






James Spudich

Douglass M. and Nola Leishman Professor of Cardiovascular Disease, Emeritus
Biochemistry

 NIH Biosketch available Online

 Curriculum Vitae available Online

 Resume available Online

Bio

BIO

James Spudich, Douglass M. and Nola Leishman Professor of Cardiovascular Disease, is in the Department of Biochemistry at Stanford University School of Medicine. He received his B.S. in chemistry from the University of Illinois in 1963 and his Ph.D. in biochemistry from Stanford in 1968. He did postdoctoral work in genetics at Stanford and in structural biology at the MRC Laboratory in Cambridge, England. From 1971 to 1977 he was Assistant, Associate, and Full Professor in the Department of Biochemistry and Biophysics, University of California, San Francisco. In 1977 he was appointed Professor in the Department of Structural Biology at Stanford University. Spudich served as Chairman of the Department of Structural Biology from 1979-1984. Since 1992 he has been Professor in the Department of Biochemistry, and served as Chairman from 1994-1998. He has held a joint appointment as Professor in the Department of Developmental Biology since 1989. From 1998 to 2002, he was Co-Founder and first Director of the Stanford Interdisciplinary Program in Bioengineering, Biomedicine and Biosciences called Bio-X. At present he is also an Adjunct Professor at the National Center for Biological Sciences, Tata Institute of Fundamental Research and InStem in Bangalore, India.

Spudich has given more than 40 named lectureships and keynote addresses, including the First Annual Lecture of the series "The James Spudich AHA Research Committee Lecture," named in his honor; the Pauling Lecture, Stanford; the Paul Dudley White Lecture, Mass General Hospital of Harvard University; the DeWitt Stetten, Jr. Lecture, NIH; the Meyerhof Lecture, Heidelberg; the Keith R. Porter Lecture, ASCB; the Hans Neurath Lecture, University of Washington; the National Lecture, Biophysical Society; the Mayer Lecture, MIT; Plenary Lecture (shared with Aaron Klug), Madrid International Congress on Cell Biology; the Friday Evening Lecture, Woods Hole; and the Cori Lecture, Washington University.

Spudich was a recipient of a Guggenheim Fellowship in 1978. He was elected to the National Academy of Sciences in 1991. He is also a member of the American Academy of Arts and Sciences, and the American Association for the Advancement of Science. Spudich received the American Heart Association Basic Research Prize, the Alexander von Humboldt Research Award, the Biophysical Society Lifetime Research Career Award, the Lewis S. Rosenstiel Award for Outstanding Research Achievement in the Field of Basic Medical Studies, the American Chemical Society's Award for the Chemistry of Biological Processes, the Biophysics Society Award for Outstanding Investigator in the Field of Single Molecule Biology, the E.B. Wilson Medal, the Arthur Kornberg and Paul Berg Lifetime Achievement Award in Biomedical Sciences, the Wiley Prize in Biomedical Sciences, the Ahmed H. Zewail Award and the Massry Prize. In 2012, he received the Albert Lasker Basic Medical Research Award.

ACADEMIC APPOINTMENTS

- Professor Emeritus-Hourly, Biochemistry
- Member, Bio-X
- Member, Cardiovascular Institute
- Member, Maternal & Child Health Research Institute (MCHRI)

- Member, Stanford Cancer Institute

ADMINISTRATIVE APPOINTMENTS

- Assistant Professor, Department of Biochemistry & Biophysics, University of California, San Francisco, (1971-1974)
- Associate Professor, Department of Biochemistry & Biophysics, University of California, San Francisco, (1974-1976)
- Professor, Department of Biochemistry and Biophysics, University of California, San Francisco, (1976-1977)
- Professor, Department of Structural Biology, Stanford University School of Medicine, (1977-1992)
- Chairman, Department of Structural Biology, Stanford University School of Medicine, (1979-1984)
- Professor, Department of Developmental Biology, Stanford University School of Medicine, (1989-2011)
- Professor, Department of Biochemistry, Stanford University School of Medicine, (1992- present)
- Chairman, Department of Biochemistry, Stanford University School of Medicine, (1994-1998)
- Co-Founder and first Director, Interdisciplinary Program, Bio-X, Stanford University, (1998-2002)
- Co-Founder, Cytokinetics, Inc, (1998- present)
- Co-Founder, MyoKardia, Inc., (2012- present)
- Co-Founder, Kainomyx, Inc., (2019- present)

HONORS AND AWARDS

- Alumni Achievement Award, University of Illinois (2018)
- Founders Award, Biophysical Society (2018)
- Inaugural ASCB Fellow, American Society for Cell Biology (2016)
- Liberal Arts and Sciences Alumni Achievement Award, University of Illinois (2015)
- Honorary Doctor of Sciences Degree, Guelph University, Guelph University (2014)
- Ahmed H. Zewail Award Gold Medal, Wayne State University (2013)
- Massry Prize, Massry Foundation (2013)
- Albert Lasker Basic Medical Research Award, Lasker Foundation (2012)
- Arthur Kornberg and Paul Berg Lifetime Achievement Award in Biomedical Sciences, Stanford University School of Medicine (2012)
- Wiley Prize in Biomedical Sciences, Rockefeller University (2012)
- E.B. Wilson Medal, The American Society for Cell Biology (2011)
- U.S. Genomics Award for Outstanding Investigator in the field of Single Molecule Biology, Biophysical Society (2006)
- Elected Fellow of the American Association for the Advancement of Science, the American Association for the Advancement of Science (2001)
- Elected Fellow of the American Academy of Arts and Sciences, the American Academy of Arts and Sciences (1997)
- 1997 Repligen Award in Chemistry of Biological Processes, Division of Biological Chemistry of the American Chemical Society (1996)
- Lewis S. Rosenstiel Award, Brandeis University (1996)
- Biophysical Society Lifetime Research Career Award, Biophysical Society (1995)
- External Scientific Member of the Max-Planck-Institute für Biochemie in Martinsried bei München, Max-Planck Society (1994)
- Alexander von Humboldt Research Award, Alexander von Humboldt Research Foundation (1991)
- American Heart Association Research Prize, National American Heart Association (1991)
- Elected Member of the National Academy of Sciences, the National Academy of Sciences (1991)
- NIH Merit Award, National Institutes of Health (1991)
- Named the "Douglass M. and Nola Leishman Professor of Cardiovascular Disease", Stanford University (1987 - present)

PROFESSIONAL EDUCATION

- B.S., University of Illinois , Chemistry (1963)
- Ph.D., Stanford University , Biochemistry (1968)
- Postdoctoral, Stanford University , Genetics (1969)
- Postdoctoral, Cambridge University, MRC LMB , Structural Biology (1971)

LINKS

- My Lab Site: <http://spudlab.stanford.edu/>

Research & Scholarship

CURRENT RESEARCH AND SCHOLARLY INTERESTS

Our general research interest is the structure and function of molecular motors in vitro and in vivo, with emphasis on understanding the molecular basis of muscle contraction. Our major areas of specific interest are the molecular basis of energy transduction that leads to ATP-driven myosin movement on actin, the roles of the myosin family of molecular motors in eukaryotic cells, the regulation of actin and myosin interaction and their assembly states, and the biochemistry and regulation of the attachment of molecular motors to their corresponding cargo.

Our approaches include biochemical, genetic, biophysical and structural studies of actin, myosin, and associated proteins from eukaryotic cells. We have designed and developed in vitro assays for ATP-dependent movement of purified myosin on filaments reconstituted from purified actin. We have taken this assay to the single molecule level, using laser traps, total internal reflection fluorescence microscopy, and gold nanoparticle tracking. Myosin cloning and expression of mutagenized forms that are analyzed for altered functions is routine in our laboratory.

The detailed understanding we have developed of how myosin transduces the chemical energy of ATP hydrolysis into mechanical movement has led us to our current focus on human hypertrophic cardiomyopathy (HCM) caused by missense mutations in human β -cardiac myosin. Our goal is to elucidate the molecular basis of hypercontractility seen clinically resulting from HCM mutations. We postulated in 2015 that a majority of HCM mutations shift β -cardiac myosin heads from a sequestered off-state to an active on-state for interaction with actin, resulting in the hypercontractility seen clinically. This hypothesis is different from earlier prevailing views, and this viewing an old disease in a new light is the basis of all of our current research. We now have extensive evidence for this hypothesis using a combination of the various high-resolution technologies we have developed over the years as well as new approaches. Our work is now providing possible paths forward for therapeutic intervention for cardiomyopathy patients.

Teaching

COURSES

2019-20

- Currents in Biochemistry: BIOC 257 (Aut)

STANFORD ADVISEES

Postdoctoral Faculty Sponsor

Rama Reddy Goluguri, Neha Nandwani, Divya Pathak

Doctoral Dissertation Co-Advisor (AC)

Ramon Lorenzo Labitigan

GRADUATE AND FELLOWSHIP PROGRAM AFFILIATIONS

- Biochemistry (Phd Program)
- Biophysics (Phd Program)

Publications

PUBLICATIONS

- **Hypertrophic cardiomyopathy mutations in the pliant and light chain-binding regions of the lever arm of human β -cardiac myosin have divergent effects on myosin function.** *eLife*
Morck, M. M., Bhowmik, D., Pathak, D., Dawood, A., Spudich, J., Ruppel, K. M.
2022; 11
- **Hypertrophic cardiomyopathy: Mutations to mechanisms to therapies.** *Frontiers in physiology*
Kawana, M., Spudich, J. A., Ruppel, K. M.
2022; 13: 975076
- **Hypertrophic cardiomyopathy beta-cardiac myosin mutation (P710R) leads to hypercontractility by disrupting super relaxed state.** *Proceedings of the National Academy of Sciences of the United States of America*
Vander Roest, A. S., Liu, C., Morck, M. M., Kooiker, K. B., Jung, G., Song, D., Dawood, A., Jhingran, A., Pardon, G., Ranjbarvaziri, S., Fajardo, G., Zhao, M., Campbell, et al
2021; 118 (24)
- **Single Residue Variation in Skeletal Muscle Myosin Enables Direct and Selective Drug Targeting for Spasticity and Muscle Stiffness.** *Cell*
Gyimesi, M., Horvath, A. I., Turos, D., Suthar, S. K., Penzes, M., Kurdi, C., Canon, L., Kikuti, C., Ruppel, K. M., Trivedi, D. V., Spudich, J. A., Lorincz, I., Rauscher, et al
2020
- **The hypertrophic cardiomyopathy mutations R403Q and R663H increase the number of myosin heads available to interact with actin** *SCIENCE ADVANCES*
Sarkar, S. S., Trivedi, D., Morck, M. M., Adhikari, A. S., Pasha, S. N., Ruppel, K. M., Spudich, J. A.
2020; 6 (14): eaax0069
- **The Myosin Family of Mechanoenzymes: From Mechanisms to Therapeutic Approaches.** *Annual review of biochemistry*
Trivedi, D. V., Nag, S., Spudich, A., Ruppel, K. M., Spudich, J. A.
2020
- **β -Cardiac myosin hypertrophic cardiomyopathy mutations release sequestered heads and increase enzymatic activity.** *Nature communications*
Adhikari, A. S., Trivedi, D. V., Sarkar, S. S., Song, D., Kooiker, K. B., Bernstein, D., Spudich, J. A., Ruppel, K. M.
2019; 10 (1): 2685
- **Three perspectives on the molecular basis of hypercontractility caused by hypertrophic cardiomyopathy mutations** *PFLUGERS ARCHIV-EUROPEAN JOURNAL OF PHYSIOLOGY*
Spudich, J. A.
2019; 471 (5): 701–17
- **Three perspectives on the molecular basis of hypercontractility caused by hypertrophic cardiomyopathy mutations.** *Pflugers Archiv : European journal of physiology*
Spudich, J. A.
2019
- **The Myosin Mesa and Hypertrophic Cardiomyopathy: Mutations to Mechanisms to Therapies**
Spudich, J.
CELL PRESS.2019: 35A
- **On the Functional Assessment of Hypertrophic Cardiomyopathy-Causing Mutations in Human β -Cardiac Myosin and the Role of Myosin Binding Protein-C**
Trivedi, D. V., Sarkar, S. S., Adhikari, A. S., Morck, M. M., Kooiker, K. B., Bernstein, D., Ruppel, K. M., Spudich, J. A.
CELL PRESS.2019: 466A–467A

- **Deciphering the super relaxed state of human beta-cardiac myosin and the mode of action of mavacamten from myosin molecules to muscle fibers** *PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA*
Anderson, R. L., Trivedi, D. V., Sarkar, S. S., Henze, M., Ma, W., Gong, H., Rogers, C. S., Gorham, J. M., Wong, F. L., Morck, M. M., Seidman, J. G., Ruppel, K. M., Irving, et al
2018; 115 (35): EB143–EB152
- **Controlling load-dependent kinetics of beta-cardiac myosin at the single-molecule level.** *Nature structural & molecular biology*
Liu, C., Kawana, M., Song, D., Ruppel, K. M., Spudich, J. A.
2018; 25 (6): 505–14
- **The myosin mesa and the basis of hypercontractility caused by hypertrophic cardiomyopathy mutations.** *Nature structural & molecular biology*
Nag, S., Trivedi, D. V., Sarkar, S. S., Adhikari, A. S., Sunitha, M. S., Sutton, S., Ruppel, K. M., Spudich, J. A.
2017; 24 (6): 525-533
- **Hypertrophic cardiomyopathy and the myosin mesa: viewing an old disease in a new light.** *Biophysical reviews*
Trivedi, D. V., Adhikari, A. S., Sarkar, S. S., Ruppel, K. M., Spudich, J. A.
2017
- **Multidimensional structure-function relationships in human beta-cardiac myosin from population-scale genetic variation** *PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA*
Homburger, J. R., Green, E. M., Caleshu, C., Sunitha, M. S., Taylor, R. E., Ruppel, K. M., Metpally, R. P., Colan, S. D., Michels, M., Day, S. M., Olivotto, I., Bustamante, C. D., Dewey, et al
2016; 113 (24): 6701-6706
- **A small-molecule inhibitor of sarcomere contractility suppresses hypertrophic cardiomyopathy in mice** *SCIENCE*
Green, E. M., Wakimoto, H., Anderson, R. L., Evanchik, M. J., Gorham, J. M., Harrison, B. C., Henze, M., Kawas, R., Oslob, J. D., Rodriguez, H. M., Song, Y., Wan, W., Leinwand, et al
2016; 351 (6273): 617-621
- **Effects of hypertrophic and dilated cardiomyopathy mutations on power output by human beta-cardiac myosin** *JOURNAL OF EXPERIMENTAL BIOLOGY*
Spudich, J. A., Aksel, T., Bartholomew, S. R., Nag, S., Kawana, M., Yu, E. C., Sarkar, S. S., Sung, J., Sommese, R. F., Sutton, S., Cho, C., Adhikari, A. S., Taylor, et al
2016; 219 (2): 161-167
- **Ensemble force changes that result from human cardiac myosin mutations and a small-molecule effector.** *Cell reports*
Aksel, T., Choe Yu, E., Sutton, S., Ruppel, K. M., Spudich, J. A.
2015; 11 (6): 910-920
- **The myosin mesa and a possible unifying hypothesis for the molecular basis of human hypertrophic cardiomyopathy** *BIOCHEMICAL SOCIETY TRANSACTIONS*
Spudich, J. A.
2015; 43: 64-72
- **Harmonic force spectroscopy measures load-dependent kinetics of individual human β -cardiac myosin molecules.** *Nature communications*
Sung, J., Nag, S., Mortensen, K. I., Vestergaard, C. L., Sutton, S., Ruppel, K., Flyvbjerg, H., Spudich, J. A.
2015; 6: 7931-?
- **Hypertrophic and Dilated Cardiomyopathy: Four Decades of Basic Research on Muscle Lead to Potential Therapeutic Approaches to These Devastating Genetic Diseases** *BIOPHYSICAL JOURNAL*
Spudich, J. A.
2014; 106 (6): 1236-1249
- **One path to understanding energy transduction in biological systems** *NATURE MEDICINE*
Spudich, J. A.
2012; 18 (10): 1478-1482
- **The power stroke of myosin VI and the basis of reverse directionality** *PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA*
Bryant, Z., Altman, D., Spudich, J. A.
2007; 104 (3): 772-777

- **Characterization of the one-head bound intermediate that occurs as myosin V walks on actin** *51st Annual Meeting of the Biophysical-Society*
Dunn, A. R., Spudich, J. A.
CELL PRESS.2007: 190A-190A
- **Single molecule high-resolution colocalization of Cy3 and Cy5 attached to macromolecules measures intramolecular distances through time** *PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA*
Churchman, L. S., Okten, Z., Rock, R. S., Dawson, J. F., Spudich, J. A.
2005; 102 (5): 1419-1423
- **A FRET-based sensor reveals large ATP hydrolysis-induced conformational changes and three distinct states of the molecular motor myosin** *CELL*
Shih, W. M., Gryczynski, Z., Lakowicz, J. R., Spudich, J. A.
2000; 102 (5): 683-694
- **The neck region of the myosin motor domain acts as a lever arm to generate movement** *PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA*
Uyeda, T. Q., ABRAMSON, P. D., Spudich, J. A.
1996; 93 (9): 4459-4464
- **SINGLE MYOSIN MOLECULE MECHANICS - PICONEWTON FORCES AND NANOMETER STEPS** *NATURE*
Finer, J. T., Simmons, R. M., Spudich, J. A.
1994; 368 (6467): 113-119
- **MYOSIN SUBFRAGMENT-1 IS SUFFICIENT TO MOVE ACTIN-FILAMENTS INVITRO** *NATURE*
Toyoshima, Y. Y., Kron, S. J., McNally, E. M., NIEBLING, K. R., Toyoshima, C., Spudich, J. A.
1987; 328 (6130): 536-539
- **DISRUPTION OF THE DICTYOSTELIUM MYOSIN HEAVY-CHAIN GENE BY HOMOLOGOUS RECOMBINATION** *SCIENCE*
DeLozanne, A., Spudich, J. A.
1987; 236 (4805): 1086-1091
- **Myosin Subfragment-1 is Sufficient to Move Actin Filaments In Vitro** *Myosin Subfragment-1 is Sufficient to Move Actin Filaments In Vitro*
Toyoshima, Y. Y., et al
1987; 328: 536-539
- **FLUORESCENT ACTIN-FILAMENTS MOVE ON MYOSIN FIXED TO A GLASS-SURFACE** *PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA*
Kron, S. J., Spudich, J. A.
1986; 83 (17): 6272-6276
- **MOVEMENT OF MYOSIN-COATED BEADS ON ORIENTED FILAMENTS RECONSTITUTED FROM PURIFIED ACTIN** *NATURE*
Spudich, J. A., Kron, S. J., Sheetz, M. P.
1985; 315 (6020): 584-586
- **CYTOSKELETAL ELEMENTS OF CHICK-EMBRYO FIBROBLASTS REVEALED BY DETERGENT EXTRACTION** *JOURNAL OF SUPRAMOLECULAR STRUCTURE*
Brown, S., Levinson, W., Spudich, J. A.
1976; 5 (2): 119-130
- **First-in-class drug candidate (MPH-220) efficiently improves spastic gait disorders by selective inhibition of fast skeletal muscle myosin-2**
Gyimesi, M., Horvath, A. I., Turos, D., Suthar, S., Penzes, M., Canon, L., Kikutu, C., Ruppel, K., Trivedi, D. V., Spudich, J. A., Rauscher, A. A., Kovacs, M., Komoly, et al
CELL PRESS.2022: 291A
- **Understanding the molecular basis of HCM-causing mutations in cardiac myosin and cardiac myosin binding protein-C**
Pathak, D., Nandwani, N., Ruppel, K., Spudich, J. A.
CELL PRESS.2022: 255A
- **Allosteric destabilization of the super-relaxed state of cardiac myosin by hypertrophic cardiomyopathy-causing mutations**
Nandwani, N., Bhowmik, D., Dawood, A., Ruppel, K., Spudich, J. A.
CELL PRESS.2022: 292A

- **Altered Cardiac Energetics and Mitochondrial Dysfunction in Hypertrophic Cardiomyopathy.** *Circulation*
Ranjbarvaziri, S., Kooiker, K. B., Ellenberger, M., Fajardo, G., Zhao, M., Vander Roest, A. S., Woldeyes, R. A., Koyano, T. T., Fong, R., Ma, N., Tian, L., Traber, G. M., Chan, et al
2021
- **Nanomechanical Phenotypes in Cardiac Myosin-Binding Protein C Mutants That Cause Hypertrophic Cardiomyopathy.** *ACS nano*
Suay-Corredera, C., Pricolo, M. R., Velazquez-Carreras, D., Pathak, D., Nandwani, N., Pimenta-Lopes, C., Sanchez-Ortiz, D., Urrutia-Irazabal, I., Vilches, S., Dominguez, F., Frisso, G., Monserrat, L., Garcia-Pavia, et al
2021
- **Molecular Mechanisms and Cellular Models of Hypertrophic Cardiomyopathy: Insights from a Surprising Mutation**
Vander Roest, A. S., Liu, C., Kooiker, K. B., Morck, M. M., Pruitt, B., Campbell, K. S., Ruppel, K., Spudich, J. A., Bernstein, D.
CELL PRESS.2021: 253A
- **Hypertrophic Cardiomyopathy, a Disease of Altered Cardiac Energetics**
Ranjbarvaziri, S., Ellenberger, M., Kooiker, K., Fajardo, G., Zhao, M., Schroer, A., Woo, Y. Y., Ruppel, K. M., Spudich, J. A., Snyder, M., Contrepolis, K., Bernstein, D.
LIPPINCOTT WILLIAMS & WILKINS.2020
- **Study of Hcm Causing beta-Cardiac Myosin Mutations Located at Different Structurally Significant Regions of the Myosin-Head**
Bhowmik, D., Nandwani, N., Ruppel, K., Liu, C., Spudich, J. A.
CELL PRESS.2020: 435A
- **Functional Comparison of Homologous Mutations in Human Beta, Perinatal, and Embryonic Muscle Myosin Isoforms**
Karabina, A., Liu, C., Spudich, J. A., Leinwand, L. A.
CELL PRESS.2020: 433A
- **Uncovering the Molecular and Structural Basis of Hypertrophic Cardiomyopathy-Causing Mutations in Myosin and Myosin Binding Protein-C**
Nandwani, N., Trivedi, D. V., Sarkar, S. S., Morck, M., Ruppel, K., Spudich, J. A.
CELL PRESS.2020: 435A
- **Hypertrophic Cardiomyopathy Mutations With Opposite Effects on [latin sharp s]-myosin Biomechanics Show Similar Structural and Biomechanical Phenotypes in Human Induced Pluripotent Stem Cell Derived Cardiomyocytes (hipsc-cms)**
Schroer, A., Jung, G., Kooiker, K., Adhikari, A., Song Linda, Liu Chao, Ruppel, K., Wu Sean, Pruitt, B., Spudich, J., Bernstein, D.
LIPPINCOTT WILLIAMS & WILKINS.2019
- **Myosin motor domains carrying mutations implicated in early or late onset hypertrophic cardiomyopathy have similar properties.** *The Journal of biological chemistry*
Vera, C. D., Johnson, C. A., Walklate, J. n., Adhikari, A. n., Svicevic, M. n., Mijailovich, S. M., Combs, A. C., Langer, S. J., Ruppel, K. M., Spudich, J. A., Geeves, M. A., Leinwand, L. A.
2019
- **Dilated cardiomyopathy myosin mutants have reduced force-generating capacity** *JOURNAL OF BIOLOGICAL CHEMISTRY*
Ujfalusi, Z., Vera, C. D., Mijailovich, S. M., Svicevic, M., Yu, E., Kawana, M., Ruppel, K. M., Spudich, J. A., Geeves, M. A., Leinwand, L. A.
2018; 293 (23): 9017–29
- **Mechanobiology of Myosin Mutations and Myofibril Remodeling in iPSC-Cardiomyocytes**
Schroer, A., Kooiker, K., Adhikari, A., Ruppel, K., Bernstein, D., Spudich, J., Pruitt, B.
CELL PRESS.2018: 496A–497A
- **A Molecular Approach to Understand the Super-Relaxed State of Myosin Observed in Cardiac Muscle**
Sarkar, S. S., Trivedi, D. V., Morck, M. M., Adhikari, A. S., Ruppel, K. M., Spudich, J. A.
CELL PRESS.2018: 141A
- **Impact of Hypertrophic Cardiomyopathy Mutations and the Role of Myosin Binding Protein-C on the Sequestered State of Myosin**
Trivedi, D. V., Sarkar, S. S., Morck, M. M., Adhikari, A. A., Ruppel, K. M., Spudich, J. A.
CELL PRESS.2018: 317A
- **Controlling Cardiac Contractility at the Single Molecule Level**
Liu, C., Song, D. L., Kawana, M., Ruppel, K. M., Spudich, J. A.

CELL PRESS.2018: 37A

- **SETD3 is an actin histidine methyltransferase that prevents primary dystocia.** *Nature*
Wilkinson, A. W., Diep, J. n., Dai, S. n., Liu, S. n., Ooi, Y. S., Song, D. n., Li, T. M., Horton, J. R., Zhang, X. n., Liu, C. n., Trivedi, D. V., Ruppel, K. M., Vilches-Moure, et al
2018
- **Molecular mechanisms and structural features of cardiomyopathy-causing troponin T mutants in the tropomyosin overlap region** *PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA*
Gangadharan, B., Sunitha, M. S., Mukherjee, S., Chowdhury, R., Haque, F., Sekar, N., Sowdhamini, R., Spudich, J. A., Mercer, J. A.
2017; 114 (42): 11115–20
- **Biophysical properties of human β -cardiac myosin with converter mutations that cause hypertrophic cardiomyopathy.** *Science advances*
Kawana, M., Sarkar, S. S., Sutton, S., Ruppel, K. M., Spudich, J. A.
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- **How to Measure Load-Dependent Kinetics of Individual Motor Molecules Without a Force-Clamp.** *Methods in enzymology*
Sung, J., Mortensen, K. I., Spudich, J. A., Flyvbjerg, H.
2017; 582: 1-29
- **Early-Onset Hypertrophic Cardiomyopathy Mutations Significantly Increase the Velocity, Force, and Actin-Activated ATPase Activity of Human beta-Cardiac Myosin** *CELL REPORTS*
Adhikari, A. S., Kooiker, K. B., Sarkar, S. S., Liu, C., Bernstein, D., Spudich, J. A., Ruppel, K. M.
2016; 17 (11): 2857-2864
- **How to Measure Separations and Angles Between Intramolecular Fluorescent Markers.** *Methods in enzymology*
Mortensen, K. I., Sung, J., Spudich, J. A., Flyvbjerg, H.
2016; 581: 147-185
- **Effects of hypertrophic and dilated cardiomyopathy mutations on power output by human β -cardiac myosin.** *The Journal of experimental biology*
Spudich, J. A., Aksel, T., Bartholomew, S. R., Nag, S., Kawana, M., Yu, E. C., Sarkar, S. S., Sung, J., Sommese, R. F., Sutton, S., Cho, C., Adhikari, A. S., Taylor, et al
2016; 219 (Pt 2): 161-7
- **Optimized measurements of separations and angles between intra-molecular fluorescent markers** *NATURE COMMUNICATIONS*
Mortensen, K. I., Sung, J., Flyvbjerg, H., Spudich, J. A.
2015; 6
- **Contractility parameters of human β -cardiac myosin with the hypertrophic cardiomyopathy mutation R403Q show loss of motor function.** *Science advances*
Nag, S., Sommese, R. F., Ujfalusi, Z., Combs, A., Langer, S., Sutton, S., Leinwand, L. A., Geeves, M. A., Ruppel, K. M., Spudich, J. A.
2015; 1 (9)
- **A mitochondria-anchored isoform of the actin-nucleating spire protein regulates mitochondrial division** *ELIFE*
Manor, U., Bartholomew, S., Golani, G., Christenson, E., Kozlov, M., Higgs, H., Spudich, J., Lippincott-Schwartz, J.
2015; 4
- **Mechanical coordination in motor ensembles revealed using engineered artificial myosin filaments** *NATURE NANOTECHNOLOGY*
Hariadi, R. F., Sommese, R. F., Adhikari, A. S., Taylor, R. E., Sutton, S., Spudich, J. A., Sivaramakrishnan, S.
2015; 10 (8): 696-700
- **Harmonic force spectroscopy measures load-dependent kinetics of individual human beta-cardiac myosin molecules** *NATURE COMMUNICATIONS*
Sung, J., Nag, S., Mortensen, K. I., Vestergaard, C. L., Sutton, S., Ruppel, K., Flyvbjerg, H., Spudich, J. A.
2015; 6
- **Ensemble Force Changes that Result from Human Cardiac Myosin Mutations and a Small-Molecule Effector** *CELL REPORTS*
Aksel, T., Yu, E. C., Sutton, S., Ruppel, K. M., Spudich, J. A.
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