CURRICULUM VITAE

Luis Fernando Santana
Professor and Chair of the Department of Physiology and Membrane Biology
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Education

Institution	*Degree*	*Area of specialization*	*Dates*
University of Puerto Rico	B.S.	Marine Biology	1987-1991
University of Maryland	Ph.D.	Physiology and Biophysics	1992-1996

Postgraduate training

Institution	*Mentor*	*Area of specialization*	*Dates*
Medical Biotechnology Center, University of Maryland	W. J. Lederer	Biophysics	1996-1997
The University of Vermont	Mark T. Nelson	Vascular Physiology	1997-1999

Faculty and leadership positions held

Institution	*Position*	*Dates*
Institute of Neurobiology, University of Puerto Rico	Assistant Professor	1999-2001
Department of Physiology and Biophysics, University of Washington	Assistant Professor	2001-2006
Department of Physiology and Biophysics, University of Washington	Associate Professor	2006-2010
Department of Physiology and Biophysics, University of California, Davis	Professor	2010-2015
Department of Physiology and Membrane Biology	Professor and Chair	2015-present
Department of Biochemistry and Molecular Medicine	Interim Chair	2021-present
Department of Medical Microbiology and Immunology	Interim Chair	2022
UC Davis School of Medicine	Interim Vice Dean for Basic Sciences	2022-present

Honors

NIH-MARC predoctoral fellowship (1989-1991)
NIH Neuroscience Postdoctoral Fellowship (1996-1997)
NSF Postdoctoral Fellowship (1997-1998)
NSF-EPSCoR Success Story (2001)
University of Washington, New Investigator Science in Medicine Lecture (2006)
American Heart Association Established Investigator Award (2008-2012)
Fellow of the American Heart Association (2010)
Totman Lecturer 2013 (The University of Vermont)
UC Davis Dean's Award for Excellence in Team Research (2019)
UC Davis Master Educator (2019-2020)

One of the Cell Press' list of "100 inspiring Hispanic/Latinx scientists in America"

Special national and international responsibilities
Member of American Heart Association Cardiovascular Pathology II study section (2001-2003)
Member of the American Heart Association Basic Science Research Council (2001-2003)
Member of special review group for Dr. Donald Bers' NIH PPG application (2004-2005)
Ad-hoc member of NIH/CSR NTRC study section (2005)
Member of the External Advisory Committee for University of Missouri-Columbia PPG "Ion channel regulation of coronary smooth muscle phenotype" (2008)
Member of special review group for Dr. Richard Moss' NIH PPG application (2007-2008)
Member of special review group for Dr. Paul Allen's NIH PPG application (2009)
Vice-chair 2012 FASEB Smooth Muscle Summer Research conference
Chair 2016 FASEB Smooth Muscle Summer Research conference
Reviewer of research grant applications for FONDECYT (Chile; 2009)
Member of NINDS Advisory Panel on Workplace Disparities (2010)
Ad-hoc member of NIH/CSR ESTA study section (October 2011)
Regular member of the Hypertension and Microcirculation study section (2007-2011)
Regular member of the ESTA study section (2013-2018)
Chair of the ESTA study section (2015-2017)
Editorial Board Journal of General Physiology (2015-present)
Editorial Board Journal of Molecular and Cellular Cardiology (2007-present)
Reviewing Editor, The Journal of Physiology (2016-2019)
NHLBI Program Project Review Committee (2019-present)
Editorial Board Vascular Pharmacology (2020-present)
Editorial Board Annual Reviews Physiology (2021-present)

Special responsibilities at UC-Davis
Search committee for Chair of Pediatrics (2015)
Search for Chair of Pediatrics (2016)
Chair of the Search Committee for Associate Dean of Faculty Development and Diversity (2016)
Search committee for Chair of Psychiatry (2017)
Search committee for Dean of UC Davis School of Medicine (2018-2019)
Faculty Executive Committee (2018-present)
Co-Chair of the search committee for the Chair of Radiology of UC Davis School of Medicine (2019-2020)
UC Davis School of Medicine Curriculum Development Team (2019)
UC Davis School of Medicine Curriculum Implementation Team (2020-2021)
Council of Chairs (2015-present)
Pre-Clinical Chairs Committee (2015-present)
Committee on Budget and Projects (2018-present)
Founding Co-Director ARC-MD Program (2018-present)
Co-Director M.D./Ph.D. Program (2019-present)

Professional organizations
American Heart Association BCVS
American Physiological Society (Cardiovascular section)
Biophysical Society
Society of General Physiologists

Contribution to Teaching, Mentoring, and Diversity
As a Puerto Rican, I have made sure that through my teaching, research, and outreach activities I contribute to creating a diverse academic community. I have over twenty years of experience in graduate and medical education, including teaching courses in biophysics, cell signaling, and

cardiovascular physiology. I have mentored 30 postdoctoral fellows and graduate students, 8th of who have gone on to tenured or tenure-track faculty positions, and all of whom are employed in the biomedical workforce. Six of these trainees are from underrepresented minority groups.

My approach to teaching is like my approach to research. I value inclusion, innovation, and thoroughness. I am the founding co-director of the Academic Research Careers for Medical Doctors (ARC-MD) program. The goal of this program is to provide medical students with the foundational skills and professional development that promote a successful career as a physician scientist. The five-year program provides students with research and career mentorship, special experiences, a unique curriculum, and community engagement within a supportive longitudinal learning community. I am also co-director of the M.D./Ph.D. at UC Davis. The program is in its third year. We currently have 22 students at different stages. Seventy percent of these students are URM or disadvantaged groups. I am proud to say that ARC-MD is a national model for the training diverse, community-oriented physician-scientists of the future.

As departmental Chair, over 60% of the faculty that I have recruited are women or from URM groups. Indeed, under my leadership the Department of Physiology and Membrane Biology underwent a massive diversification of its faculty that was associated with an exponential increase in research funding (currently ranked 14 nationally among physiology departments), supporting the view that diverse teams are more productive.

Finally, I will add that as the Interim Chair of the Department of Biochemistry and Molecular Medicine, I recruited the first African American to this Department.

Personal and Team-Based Contributions to Science

I have had a long-standing interest in cardiac and vascular biology with an emphasis on ion channels, Ca^{2+} signaling, and Ca^{2+}-dependent transcription factors. I joined the Institute of Neurobiology of the University of Puerto Rico as an assistant professor in 1999. Two years later, I accepted an offer to join the Department of Physiology and Biophysics of the University of Washington (UW). I worked at the UW for 14 years. In July 2015, I left the UW to become the Chair of the Department of Physiology and Membrane Biology of the University of California, Davis (UC Davis).

In August of 2021, I was named interim Chair of Biochemistry and Molecular Medicine. From June to December of 2022, I served as interim Chair of Medical Microbiology and Immunology. Collectively these departments have over 60 primary faculty members and a research portfolio of about $35M per year.

My approach to science is multi-disciplinary, involving state-of-the-art biophysical, electrophysiological, imaging, cellular, molecular, and computational approaches. Together with colleagues, I have discovered multiple kinds of Ca^{2+} signaling modalities and I am now focused on how they control excitation-contraction coupling in cardiac and arterial smooth muscle. To date, I have published over 110 peer-reviewed papers in top-tier journals, including *Science*, *Nature*, *Proceedings of the National Academy of Sciences*, *Journal of Clinical Investigation*, *Circulation Research*, *Journal of General Physiology*, and *Science Signaling*.

According to Google Scholar, my papers have been cited more than 11,823. These citations are not simply the result of one or two of my articles being cited a disproportionate number of times. Rather, this has been the result of a stream of primary papers receiving a significant number of citations. Indeed, 54 of my papers have been cited at least 54 times (i.e., *h-index* = 54).

Recent work in my lab has focused on how voltage-gated $Ca_V1.2$ and $Ca_V1.3$ channels contribute to excitation-contraction coupling in cardiac and arterial smooth muscle. I use my expertise in optical imaging and electrophysiology, together with newly developed optogenetic and super-resolution

approaches, to study the subcellular organization of Ca^{2+} and K^+ channels and determine how the signaling nano-domains formed by these proteins control the function of pace-makings cells, ventricular myocytes, and arterial smooth muscle cells during physiological and pathological conditions.

I have been funded to do research, uninterruptedly, for 22 years by the NHLBI and NINDS. Postdocs and students have also been awarded extramural funding from the NHLBI and American Heart Association. Indeed, I am proud to note that all the postdocs interested in academic positions left my lab with their own funding.

My research team has a long track record of **technical and conceptual innovation** through **flexibility and adaptability**. We developed optical techniques to image and analyze Ca^{2+} influx via single sarcolemmal Ca^{2+}-permeable channels in cardiac and vascular smooth muscle. These approaches have been used by other groups to image Ca^{2+} influx via TRP channels in endothelial and smooth muscle cells. We also pioneered the use of super-resolution imaging to study signaling nano-domains in vascular smooth muscle.

Below, I highlight our contributions in five specific areas of research.

1. **Local control of Ca^{2+} release in muscle.** I began my research career investigating the mechanisms controlling the release of Ca^{2+} from the sarcoplasmic reticulum of cardiac, skeletal, and smooth muscle cells during the process of excitation-contraction coupling. Together with colleagues, I discovered that, in cardiac muscle, subcellular Ca^{2+} signals, called "Ca^{2+} sparks", resulting from the opening of a small cluster of ryanodine receptors are activated by Ca^{2+} entry via L-type Ca^{2+} channels. In amphibian skeletal muscle, Ca^{2+} sparks are activated by activation of the voltage sensor, while secondary Ca^{2+} sparks are activated by a Ca^{2+}-induced Ca^{2+} release mechanism. In cardiac and skeletal muscle, the synchronous activation of multiple Ca^{2+} sparks cause a cell-wide Ca^{2+} transient that triggers contraction. In smooth muscle, however, Ca^{2+} sparks are rare and activate nearby Ca^{2+}-sensitive K^+ channels. This hyperpolarizes smooth muscle, closing Ca^{2+} channels and decreasing cytosolic Ca^{2+}, which causes relaxation. Thus, depending on identity of Ca^{2+}-sensitive proteins near a Ca^{2+} spark site, the same Ca^{2+} spark can elicit opposite physiological responses.

   A. Nelson, M.T., H. Cheng, M. Rubart, L.F. Santana, A.D. Bonev, H.J. Knot, and W.J. Lederer. 1995. Relaxation of arterial smooth muscle by calcium sparks. *Science*. 270:633-637.
   B. Klein, M.G., H. Cheng, L.F. Santana, Y.H. Jiang, W.J. Lederer, and M.F. Schneider. 1996. Two mechanisms of quantized calcium release in skeletal muscle. *Nature*. 379:455-458.
   C. Santana, L.F., H. Cheng, A.M. Gomez, M.B. Cannell, and W.J. Lederer. 1996. Relation between the sarcolemmal Ca^{2+} current and Ca^{2+} sparks and local control theories for cardiac excitation-contraction coupling. *Circulation Research*. 78:166-171.
   D. Gomez, A.M., H.H. Valdivia, H. Cheng, M.R. Lederer, L.F. Santana, M.B. Cannell, S.A. McCune, R.A. Altschuld, and W.J. Lederer. 1997. Defective excitation-contraction coupling in experimental cardiac hypertrophy and heart failure. *Science*. 276:800-806.
   E. Santana, L.F., A.M. Gomez, and W.J. Lederer. 1998. Ca^{2+} flux through promiscuous cardiac Na^+ channels: slip-mode conductance. *Science*. 279:1027-1033.

2. **Excitation-transcription coupling in cardiac and vascular smooth muscle.** My team determined the biophysical mechanisms linking specific Ca^{2+} signaling modalities (e.g., sparks, sparklets, waves) to the activation of Ca^{2+}-dependent transcription factors. We discovered that the transcription factor NFATc3 is activated by local Ca^{2+} signals in cardiac and vascular smooth muscle. Furthermore, we found that activation of NFATc3 underlies reductions in K^+

channel expression in heart after myocardial infarction and smooth muscle during the development of hypertension.

A. Amberg, G.C., A.D. Bonev, C.F. Rossow, M.T. Nelson, and L.F. Santana. 2003. Modulation of the molecular composition of large conductance, Ca^{2+} activated K^+ channels in vascular smooth muscle during hypertension. *Journal of Clinical Investigation*. 112:717-724.
B. Amberg, G.C., C.F. Rossow, M.F. Navedo, and L.F. Santana. 2004. NFATc3 regulates Kv2.1 expression in arterial smooth muscle. *Journal Biological Chemistry*. 279:47326-47334.
C. Rossow, C.F., E. Minami, E.G. Chase, C.E. Murry, and L.F. Santana. 2004. NFATc3-Induced Reductions in Voltage-Gated K^+ Currents After Myocardial Infarction. *Circulation Research*. 94:1340-1350.
D. Nieves-Cintrón, M., G.C. Amberg, C.B. Nichols, J.D. Molkentin, and L.F. Santana. 2007. Activation of NFATc3 down-regulates the β1 subunit of large conductance, calcium-activated K^+ channels in arterial smooth muscle and contributes to hypertension. *Journal of Biological Chemistry*. 282:3231-3240.
E. Nieves-Cintrón, M., G.C. Amberg, M.F. Navedo, J.D. Molkentin, and L.F. Santana. 2008. The control of Ca^{2+} influx and NFATc3 signaling in arterial smooth muscle during hypertension. *Proceedings of the National Academy of Sciences*. 105:15623-15628.

3. **Coupled gating of voltage-gated Ca^{2+} channels.** Since 2004, my team has been developing strategies to perform optical recordings of Ca^{2+}-permeable channels with high temporal and spatial resolution. Using these approaches, we discovered that contrary to long-held views in the field of ion channel biophysics, Ca^{2+} channel activity along the surface membrane of cardiac and smooth muscle cells was not homogeneous. Instead, Ca^{2+} channel activity was higher at specific regions of the cell where these channels cluster. The formation of these clusters allows channels to undergo dynamic physical interactions that enhance the activity of the adjoined channels. In the case of $Ca_V1.2$ channels, these interactions last longer than the local Ca^{2+} signal that induces it, constituting a form of molecular memory.

A. Moreno, C.M., R.E. Dixon, S. Tajada, C. Yuan, X. Opitz-Araya, M.D. Binder, and L.F. Santana. 2016. Ca^{2+} entry into neurons is facilitated by cooperative gating of clustered $Ca_V1.3$ channels. *eLife*. 5.
B. Dixon, R.E., C.M. Moreno, C. Yuan, X. Opitz-Araya, M.D. Binder, M.F. Navedo, and L.F. Santana. 2015. Graded Ca^{2+}/calmodulin-dependent coupling of voltage-gated CaV1.2 channels. *eLife*. 4.
C. Dixon, R.E., C. Yuan, E.P. Cheng, M.F. Navedo, and L.F. Santana. 2012. Ca^{2+} signaling amplification by oligomerization of L-type $Ca_V1.2$ channels. *Proceedings of the National Academy of Sciences*. 109:1749-1754.
D. Navedo, M.F., E.P. Cheng, C. Yuan, S. Votaw, J.D. Molkentin, J.D. Scott, and L.F. Santana. 2010. Increased coupled gating of L-type Ca^{2+} channels during hypertension and Timothy syndrome. *Circulation research*. 106:748-756.
E. Navedo, M.F., G.C. Amberg, M. Nieves, J.D. Molkentin, and L.F. Santana. 2006. Mechanisms Underlying Heterogeneous Ca^{2+} Sparklet Activity in Arterial Smooth Muscle. *Journal of General Physiology*. 127:611-622.
F. Navedo, M.F., G.C. Amberg, V.S. Votaw, and L.F. Santana. 2005. Constitutively active L-type Ca^{2+} channels. *Proceedings of the National Academy of Sciences*. 102:11112-11117.

4. **AKAP150 control of endothelial cells and vascular smooth muscle.** During the last seven years, our group has been working arduously on trying to determine the cellular mechanisms controlling local TRPV4 and $Ca_V1.2$ channel gating. This work has revealed that the anchoring

protein AKAP150 plays a critical role in targeting kinases and phosphatases to specific regions of the surface membrane smooth muscle and endothelial cells where these proteins can control the function of adjacent $Ca_V1.2$ and TRPV4 channels. Our most recent finding related to this application is that loss or delocalization of AKAP150 alters TRPV4 and $Ca_V1.2$ channel function and likely myogenic tone in pial and parenchymal arterioles.

A. Sonkusare, S.K., T. Dalsgaard, A.D. Bonev, D.C. Hill-Eubanks, M.I. Kotlikoff, J.D. Scott, L.F. Santana, and M.T. Nelson. 2014. AKAP150-dependent cooperative TRPV4 channel gating is central to endothelium-dependent vasodilation and is disrupted in hypertension. *Science Signaling*. 7:ra66.
B. Nystoriak, M.A., M. Nieves-Cintron, P.J. Nygren, S.A. Hinke, C.B. Nichols, C.Y. Chen, J.L. Puglisi, L.T. Izu, D.M. Bers, M.L. Dell'acqua, J.D. Scott, L.F. Santana, and M.F. Navedo. 2014. AKAP150 contributes to enhanced vascular tone by facilitating large-conductance Ca^{2+}-activated K^+ channel remodeling in hyperglycemia and diabetes mellitus. *Circulation Research*. 114:607-615.
C. Mercado, J., R. Baylie, M.F. Navedo, C. Yuan, J.D. Scott, M.T. Nelson, J.E. Brayden, and L.F. Santana. 2014. Local control of TRPV4 channels by AKAP150-targeted PKC in arterial smooth muscle. *Journal of General Physiology*. 143:559-575.
D. Navedo, M.F., M. Nieves-Cintrón, G.C. Amberg, C. Yuan, V.S. Votaw, W.J. Lederer, G.S. McKnight, and L.F. Santana. 2008. AKAP150 Is Required for Stuttering Persistent Ca^{2+} Sparklets and Angiotensin II Induced Hypertension. *Circulation Research*. 102:e1-e11.

Active Research Funding

1. Multi-Scale Modeling of Vascular Signaling Units (NHLBI grant R01-HL152621). Total award: $545,332 + 227,336 supplement per year. Role: Contact MPI.
2. Tuning L-type Ca Channel Activity in Arterial Smooth Muscle by Kv Channel-Mediated Clustering (NHLBI grant R01-HL144971). Total award: $508,101 per year. Role: Contact MPI.
3. Neuronal Kv2.1 Potassium Channels as Organizers of Somatic L-Type Calcium Channels Microdomains (NINDS grant R01-NS114210). Total award: $482,574 per year. Role: MPI
4. Development of a Predictive NeuroCardiovascular simulator (OT2-OD026580). Total award: $1,498,542 per year. Role: MPI.
5. *In silico* safety pharmacology (NHLBI grant R01-128537). Total award: $648,797 per year. Role: MPI.

Bibliography

Citation statistics
An up-to-date version of the table below can be obtained using this link: https://scholar.google.com/citations?user=ybXybb0AAAAJ&hl=en

	All	**Since 2018**
Citations	12059	3529
h-index*	56	34
i10-index**	97	84

* h-index is the number of h papers that have been cited in other papers at least h times. Accordingly, an h-index of 56 indicates that 56 of the papers below have been cited at least 56 times.
** i10-index is the number of publications with at least 10 citations.

Peer-reviewed articles:

1. **Ren L, Thai PN, Gopireddy RR, Timofeyev V, Ledford HA, Woltz RL, Park S, Puglisi JL, Moreno CM, Santana LF, Conti AC, Kotlikoff MI, Xiang YK, Yarov-Yarovoy V, Zaccolo M, Zhang XD, Yamoah EN, Navedo MF, and Chiamvimonvat N**. Adenylyl cyclase isoform 1 contributes to sinoatrial node automaticity via functional microdomains. *JCI Insight* 7: 2022.
2. **Manning D, and Santana LF**. Regulating voltage-gated ion channels with nanobodies. *Nat Commun* 13: 7557, 2022.
3. **Guarina L, Moghbel AN, Pourhosseinzadeh MS, Cudmore RH, Sato D, Clancy CE, and Santana LF**. Biological noise is a key determinant of the reproducibility and adaptability of cardiac pacemaking and EC coupling. *J Gen Physiol* 154: 2022.
4. **Grainger N, and Santana LF**. The Central Brain of the Heart: The Sinoatrial Node. *JACC Clin Electrophysiol* 8: 1216-1218, 2022.
5. **Grainger N, and Santana LF**. The Inferior Sinoatrial Node Suffers the Most During Heart Failure. *JACC Clin Electrophysiol* 8: 1354-1356, 2022.
6. **Earley S, Santana LF, and Lederer WJ**. The physiological sensor channels TRP and piezo: Nobel Prize in Physiology or Medicine 2021. *Physiol Rev* 102: 1153-1158, 2022.
7. **Dixon RE, Navedo MF, Binder MD, and Santana LF**. Mechanisms and physiological implications of cooperative gating of clustered ion channels. *Physiol Rev* 102: 1159-1210, 2022.
8. **Cudmore RH, and Santana LF**. Piezo1 Tunes Blood Flow in the Central Nervous System. *Circ Res* 130: 1547-1549, 2022.
9. **Vierra NC, O'Dwyer SC, Matsumoto C, Santana LF, and Trimmer JS**. Regulation of neuronal excitation-transcription coupling by Kv2.1-induced clustering of somatic L-type Ca(2+) channels at ER-PM junctions. *Proc Natl Acad Sci U S A* 118: 2021.
10. **Tiscione SA, Casas M, Horvath JD, Lam V, Hino K, Ory DS, Santana LF, Simo S, Dixon RE, and Dickson EJ**. IP3R-driven increases in mitochondrial Ca(2+) promote neuronal death in NPC disease. *Proc Natl Acad Sci U S A* 118: 2021.
11. **Lee FK, Lee JC, Shui B, Reining S, Jibilian M, Small DM, Jones JS, Allan-Rahill NH, Lamont MR, Rizzo MA, Tajada S, Navedo MF, Santana LF, Nishimura N, and Kotlikoff MI**. Genetically engineered mice for combinatorial cardiovascular optobiology. *Elife* 10: 2021.
12. **Grainger N, Guarina L, Cudmore RH, and Santana LF**. The organization of the sino-atrial node microvasculature varies regionally to match local myocyte excitability. *Function* 2021.
13. **Prada MP, Syed AU, Reddy GR, Martin-Aragon Baudel M, Flores-Tamez VA, Sasse KC, Ward SM, Sirish P, Chiamvimonvat N, Bartels P, Dickson EJ, Hell JW, Scott JD, Santana LF, Xiang YK, Navedo MF, and Nieves-Cintron M**. AKAP5 complex facilitates purinergic modulation of vascular L-type Ca(2+) channel CaV1.2. *Nat Commun* 11: 5303, 2020.
14. **O'Dwyer SC, Palacio S, Matsumoto C, Guarina L, Klug NR, Tajada S, Rosati B, McKinnon D, Trimmer JS, and Santana LF**. Kv2.1 channels play opposing roles in regulating membrane potential, Ca(2+) channel function, and myogenic tone in arterial smooth muscle. *Proc Natl Acad Sci U S A* 117: 3858-3866, 2020.
15. **O'Dwyer SC, Navedo MF, and Santana LF**. Maladaptive response of arterial myocytes to chronic exposure to Ca(2+) channel blockers. *Proc Natl Acad Sci U S A* 117: 18151-18153, 2020.
16. **Nieves-Cintron M, Santana LF, and Navedo MF**. TRPML1ng on sparks. *Sci Signal* 13: 2020.
17. **Grainger N, and Santana LF**. Metabolic-electrical control of coronary blood flow. *Proc Natl Acad Sci U S A* 117: 8231-8233, 2020.
18. **Drum BM, Yuan C, de la Mata A, Grainger N, and Santana LF**. Junctional sarcoplasmic reticulum motility in adult mouse ventricular myocytes. *Am J Physiol Cell Physiol* 318: C598-C604, 2020.
19. **Clancy CE, and Santana LF**. Evolving Discovery of the Origin of the Heartbeat: A New Perspective on Sinus Rhythm. *JACC Clin Electrophysiol* 6: 932-934, 2020.
20. **Vierra NC, Kirmiz M, van der List D, Santana LF, and Trimmer JS**. Kv2.1 mediates spatial and functional coupling of L-type calcium channels and ryanodine receptors in mammalian neurons. *Elife* 8: 2019.

21. **Tiscione SA, Vivas O, Ginsburg KS, Bers DM, Ory DS, Santana LF, Dixon RE, and Dickson EJ**. Disease-associated mutations in Niemann-Pick type C1 alter ER calcium signaling and neuronal plasticity. *J Cell Biol* 218: 4141-4156, 2019.
22. **Syed AU, Reddy GR, Ghosh D, Prada MP, Nystoriak MA, Morotti S, Grandi E, Sirish P, Chiamvimonvat N, Hell JW, Santana LF, Xiang YK, Nieves-Cintron M, and Navedo MF**. Adenylyl cyclase 5-generated cAMP controls cerebral vascular reactivity during diabetic hyperglycemia. *J Clin Invest* 129: 3140-3152, 2019.
23. **Sato D, Hernandez-Hernandez G, Matsumoto C, Tajada S, Moreno CM, Dixon RE, O'Dwyer S, Navedo MF, Trimmer JS, Clancy CE, Binder MD, and Santana LF**. A stochastic model of ion channel cluster formation in the plasma membrane. *J Gen Physiol* 151: 1116-1134, 2019.
24. **Prada MP, Syed AU, Buonarati OR, Reddy GR, Nystoriak MA, Ghosh D, Simo S, Sato D, Sasse KC, Ward SM, Santana LF, Xiang YK, Hell JW, Nieves-Cintron M, and Navedo MF**. A Gs-coupled purinergic receptor boosts Ca(2+) influx and vascular contractility during diabetic hyperglycemia. *Elife* 8: 2019.
25. **Dong JX, Lee Y, Kirmiz M, Palacio S, Dumitras C, Moreno CM, Sando R, Santana LF, Sudhof TC, Gong B, Murray KD, and Trimmer JS**. A toolbox of nanobodies developed and validated for use as intrabodies and nanoscale immunolabels in mammalian brain neurons. *Elife* 8: 2019.
26. **De La Mata A, Tajada S, O'Dwyer S, Matsumoto C, Dixon RE, Hariharan N, Moreno CM, and Santana LF**. BIN1 Induces the Formation of T-Tubules and Adult-Like Ca(2+) Release Units in Developing Cardiomyocytes. *Stem Cells* 37: 54-64, 2019.
27. **Smith FD, Omar MH, Nygren PJ, Soughayer J, Hoshi N, Lau HT, Snyder CG, Branon TC, Ghosh D, Langeberg LK, Ting AY, Santana LF, Ong SE, Navedo MF, and Scott JD**. Single nucleotide polymorphisms alter kinase anchoring and the subcellular targeting of A-kinase anchoring proteins. *Proc Natl Acad Sci U S A* 115: E11465-E11474, 2018.
28. **Sato D, Dixon RE, Santana LF, and Navedo MF**. A model for cooperative gating of L-type Ca2+ channels and its effects on cardiac alternans dynamics. *PLoS Comput Biol* 14: e1005906, 2018.
29. **Nieves-Cintron M, Tajada S, Santana LF, and Navedo MF**. Total internal reflection fluorescence microscopy in vascular smooth muscle. *Signal Transduct Smooth Muscle* 5: 87-103, 2018.
30. **Ghosh D, Nieves-Cintron M, Tajada S, Brust-Mascher I, Horne MC, Hell JW, Dixon RE, Santana LF, and Navedo MF**. Dynamic L-type CaV1.2 channel trafficking facilitates CaV1.2 clustering and cooperative gating. *Biochim Biophys Acta Mol Cell Res* 1865: 1341-1355, 2018.
31. **Vivas O, Moreno CM, Santana LF, and Hille B**. Proximal clustering between BK and CaV1.3 channels promotes functional coupling and BK channel activation at low voltage. *Elife* 6: 2017.
32. **Tajada S, Moreno CM, O'Dwyer S, Woods S, Sato D, Navedo MF, and Santana LF**. Distance constraints on activation of TRPV4 channels by AKAP150-bound PKCalpha in arterial myocytes. *J Gen Physiol* 149: 639-659, 2017.
33. **Nieves-Cintron M, Syed AU, Buonarati OR, Rigor RR, Nystoriak MA, Ghosh D, Sasse KC, Ward SM, Santana LF, Hell JW, and Navedo MF**. Impaired BKCa channel function in native vascular smooth muscle from humans with type 2 diabetes. *Sci Rep* 7: 14058, 2017.
34. **Li L, Li J, Drum BM, Chen Y, Yin H, Guo X, Luckey SW, Gilbert ML, McKnight GS, Scott JD, Santana LF, and Liu Q**. Loss of AKAP150 promotes pathological remodelling and heart failure propensity by disrupting calcium cycling and contractile reserve. *Cardiovasc Res* 113: 147-159, 2017.
35. **Ghosh D, Syed AU, Prada MP, Nystoriak MA, Santana LF, Nieves-Cintron M, and Navedo MF**. Calcium Channels in Vascular Smooth Muscle. *Adv Pharmacol* 78: 49-87, 2017.
36. **Gentil BJ, O'Ferrall E, Chalk C, Santana LF, Durham HD, and Massie R**. A New Mutation in FIG4 Causes a Severe Form of CMT4J Involving TRPV4 in the Pathogenic Cascade. *J Neuropathol Exp Neurol* 76: 789-799, 2017.
37. **Nieves-Cintron M, Hirenallur-Shanthappa D, Nygren PJ, Hinke SA, Dell'Acqua ML, Langeberg LK, Navedo M, Santana LF, and Scott JD**. AKAP150 participates in calcineurin/NFAT activation during the down-regulation of voltage-gated K(+) currents in ventricular myocytes following myocardial infarction. *Cell Signal* 28: 733-740, 2016.

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