecular biology to investigate the structure and function relationship between ionotropic glutamate receptors and their auxiliary subunits. This work was the first study to identify regions within kainate receptors targeted for modulation by auxiliary subunits. As a postdoctoral fellow at Columbia University, Dr. Griffith harnessed her knowledge of ion channel function to investigate the molecular mechanisms governing excitability of mammalian sensory neurons. This project found an unexpected role for the voltage-gated sodium channel, $Na_V1.1$, in mediating action potential firing in a subpopulation of cold-sensing neurons. Dr. Griffith is currently an Assistant Professor in the Department of Physiology and Membrane Biology at The University of California (UC), Davis, where her lab investigates the cellular and molecular mechanisms of somatosensory transmission in health and disease, with current projects focusing on voltage-gated sodium channels in proprioception and pain. Her lab uses a combination of electrophysiology, transgenic mouse models, behavior, imaging, and molecular profiling. Dr. Griffith is a UC Davis Center for Advancing Multicultural Perspectives on Science (CAMPOS) Scholar, and a UC Davis Public Scholarship Faculty Fellow. In addition to her research, Dr. Griffith is a children's book author of the science adventure chapter book series, The Magnificent Makers, which is published by Random House Children's Books. She also co-writes the non-fiction science series, Ada Twist, Scientist: The Why Files, which accompanies the Netflix show of the same name. Photo courtesy of Samantha Jovan Photography.

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