Stanford



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)
- Douglass M. and Nola Leishman Professor of Cardiovascular Disease, Stanford University School of Medicine, (1990- present)
- Professor, Department of Biochemistry, Stanford University School of Medicine, (1992-2022)
- Chairman, Department of Biochemistry, Stanford University School of Medicine, (1994-1998)
- Co-Founder and first Director, Bio-X Interdisciplinary Program, Stanford University, (1998-2002)
- Co-Founder, Cytokinetics, Inc, (1998- present)
- Co-Founder, MyoKardia, Inc., (2012- present)
- Co-Founder & CEO, Kainomyx, Inc., (2019-2024)
- Professor, Department of Biochemistry, emeritus, Stanford University School of Medicine, (2022- present)
- Co-Founder & Executive Chairman, Kainomyx Inc., (2024- 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 ß-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 ß-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

STANFORD ADVISEES

Postdoctoral Faculty Sponsor

Sebastian Duno Miranda, Anne Snyder

GRADUATE AND FELLOWSHIP PROGRAM AFFILIATIONS

- Biochemistry (PhD Program)

- Biophysics (PhD Program)

Publications

PUBLICATIONS

- **Structure-Function Relationship as the Foundation for Cardiac Myosin Inhibition in Hypertrophic Cardiomyopathy.** *Circulation*
  Kawana, M., Ruppel, K. M., Spudich, J. A.
  2026

- **Antimalarial cytoskeletal targeting with broad apicomplexan activity** *PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA*
  Trivedi, D. V., Karabina, A., Jiang, T., Chen, D., Mittal, N., Huijs, T., van der Laak, R., Jeon, J. H., Yeo, T., Moussaoui, D., Robblee, J. P., Akey, M., Freese, et al
  2026; 123 (30): e2608709123

- **Mavacamten shows broad benefit in human and mouse models of MYBPC3-related hypertrophic cardiomyopathy.** *Nature cardiovascular research*
  Sen-Martín, L., Fernández-Trasancos, Á., López-Unzu, M. Á., Bak, A., Zafra-Castellano, M., Srikantharajah, R., Pathak, D., Klotz, A., Kuehn, M. N., Ferrarini, A., Labrador-Cantarero, V., Sánchez-Ortiz, D., Pricolo, et al
  2026

- **Exploring the dynamics of β-cardiac myosin auto-inhibited state to predict the development of inherited cardiomyopathies**
  Auguin, D., Lannes, L., Kikuti, C. M., Juille, M., Ayoub, N., Ruppel, K., Spudich, J. A., Robert-Paganin, J., Houdusse, A. M.
  CELL PRESS.2026: 38a

- **Hypertrophic cardiomyopathy mutations Y115H and E497D disrupt the folded-back state of human β-cardiac myosin allosterically.** *Nature communications*
  Nandwani, N., Bhowmik, D., Glaser, C., Childers, M. C., Goluguri, R. R., Dawood, A., Regnier, M., Houdusse, A., Spudich, J. A., Ruppel, K. M.
  2025; 16 (1): 8751

- **A FRET assay to monitor different structural states of human β-cardiac myosin including the interacting-heads motif.** *Proceedings of the National Academy of Sciences of the United States of America*
  Goluguri, R. R., Guhathakurta, P., Nandwani, N., Dawood, A., Yokota, S., Roopnarine, O., Thomas, D. D., Ruppel, K. M., Spudich, J. A.
  2025; 122 (34): e2504562122

- **Functional assessment of recombinant human alpha-cardiac myosin and the effect of atrial arrhythmia mutations**
  Pawar, A. D., Yokota, S. C., Spudich, J. A., Ruppel, K., Kawana, M.
  CELL PRESS.2025: 615A

- **From amoeboid myosin to unique targeted medicines for a genetic cardiac disease.** *Frontiers in physiology*
  Spudich, J. A.
  2024; 15: 1496569

- **Reassessing the unifying hypothesis for hypercontractility caused by myosin mutations in hypertrophic cardiomyopathy.** *The EMBO journal*
  Spudich, J. A., Nandwani, N., Robert-Paganin, J., Houdusse, A., Ruppel, K. M.
  2024

- **One must reconstitute the functions of interest from purified proteins.** *Frontiers in physiology*
  Spudich, J. A.
  2024; 15: 1390186

- **Mavacamten, a precision medicine for hypertrophic cardiomyopathy: From a motor protein to patients.** *Science advances*
  Nag, S., Gollapudi, S. K., Del Rio, C. L., Spudich, J. A., McDowell, R.
  2023; 9 (30): eabo7622

- **Cryo-EM structure of the folded-back state of human beta-cardiac myosin**
  Grinzato, A., Auguin, D., Kikuti, C., Nandwani, N., Moussaoui, D., Pathak, D., Kandiah, E., Ruppel, K., Spudich, J. A., Houdusse, A. M., Robert-Paganin, J.
  CELL PRESS.2023: 258A-259A

- **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

- **beta-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 beta-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 ß-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
- **The Dilated Cardiomyopathy E525K β-Myosin Mutation Causes Hypocontractility in Cardiomyocytes Without Altering Crossbridge Cycling.** *bioRxiv : the preprint server for biology*
  Robeson, K. Z., McMillen, T. S., Kooiker, K., Kao, K. Y., Fruebis, K., Geeves, M. A., Wescott, A. P., Soriano, R., Goldstein, A. J., Childers, M. C., Goluguri, R. R., Pathak, D., Sniadecki, et al
  2026
- **The hypertrophic cardiomyopathy mutation G768R makes cardiac myosin a high duty ratio motor.** *bioRxiv : the preprint server for biology*
  Pathak, D., Ozturk, T. N., Dawood, A., Childers, M. C., Regnier, M., Spudich, J. A., Liu, C., Ruppel, K. M.
  2026
- **Dynamics of the β-cardiac myosin auto-inhibited state explain cardiomyopathy pathogenesis.** *Nature communications*
  Auguin, D., Lannes, L., Kikuti, C., Ayoub, N., Juillé, M., Réty, S., Nandwani, N., Pathak, D., Ruppel, K. M., Spudich, J. A., Robert-Paganin, J., Houdusse, A.
  2026
- **The hypertrophic cardiomyopathy myosin variant G256E prolongs cardiac muscle relaxation via altered nucleotide handling.** *Communications chemistry*
  Kao, K. Y., Childers, M. C., Pathak, D., Goluguri, R. R., McMillen, T. S., Powers, J. D., Ruppel, K. M., Spudich, J. A., Regnier, M.
  2026

- **The G256E HCM mutation prolongs relaxation via altered nucleotide handling**
  Kao, K. Y., Childers, M. C., Pathak, D., Goluguri, R., McMillen, T. S., Ruppel, K. M., Spudich, J. A., Regnier, M.
  CELL PRESS.2026: 90a
- **A FRET assay to monitor different structural states of β-cardiac myosin including the interacting-heads motif**
  Goluguri, R., Guhathakurta, P., Nandwani, N., Dawood, A., Yokota, S., Roopnarine, O., Thomas, D. D., Ruppel, K., Spudich, J. A.
  CELL PRESS.2026: 8a
- **The hypertrophic cardiomyopathy mutation G768R makes cardiac myosin a high duty ratio motor**
  Pathak, D., Ozturk, T. N., Childers, M. C., Regnier, M., Liu, C., Spudich, J. A., Ruppel, K.
  CELL PRESS.2026: 90a
- **The G256E HCM mutation prolongs cardiac muscle relaxation via altered nucleotide handling.** *Research square*
  Regnier, M., Kao, K., Childers, M., Pathak, D., Goluguri, R. R., McMillen, T., Ruppel, K., Spudich, J.
  2025
- **Michael Patrick Sheetz, 1946-2025, a devotee of and major contributor to membrane and cytoskeletal biology.** *Molecular biology of the cell*
  Kenney, L., Vale, R., Spudich, J.
  2025: mbcE25050208
- **A FRET assay to quantitate levels of the human β-cardiac myosin interacting heads motif based on its near-atomic resolution structure.** *bioRxiv : the preprint server for biology*
  Goluguri, R. R., Guhathakurta, P., Nandwani, N., Dawood, A., Yakota, S., Roopnarine, O., Thomas, D. D., Spudich, J. A., Ruppel, K. M.
  2024
- **Motility Assay to Probe the Calcium Sensitivity of Myosin and Regulated Thin Filaments.** *Methods in molecular biology (Clifton, N.J.)*
  Liu, C., Ruppel, K. M., Spudich, J. A.
  2024; 2735: 169-189
- **Incomplete-penetrant hypertrophic cardiomyopathy MYH7 G256E mutation causes hypercontractility and elevated mitochondrial respiration.** *Proceedings of the National Academy of Sciences of the United States of America*
  Lee, S., Vander Roest, A. S., Blair, C. A., Kao, K., Bremner, S. B., Childers, M. C., Pathak, D., Heinrich, P., Lee, D., Chirikian, O., Mohran, S. E., Roberts, B., Smith, et al
  2024; 121 (19): e2318413121
- **Homologous mutations in human β, embryonic, and perinatal muscle myosins have divergent effects on molecular power generation.** *Proceedings of the National Academy of Sciences of the United States of America*
  Liu, C., Karabina, A., Meller, A., Bhattacharjee, A., Agostino, C. J., Bowman, G. R., Ruppel, K. M., Spudich, J. A., Leinwand, L. A.
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- **Cryo-electron tomography reveals the structural diversity of cardiac proteins in their cellular context.** *bioRxiv : the preprint server for biology*
  Woldeyes, R. A., Nishiga, M., Vander Roest, A. S., Engel, L., Giri, P., Montenegro, G. C., Wu, A. C., Dunn, A. R., Spudich, J. A., Bernstein, D., Schmid, M. F., Wu, J. C., Chiu, et al
  2023
- **Homologous mutations in β, embryonic, and perinatal muscle myosins have divergent effects on molecular power generation.** *bioRxiv : the preprint server for biology*
  Liu, C., Karabina, A., Meller, A., Bhattacharjee, A., Agostino, C. J., Bowman, G. R., Ruppel, K. M., Spudich, J. A., Leinwand, L. A.
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- **Cryo-EM structure of the folded-back state of human β-cardiac myosin.** *Nature communications*
  Grinzato, A., Auguin, D., Kikuti, C., Nandwani, N., Moussaoui, D., Pathak, D., Kandiah, E., Ruppel, K. M., Spudich, J. A., Houdusse, A., Robert-Paganin, J.
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