Nipavan Chiamvimonvat, M.D.

Clinical Interests

I am a physician scientist and have dedicated my career in treating patients with cardiac arrhythmia disorders with my early training in cardiac electrophysiology at the University of Calgary, Canada. Critical knowledge gaps in the treatment of cardiac arrhythmias compelled me to pursue fundamental and translational research in cardiac arrhythmias that began with my postdoctoral training at Johns Hopkins University. The combination of research and clinical practice provides me with a unique perspective in cardiac arrhythmia disorders. My experiences in patient care both drive and inform my fundamental, translational and clinical research. In addition, by bridging these two different worlds, I have the opportunities to mentor young clinicians and basic scientists to understand the importance of cross-disciplinary interactions in their work. By uniting these separate approaches, our endeavors lay the foundation for translational and clinical investigations that may someday lead to advanced new therapies.

Research/Academic Interests

Our laboratory was the first to identify several isoforms of small conductance Ca2+-activated K+ channels (SK channels) that underlie Ca2+-activated K+ current in human and mouse atrial myocytes. Even though the roles of these channels have been extensively investigated in neurons, their roles in the heart are only beginning to be recognized. We demonstrated that the channels are highly expressed in atrial compared to ventricular tissues. Null mutation of the SK2 channels results in atrial arrhythmias and AV node dysfunction. Subsequently, our findings have been supported by several different laboratories. Even more relevant to translational medicine is the finding from recent studies using genome-wide association analysis that has provided a genetic link between SK channel polymorphisms and lone atrial fibrillation (AF) in humans.

Our studies have unmasked a functional crosstalk between Cav1.3 and SK channels. We have obtained new evidence to demonstrate that the expression of several interacting proteins, -actinin2, myosin light chain 2, and filamin A, are required for the proper membrane localization of SK channels. Even more exciting is the finding that an increase in intracellular Ca2+, as seen in AF, results in an increase in membrane localization of SK channels which would be predicted to lead to a decrease in effective refractory periods, an increase in functional reentry, and the maintenance of atrial arrhythmias. Our efforts thus far have paid off. Recent studies from several laboratories have provided additional support for the importance of SK channels. Indeed, SK channels may represent a novel drug target for AF. Insights into the molecular interactions between these new classes of cardiac ion channels are likely to be highly significant clinically. Thus, our studies have provided unique insights regarding the roles of these newly described channels in the heart.

Our laboratory was the first to demonstrate beneficial effects of a novel class of soluble epoxide hydrolase (sEH) inhibitors in cardiac hypertrophy, failure and arrhythmias. This work is a

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collaborative effort with Dr. Hammock at UC Davis. Treatment with sEH inhibitors (sEHIs) results in the prevention of ventricular myocyte hypertrophy and electrical remodeling in pressure overload and myocardial infarction models. We further demonstrate that treatment with sEHIs prevents cardiac fibroblast (CF) proliferation and fibrosis. These results are exciting because they demonstrate broader salutary effects in cardiac remodeling (not limited to myocyte hypertrophy alone) of this novel class of compounds. sEHIs have been shown to potently block the catalytic conversion of epoxyeicosatrienoic acids (EETs) to the corresponding dihydroxyeicosatrienoic acids (DHETs). EETs are products of cytochrome P450 epoxygenases that are known to have vasodilatory as well as anti-inflammatory properties. Since atrial fibrosis has been shown to play critical roles in atrial fibrillation, we are actively testing our hypothesis that treatment with sEHIs will be beneficial in the prevention of atrial fibrillation by reducing 1) atrial fibrosis, and 2) electrical remodeling in atrial myocytes.

Title Professor, Roger Tatarian Endowed Professor of Cardiovascular Medicine
Associate Chief for Research, Division of Cardiovascular Medicine
Co-Director, UC Davis Cardiovascular Research Institute (CVRI)
Staff Cardiologist at VA Medical Center

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Education M.D., University of Toronto, Toronto, Ontario, Canada 1984
Biology, University of Toronto, Toronto, Ontario, Canada 1980

Internships Internal Medicine, University of Toronto, Toronto, Ontario, Canada 1984-1985

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Residency
Internal Medicine, University of Toronto, Toronto, Ontario, Canada 1985-1987

Fellowships
Clinical Cardiac Electrophysiology, University of Calgary, Calgary, Alberta, Canada 1989-1991
Cardiology, University of Western Ontario, Toronto, Ontario, Canada 1987-1989

Board Certifications
American Board of Internal Medicine
American Board of Internal Medicine, Cardiovascular Medicine
Diplomate, National Board of Medical Examiners
Fellow, Royal College of Physicians and Surgeons of Canada, Cardiology
Fellow, Royal College of Physicians and Surgeons of Canada, Internal Medicine
Licentiate, Medical Council of Canada

Professional Memberships
American Association for the Advancement of Science
American Heart Association
Biophysical Society
Heart Rhythm Society
Society of General Physiologists

Honors and Awards
Career Achievement Award, Department of Internal Medicine, UC Davis, 2017
Dean's Excellence in Mentoring Award, UC Davis, 2011
Medical Research Council of Canada Research Fellowship, 1993, 1994, 1995, 1996, 1997
Runner up for the 1996 Young Investigator Award from North American Society for Pacing and Electrophysiology (NASPE), 1996
Heart and Stroke Foundation of Canada Research Fellowship, 1991, 1992, 1993

Select Recent Publications
To view a detailed list of Dr. Chiamvimonvat's publications, please click here.

Ledford HA, Ren L, Thai PN, Park S, Timofeyev V, Sirish P, Xu W, Emigh AM, Priest JR, Perez MV, Ashley EA, Yarov-Yarovoy V, Yamoah EN, Zhang XD, Chiamvimonvat N. Disruption of protein quality control of the human ether-à-go-go related gene K+ channel results in profound long QT syndrome. Heart Rhythm. 2022 Feb;19(2):281-292. doi:10.1016/j.hrthm.2021.10.005. Epub 2021 Oct 9. PMID:34634443.

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Yechikov S, Kao HKJ, Chang CW, Pretto D, Zhang XD, Sun YH, Smithers R, Sirish P, Nolta JA, Chan JW, Chiamvimonvat N, Lieu DK. NODAL inhibition promotes differentiation of pacemaker-

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like cardiomyocytes from human induced pluripotent stem cells. Stem Cell Res. 2020 Dec;49: 102043. doi:10.1016/j.scr.2020.102043. Epub 2020 Oct 12. PMID:33128951.

Campbell KT, Silva EA. Biomaterial Based Strategies for Engineering New Lymphatic Vasculature. Adv Healthc Mater. 2020 Sep;9(18):e2000895. doi:10.1002/adhm.202000895. Epub 2020 Jul 30. PMID:32734721.

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