BIOGRAPHICAL SKETCH

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NAME:: Venkata Shiva Mandala

eRA COMMONS USER NAME (credential, e.g., agency login): VMANDALA

POSITION TITLE: Postdoctoral Fellow

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Start Date MM/YYYY	Completion Date MM/YYYY	FIELD OF STUDY
Oberlin College	B.A.	08/2011	05/2015	Biochemistry and Chemistry
Massachusetts Institute of Technology	Ph.D.	08/2015	10/2020	Biological and Physical Chemistry
Rockefeller University	n/a (Postdoc)	11/2020	Present	Molecular Neurobiology and Biophysics

A. Personal Statement

My research interests lie in the structure and function of membrane-embedded ion channels and transporters. My long-term career goal is to pursue these interests as a principal investigator at a major research institution, using biophysical tools including cryo-electron microscopy (cryo-EM), nuclear magnetic resonance (NMR) spectroscopy, and electrophysiology. My current research interests involve solving the structures of voltage-gated potassium (K_V) channels in lipid bilayers to gain insight into the mechanism of voltage regulation and voltage-sensor toxins. Investigation of these phenomena necessitates a proper membrane environment rather than detergent micelles used to typically study the structures of membrane proteins. Studying proteins in lipid bilayers by cryo-EM itself remains a significant challenge. My graduate work using NMR involved extensive work with membrane proteins in lipid bilayers, giving me the requisite background for this work. My postdoctoral work should make possible routine cryo-EM structure determination of membrane proteins and complexes in lipid bilayers with specific compositions. More importantly, the experiments planned should provide key insight into the voltage regulation mechanism of K_V channels and the basis of voltage-sensor toxin binding and channel inhibition. The cryo-EM experiments will be coupled with electrophysiology studies of purified proteins in black lipid membranes for a mechanistic understanding. My proposal for studying membrane protein localization in lipid bilayers was awarded a three-year HHMI-Jane Coffin Childs Memorial Fund Fellowship.

My application for electron microscope access through the National Center for CryoEM Access and Training (NCCAT) will contribute greatly to the completion of my postdoctoral work, specifically to the structure determination of K_V channels with different voltage sensor configurations, and with specific toxins bound. My previous research experience with membrane protein purification and reconstitution, lipid biochemistry, quantitative NMR, scanning and transmission EM, and biochemical assays will be crucial to the planned work. The skills I acquired previously are highly complementary to those present in my sponsor's group. Dr. Roderick MacKinnon has carried out seminal work on voltage gated potassium channels and pore-blocking spider toxins, and has extensive experience with membrane protein structural biology and electrophysiology.

B. Positions, Scientific Appointments and Honors

POSITION	START DATE MM/YYY	END DATE MM/YY	INSTITUTION	SUPERVISOR
Undergraduate research assistant	08/2011	06/2014	Oberlin College	Dr. Michael Moore
Undergraduate research assistant	05/2014	05/2015	Oberlin College	Dr. Manish Mehta
Undergraduate research assistant	06/2013	05/2014	Oberlin College	Dr. Catherine Oertel
Graduate research assistant	11/2015	10/2020	Massachusetts Institute of Technology	Dr. Mei Hong
Postdoctoral Fellow	11/2020	Present	Rockefeller University	Dr. Roderick MacKinnon

Awards and Honors

HHMI Fellow of The Jane Coffin Childs Memorial Fund, 2021.
Lester Wolfe Fellowship, Massachusetts Institute of Technology, 2019.
Poster Award, Protein Society, 2019.
Finn Wold Travel Award, Protein Society, 2019.
Harry N. Holmes Award for Excellence in Chemistry, Oberlin College, 2015.
Member of Phi Beta Kappa, Oberlin College, 2015.
Dean's List, Oberlin College, 2012, 2013 & 2014.
Kathryn W. Davis Projects for Peace Fellowship, Kathryn W. Davis Foundation, 2014.
Member of Sigma Xi, Oberlin College, 2014.
Civic Engagement Entrepreneur Award, Bonner Center for Service and Learning, 2014.
Venture Development Prize, LaunchU Entrepreneurship Challenge, 2014.
Ignition Grant, Creativity and Leadership Fund, Oberlin College, 2013 & 2014.
Frank Fanning Jewett Award for Excellence in Chemistry, Oberlin College, 2013.
DST-INSPIRE Scholarship for Higher Education, Government of India, 2011.
National Science Exhibition Winner, Government of India, 2010.

C. Contributions to Science

I. Undergraduate Research in Biology and Chemistry

I worked in Professor Manish A. Mehta's lab at Oberlin College for two years studying mechanisms of crystal formation in co-crystals of active pharmaceutical ingredients (API) and API mimics. Such co-crystals have desirable pharmacological properties and thus are of significant interest to the pharmaceutical industry. Powder X-ray diffraction (XRD) and solid-state nuclear magnetic resonance (SSNMR) were used to investigate: 1) factors affecting spontaneous co-crystallization, and 2) high-resolution structural characterization of co-crystals. In the first study, we discovered that relative humidity has a dramatic effect on the rate of spontaneous co-crystallization of caffeine and malonic acid. We also recorded high field SSNMR spectra of three co-crystalline systems. *In-situ*, real time monitoring of co-crystal formation using SSNMR saw no evidence of amorphous or vapor phase intermediates, although atomic-level changes in the structure were apparent.[1] The formation of co-crystal product during spontaneous co-crystallization was determined to be a second-order or pseudo second-order kinetic process, which provided insight into the mechanism of mass transfer during co-crystallization. My second project was done as part of my honors thesis, and resulted in two high resolution neutron diffraction (ND) structures of piracetam co-crystallized with *L*-tartaric acid and *p*-hydroxybenzoic acid. These ND structures were compared to literature XRD structures, and revealed systematic errors in hydrogen bonding parameters between the two structures. ND showed that hydrogen bonds in these co-crystals are consistently stronger than depicted by the XRD structures. Evidence of hydrogen-bonding was also found by correlating structural features with ^{13}C chemical shifts obtained through SSNMR.

I also worked in Professor Michael J. Moore's lab at Oberlin College studying phylogenetic relationships in angiosperms using complete plastid genomes (plastomes) obtained through next generation sequencing (NGS) data. I developed a bioinformatics pipeline in Prof. Moore's laboratory for assembling plastomes from the NGS data, which was applied to assemble nearly-complete to complete plastomes for 24 angiosperms in collaboration with Gregory Stull, a graduate student in the Soltis lab at the University of Florida, Gainesville. Sequencing information for all 24 species was obtained in a single Illumina GAIIx lane, using a novel targeted enrichment strategy developed to enable massively parallel sequencing of plastomes.[2] This enrichment strategy is essential because plastid DNA comprises only ~0.5-15% of all DNA in plant cells, which means direct sequencing of cellular DNA leads to ≥85% of unusable information. Unique barcodes were used for each sample to bin the NGS reads, and ~55,000 hybridization baits allowed for specific targeting of plastid DNA. Mean sequencing depth across all the samples was >700x, with highly enriched protein coding regions.

1. **Mandala, V. S.**; Loewus, S. J.; Mehta, M. A., Monitoring Cocrystal Formation via In Situ Solid-State NMR. *J Phys Chem Lett,* **2014,** 5 (19), 3340-3344.
2. Stull, G. W.; Moore, M. J.; **Mandala, V. S.**; Douglas, N. A.; Kates, H. R.; Qi, X. S.; Brockington, S. F.; Soltis, P. S.; Soltis, D. E.; Gitzendanner, M. A., A Targeted Enrichment Strategy for Massively Parallel Sequencing of Angiosperm Plastid Genomes. *Applications in Plant Sciences,* **2013**, 1 (2).

III. Graduate Research in Biological and Physical Chemistry

I joined Professor Mei Hong's group at the Massachusetts Institute of Technology (MIT) in November 2015. My graduate work used solid-state nuclear magnetic resonance (SSNMR) spectroscopy to understand the structure and functional mechanism of function of the tetrameric membrane-embedded matrix-2 protein (M2) from the influenza virus. M2 is an essential viral protein that conducts protons in the endosomes of infected host cells and induces membrane curvature to facilitate virus budding. The proton channel activity is mediated by a transmembrane domain (TM) that is targeted by the amantadine class of antivirals to inhibit viral replication. The TM contains a proton-selecting His-xxx-Trp motif conserved in the otherwise disparate M2 from influenza A (AM2) and influenza B (BM2) strains.

I first explored the mechanisms of asymmetry, transport and water dynamics in proton conduction by influenza A M2 (AM2), using a gating-deficient Trp41Phe mutant.[1] This bidirectionally-conducting mutant preserves the α-helical conformation and tetrameric assembly of wild-type AM2, but differs significantly in the protonation equilibria of the His37 tetrad responsible for proton conduction. The four tetrad pK_a's are more clustered compared to the wild-type, indicating that the Trp-to-Phe mutation disrupts the rates of protonation and deprotonation of the His tetrad from the C-terminal side. N-terminal pore occlusion by amantadine of the mutant preserved charged His at low pH, demonstrating that the mutation permits C-terminal access to the tetrad. Thus, asymmetric conduction in wild-type M2 is due to Trp41 inhibition of C-terminal acid activation by His37. Interestingly, for most residues labeled in this work, we observed two clear sets of chemical shifts.

Through further research into the putative dual conformational states, I demonstrated that the two sets of chemical shifts correspond to open and closed tetramers, whose relative populations depend upon pH through the charge state of the His37 tetrad.[2] The two conformations of tetramers were found to interconvert at a rate of ca. 400 s^{-1}, concomitant with the proton conduction rate, thus suggesting that this motion is the rate-limiting step of proton conduction and implicating a transporter-like conduction mechanism. Measurements of protein ^{13}C-detected water ^{1}H T_2 relaxation showed that channel water relaxes ca. 10-fold faster than bulk and membrane-associated water, undergoing nanosecond motion independent of pH.

After working on influenza A M2, I shifted my focus to determine the atomic-resolution structure of open and closed states of influenza B M2 (BM2). I recombinantly produced uniformly ^{13}C,^{15}N-labeled $BM2_{1-51}$ at high pH and assigned the chemical shifts of most residues. Secondary shifts indicated that residues 6-28 form a well-ordered α-helix spanning the membrane, while residues 1-5 and 29-35 show β-sheet conformation. The expected periodicity of α-helical amide proton chemical shifts of coiled-coils, and ^{15}N chemical shift differences between micelle- and bilayer-bound BM2 motivated further determination of the three-dimensional structure of BM2 in membranes.[3] I then completed this endeavor to determine the atomic-resolution structures of BM2 in the closed and open states at ca. 3 Å resolution.[4] The recombinant construct was optimized to remove a non-native affinity tag. Chemical shift assignments were extended to include low pH, and little shift perturbations was seen

between high and low pH barring the titratable His residues. Two key changes in the quaternary structure were determined: at low pH compared to high pH, the α-helices become ca. 6° more tilted relative to the lipid bilayer, and the average pore diameter enlarges by 2.1 Å. In contrast to the two-state transporter-like conformational interconversion observed in AM2, BM2 undergoes a subtle channel-like scissors opening motion upon activation at low pH, permitting more efficient but partially bidirectional proton conduction.

1. **Mandala, V. S.**; Liao, S. Y.; Kwon, B.; Hong, M. Structural Basis for Asymmetric Conductance of the Influenza M2 Proton Channel Investigated by Solid-State NMR Spectroscopy. *J. Mol. Biol.*, **2017**, 429 (14), 2192-2210.
2. **Mandala, V. S.**; Gelenter, M.D.; Hong, M. Transport-Relevant Protein Conformational Dynamics and Water Dynamics on Multiple Timescales in an Archetypal Proton Channel - Insights from Solid-State NMR. *J. Am. Chem. Soc.*, **2018**. 140 (4). 1514-1524.
3. **Mandala, V. S.**; Liao, S. Y.; Gelenter, M.D.; Hong, M. The Transmembrane Conformation of the Influenza B Virus M2 Protein in Lipid Bilayers. *Sci. Rep.* **2019**. 3725. 1-13.
4. **Mandala, V. S.**; Loftis, A.R.; Shcherbakov, A.A.; Pentelute, B.L. ; Hong, M. Atomic Structures of Closed and Open Influenza B M2 Proton Channel Reveal the Conduction Mechanism. *Nat. Struc. Mol. Biol.* **2020**. 27. 160-167.

A full list of peer-reviewed publications, including significant other contributions in the field of solid-state NMR spectroscopy with applications to biological problems, can be found at:
https://scholar.google.com/citations?user=2fTrEY4AAAAJ&hl=en

D. Scholastic Performance

Massachusetts Institute of Technology, Graduate School

YEAR	COURSE TITLE	GRADE
2015	Advanced Biological Chemistry	A
2016	Intro Quantum Mechanics I	A
2016	Biological Chemistry II	A
2016	Seminar in Biological Chemistry	A
2016	Intro Quantum Mechanics II	A
2017	Topics in Biophyscs & Physical Biology	P

A=exceptionally good performance, B=good performance, C=adequate performance, D=minimally acceptable performance, P=pass in case of no letter grade, F=failed.

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NAME: MacKinnon, Roderick

eRA COMMONS USER NAME (credential, e.g., agency login): MACKINNONR

POSITION TITLE: Professor of Molecular Neurobiology and Biophysics

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Brandeis University, Waltham, MA	B.A.	06/1978	Biochemistry
Tufts University School of Medicine, Boston, MA	M.D.	06/1982	Medicine
Beth Israel Hospital, Harvard Univ., Boston, MA	Postdoctoral Fellowship (NIH)	09/1985-09/1986	Medicine
Brandeis University, Waltham, MA	Postdoctoral Fellowship (NIH)	09/1986-09/1989	Biochemistry

A. Personal Statement

I am a professor at Rockefeller University in the Laboratory of Molecular Neurobiology and Biophysics and an investigator with the Howard Hughes Medical Institute.

B. Positions and Honors

Positions and Employment

1982-85 Medical House Officer, Internal Medicine, Beth Israel Hospital/Harvard Medical School, Boston, MA
1985-86 NIH Postdoctoral Fellow, Dept. of Medicine, Beth Israel Hospital, Harvard University, Boston, MA
1986-89 NIH Postdoctoral, Dept. of Biochemistry, Brandeis University, Waltham, MA
1989-91 Assistant Professor, Dept. of Cellular and Molecular Physiology, Harvard Med. School, Boston, MA
1991-92 Assistant Professor, Department of Neurobiology, Harvard Medical School, Boston, MA
1992-95 Associate Professor, Department of Neurobiology, Harvard Medical School, Boston, MA
1995-96 Professor, Department of Neurobiology, Harvard Medical School, Boston, MA
1996- Professor, Laboratory of Mol. Neurobiology and Biophysics, Rockefeller University, New York, NY
1997- Investigator, Howard Hughes Medical Institute

Other Experience and Professional Memberships

2000 Member, The U.S. National Academy of Sciences
2005 Member, The American Philosophical Society
2007 Foreign Member, The Royal Netherlands Academy of Arts and Sciences

Honors

1978 High Honors in Biochemistry, Brandeis University
1982 Alpha Omega Alpha Medical Honors Society, Tufts University School of Medicine
1992 PEW Scholar in the Biomedical Sciences

1992 McKnight Scholars Award
1995 Biophysical Society Young Investigator Award
1997 McKnight Investigator Award
1998 W. Alden Spencer Award, Columbia University
1998 AAAS Newcomb Cleveland Prize
1999 Albert Lasker Basic Medical Research Award
2000 Lewis S. Rosenstiel Award for Distinguished Work in Basic Medical Research
2000 Hodgkin-Huxley-Katz Prize
2000 Alexander M. Cruickshank Award
2000 Membership to the U.S. National Academy of Sciences
2001 Gairdner Foundation International Award
2001 George Harvey Miller Distinguished Lecturer Award, University of Illinois, Chicago
2002 Perl-UNC Neuroscience Prize
2002 The Degree of Doctor of Science, Honoris Causa – Tufts University
2003 Fritz Lipmann Memorial Lectureship Award
2003 Nobel Prize in Chemistry
2003 Louisa Gross Horwitz Prize
2003 Scientific American Research Leader of the Year
2004 Max Tishler Prize – Harvard University
2004 National Lecturer, Biophysical Society Annual Meeting
2004 Bijvoet Medal for Outstanding Research – The Netherlands
2005 The Hans Neurath Award-Protein Society
2005 The Degree of Doctor of Science, Honoris Causa – Brandeis University
2005 Membership to the American Philosophical Society
2006 Rolf Sammet Honorary Lectureship –J.W. Goethe University, Frankfurt, Germany
2006 The Degree of Doctor of Science, Honoris Causa – Pohang Univ. of Science & Technology, Korea
2007 Wolfgang Pauli Lectureship - ETH, Zurich, Switzerland
2007 Foreign Membership to the Royal Netherlands Academy of Arts and Sciences
2007 The Degree of Doctor of Science, Honoris Causa – Rene Descartes University, France
2008 Pauling Legacy Award
2013 Max Tishler Lecturer Award, Tufts University
2015 Kenneth S. Cole Award, Biophysical Society

C. Contributions to Science

1. I began my scientific career as an electrophysiologist. The first main question I focused on was how do K+ channels select for K+ over Na+? Two things intrigued me about K+ selectivity. First, K+ is larger than Na+. How can the larger ion conduct and the smaller ion not? Second, high selectivity, it seemed to me, would ordinarily be associated with high affinity and yet K+ diffuses through the channel at near diffusion-limited rates (i.e. as fast as physically possible). Thus, the K+ channel was a paradox in my mind. Using electrophysiological methods I could observe this remarkable behavior and using mutagenesis I could even identify particular amino acids that were responsible for it. But it soon became clear to me that in order to answer the question I would have to see what the channel looked like. But no one had ever determined the atomic structure of a selective ion channel, and moreover I was not trained in that sort of work. I decided to do it anyway, learned x-ray crystallography, and solved the structure of a K+ channel. The structure was very revealing because it contained a selectivity filter with structural and chemical properties that explained selectivity for K+ over Na+ and simultaneous high conduction rates. This was a good advance for our understanding of electrical impulse formation because since the famous work of Hodgkin and Huxley in 1952 scientists had wondered what does an ion channel really look like.

    a) Doyle, D.A., Cabral, J.M., Pfuetzner, R.A., Kuo, A., Gulbis, J.M., Cohen, S.L., Chait, B.T., MacKinnon, R. (1998). The structure of the potassium channel: molecular basis of K+ conduction and selectivity. *Science,* 280(5360), 69-77. PubMed PMID: 9525859.
    b) Zhou, M., Morais-Cabral, J., Mann, S., MacKinnon, R. (2001). Potassium channel receptor site for the inactivation gate and quaternary amine inhibitors. *Nature*, 411(6838), 657-661. PubMed PMID: 11395760.

2. If the first major question driving my scientific studies is how do ion channels conduct ions selectively, the second is how do ion channels turn conduction off and on in a process known as gating? My laboratory determined the first voltage-gated ion channel structure and the first ligand-gated K+ channel structure. Our studies of voltage-dependent gating led us to determine the first eukaryotic membrane protein structure from recombinant expression.

    a) Jiang, Y., Lee, A., Chen, J., Ruta, V., Cadene, M., Chait, B.T., MacKinnon, R. (2003). X-ray structure of a voltage-dependent K+ channel. *Nature*, *423*(6935), 33-41. PubMed PMID: 12721618.
    b) Jiang, Y., Lee, A., Chen, J., Cadene, M., Chait, B.T., MacKinnon, R. (2002). Crystal structure and mechanism of a calcium-gated potassium channel. *Nature*, *417*(6888), 515-522. PubMed PMID: 12037559.
    c) Long, S.B., Campbell, E.B., MacKinnon, R. (2005). Voltage sensor of Kv1.2: structural basis of electromechanical coupling. *Science, 309*(5736), 903-908. PubMed PMID: 16002579.
    d) Long, S.B., Tao X., Campbell, E.B., MacKinnon, R. (2007). Atomic structure of a voltage-dependent K+ channel in a lipid membrane-like environment. *Nature*, *450*(7168), 376-382. PubMed PMID: 18004376.

3. The K+ channel family is extraordinarily diverse. In humans there are more than 70 K+ channels. While these share the highly-conserved selectivity filter observed in the first K+ channel structure KcsA, they are architecturally distinct. As function follows form, these distinct K+ channels have evolved under the control of different stimuli: membrane voltage, intracellular Ca2+, G proteins and mechanical forces. To better understand this marvelous family of proteins we have determined structures and studied the behaviors of far-ranging members of the K+ channel family tree.

    a) Brohawn, S.G., Campbell, E.B., MacKinnon, R. (2014). Physical mechanism for gating and mechanosensitivity of the human TRAAK K+ channel. *Nature*, 516 (7529), 126-30. PubMed Central PMCID: 468236. HHMIMSID: HHMIMS639323.
    b) Tao, X., Hite, R., MacKinnon, R. (2017). Cryo-EM structure of the open high-conductance Ca^{2+}-activated K^+ channel**.** *Nature*, *541*(7635*)*, 46-51. PubMed Central PMCID: 5500982.
    c) Sun, Ji, MacKinnon, R. (2017). Cryo-EM structure of a KCNQ1/CaM complex reveals insights into congenital long QT syndrome. *Cell*, *169*(6), 1042-1050. PubMed Central PMCID: 5562354.
    d) Lee, Chia-Hsueh, MacKinnon, R. (2018). Activation mechanism of a human SK/calmodulin channel complex elucidated by cryo-EM structures. *Science* 360, 508-513. PubMed Central PMCID: 6241251.

4. G protein coupled receptor signaling controls heart rate and neuronal activity through the activity of K+ channels known as G protein gated inward rectifier K+ (GIRK) channels. We have discovered how the G proteins regulate GIRK activity through atomic structural and functional studies.

    a) Whorton, M.R., MacKinnon, R. (2011). Crystal structure of the mammalian GIRK2 K+ channel and gating regulation by G proteins, PIP(2), and sodium. *Cell*, 147(1), 199-208. PubMed Central PMCID: 3243363.
    b) Whorton, M.R., MacKinnon, R. (2013). X-ray structure of the mammalian GIRK2-βγ G-protein complex. *Nature*, published online June 5. PubMed Central PMCID: 4654628.
    c) Wang, W., Touhara, K.K., Weir, K., Bean, B.P., MacKinnon, R. (2016). Cooperative regulation by G proteins and Na+ of neuronal GIRK2 K+ channels. *eLife*, April 2016:*5*:e15751. PubMed Central PMCID: 4866826.
    d) Touhara, K, MacKinnon, R. (2018) Molecular basis of signaling specificity between GIRK channels and GPCRs. *eLife,* Dec. 2018:7:e42908. PubMed Central PMCID: 6335053.

5. Mechanosensitive ion channels allow living organisms to sense mechanical forces. These underlie touch sensation and other cellular processes. We have proposed the membrane dome model of mechanosensitive gating in Piezo1, a mechanosensitive ion channel.

    a) Guo, Y.R., MacKinnon, R. (2017). Structure-based membrane dome mechanism for Piezo mechanosensitivity. *eLife*, Dec. 2017, el.33660. PubMed Central PMCID: 5788504.

b) Haselwandter, Christoph, MacKinnon, R. (2018). Piezo's membrane footprint and its contribution to mechanosensitivity. *eLife*, Nov. 2018:7:e41968. PubMed Central PMCID: 6317911.
c) Del Marmol, J., Touhara, K.K., Croft, G., MacKinnon, R. (2018). Piezo1 forms a slowly-inactivating mechanosensory channel in mouse embryonic stem cells. ***eLife,*** Aug. 2018; 7:e33149. PubMed Central PMCID: 6128688.
d) Lin, Y.C., Guo, Y.R., Mayagi, A., Levering, J., MacKinnon, R., Scheuring, S. (2019). Force-induced conformational changes in Piezo1. ***Nature***, *573*, 230-244. PMID: 31435018. PMCID: in process.

Complete list of published work available publicly:

https://www.ncbi.nlm.nih.gov/sites/myncbi/1ZOcxVvzmaekv/bibliography/50241239/public/?sort=date&direction=ascending

or

https://www.ncbi.nlm.nih.gov/myncbi/1ZOcxVvzmaekv/bibliography/public/

or
http://lab.rockefeller.edu/mackinnon/publications

D. Research Support

Ongoing Research Support

R01 GM043949 MacKinnon (PI) 04/01/1990 - 11/30/2024
NIH/NIGMS
"Mechanisms of K+ channel gating"
The major goal of this project is to understand the structural basis of voltage-dependent channel gating.

HHMI MacKinnon (PI) 1997 – Present
(a) "The structural basis of ion selectivity in channels"
The major goal of this project is to understand how channels select K^+ and Cl^- ions and conduct them rapidly across the membrane.
(b) "The structural basis of gating in K^+ channels"
The major goal of this project is to understand how Ca^{2+}, G-protein subunits, and cyclic nucleotides open channels. Funding also contributes to the voltage-dependent channel studies by providing equipment and antibodies for protein crystallization.
(c) "Development of new methods for the expression and crystallization of membrane proteins"
The major goal of this project is to understand the structural basis and function of CLC chloride channels.

Completed Research Support

Robertson Therapeutic Development Fund MacKinnon (PI) 9/1/2015 – 12/31/2017
Project description: hERG channel – cell-free high throughput screening method development.