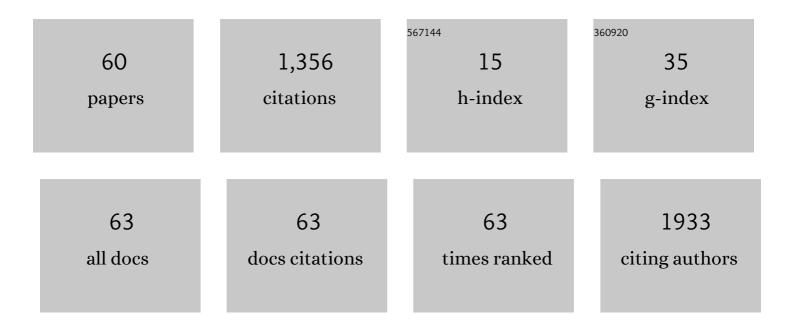
Girish C Melkani

List of Publications by Year in descending order

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CIDISH C MELKANI

#	Article	IF	CITATIONS
1	Manipulating Levels of Stressâ€Response Proteins in a Drosophila Model of Myosinâ€Based Inclusion Body Myopathy 3 Worsens Muscle Dysfunction. FASEB Journal, 2020, 34, 1-1.	0.2	0
2	Time-restricted feeding restores muscle function in Drosophila models of obesity and circadian-rhythm disruption. Nature Communications, 2019, 10, 2700.	5.8	85
3	Time-Restricted Eating to Prevent and Manage Chronic Metabolic Diseases. Annual Review of Nutrition, 2019, 39, 291-315.	4.3	239
4	Suppression of myopathic lamin mutations by muscle-specific activation of <i>AMPK</i> and modulation of downstream signaling. Human Molecular Genetics, 2019, 28, 351-371.	1.4	16
5	Increasing autophagy and blocking Nrf2 suppress laminopathyâ€induced ageâ€dependent cardiac dysfunction and shortened lifespan. Aging Cell, 2018, 17, e12747.	3.0	33
6	Prolonged cross-bridge binding triggers muscle dysfunction in a Drosophila model of myosin-based hypertrophic cardiomyopathy. ELife, 2018, 7, .	2.8	26
7	Timeâ€restricted feeding for prevention and treatment of cardiometabolic disorders. Journal of Physiology, 2017, 595, 3691-3700.	1.3	117
8	A R146N Hypertrophic Cardiomyopathy Myosin Mutation Disrupts Myosin Function, Myofibrillar Structure, and Cardiac Contraction in Drosophila. Biophysical Journal, 2017, 112, 264a.	0.2	0
9	X-Ray Crystallography Structures of Drosophila Striated Muscle MyosinÂll. Biophysical Journal, 2017, 112, 266a.	0.2	0
10	TRiC/CCT chaperonins are essential for maintaining myofibril organization, cardiac physiological rhythm, and lifespan. FEBS Letters, 2017, 591, 3447-3458.	1.3	15
11	A Drosophila model of dominant inclusion body myopathy 3 shows diminished myosin kinetics that reduce muscle power and yield myofibrillar defects. DMM Disease Models and Mechanisms, 2017, 10, 761-771.	1.2	5
12	Huntington's Disease-Induced Cardiac Disorders Affect Multiple Cellular Pathways. , 2016, 2, 325-338.		6
13	Using Drosophila as an integrated model to study mild repetitive traumatic brain injury. Scientific Reports, 2016, 6, 25252.	1.6	76
14	A Restrictive Cardiomyopathy Mutation in an Invariant Proline at the Myosin Head/Rod Junction Enhances Head Flexibility and Function, Yielding Muscle Defects in Drosophila. Journal of Molecular Biology, 2016, 428, 2446-2461.	2.0	8
15	The Relay/Converter Interface Influences Hydrolysis of ATP by Skeletal Muscle Myosin II. Journal of Biological Chemistry, 2016, 291, 1763-1773.	1.6	18
16	The R146N and R249Q Myosin Mutations Disrupt Motor Function and Myofibrillar Structure and cause Cardiomyopathy in Drosophila. Biophysical Journal, 2015, 108, 445a.	0.2	0
17	A Drosophila Model of Myosin-Based Inclusion Body Myopathy Type 3: Effects on Muscle Structure, Muscle Function and Aggregated Protein Profiles. Biophysical Journal, 2015, 108, 304a.	0.2	1
18	Time-restricted feeding attenuates age-related cardiac decline in <i>Drosophila</i> . Science, 2015, 347, 1265-1269.	6.0	223

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19	A Failure to Communicate. Journal of Biological Chemistry, 2015, 290, 29270-29280.	1.6	8
20	Mapping Interactions between Myosin Relay and Converter Domains That Power Muscle Function. Journal of Biological Chemistry, 2014, 289, 12779-12790.	1.6	14
21	The UNC-45 Myosin Chaperone. International Review of Cell and Molecular Biology, 2014, 313, 103-144.	1.6	56
22	Drosophila as a potential model to ameliorate mutant Huntington-mediated cardiac amyloidosis. Rare Diseases (Austin, Tex), 2014, 2, e968003.	1.8	4
23	X-Ray Structure Determination of the First Insect Skeletal Muscle MyosinÂll. Biophysical Journal, 2014, 106, 45a.	0.2	0
24	Myosin Storage Myopathy Mutations Disrupt Myofibrillar Assembly/ Stability and Cause Progressive Muscle Degeneration in a Drosophila Model. Biophysical Journal, 2014, 106, 777a.	0.2	0
25	Defining Myosin Relay Domain Interactions with the Converter Domain and with the SH1-SH2 Helix Region and their Significance in Muscle Contraction. Biophysical Journal, 2013, 104, 307a.	0.2	Ο
26	Alleviation of Skeletal Muscle Defects Induced by Huntington's Disease-causing Amyloid by Modulating TOR Pathway in a Drosophila Model. Biophysical Journal, 2013, 104, 483a.	0.2	0
27	Exploration and Suppression of Tau-Induced Cardiac and Skeletal Muscle Defects in a Drosophila Model. Biophysical Journal, 2013, 104, 486a.	0.2	1
28	Huntington's Disease Induced Cardiac Amyloidosis Is Reversed by Modulating Protein Folding and Oxidative Stress Pathways in the Drosophila Heart. PLoS Genetics, 2013, 9, e1004024.	1.5	75
29	Interaction of oxidized chaperonin GroEL with an unfolded protein at low temperatures. Bioscience Reports, 2012, 32, 299-303.	1.1	1
30	Expression of the inclusion body myopathy 3 mutation in Drosophila depresses myosin function and stability and recapitulates muscle inclusions and weakness. Molecular Biology of the Cell, 2012, 23, 2057-2065.	0.9	15
31	Transgenic expression and purification of myosin isoforms using the Drosophila melanogaster indirect flight muscle system. Methods, 2012, 56, 25-32.	1.9	10
32	Alternative Relay and Converter Domains Tune Native Muscle Myosin Isoform Function in Drosophila. Journal of Molecular Biology, 2012, 416, 543-557.	2.0	17
33	Interaction Between the Relay Loop and the SH1-SH2 Helix Region in Drosophila Muscle Myosin is Essential for Normal Motor Function, Myofibril Stability and Muscle Contraction. Biophysical Journal, 2012, 102, 148a-149a.	0.2	Ο
34	Kinetic Characterization of Converter and Relay Loop Domain Interaction in Drosophila Myosin Sub-Fragment 1. Biophysical Journal, 2012, 102, 149a.	0.2	0
35	A Method for the Transgenic Expression and Purification of Skeletal Muscle Myosin II Isoforms using Drosophila Melanogaster. Biophysical Journal, 2012, 102, 149a.	0.2	0
36	Myosin Storage Myopathy Mutations Cause Age Dependent Muscle Degeneration and Cardiac Dysfunction in a Drosophila Model. Biophysical Journal, 2012, 102, 253a-254a.	0.2	0

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37	Exploration and Suppression of Cardiac Amyloidosis Induced by Huntington's Disease-Causing Amyloid in the Drosophila Heart Model. Biophysical Journal, 2012, 102, 351a.	0.2	0
38	Cardiac amyloidosis and its suppression in a Huntington's disease model in the Drosophila heart. FASEB Journal, 2012, 26, 1135.6.	0.2	0
39	Transgenic Expression and Purification of Myosin Isoforms Using the Drosophila melanogaster Indirect Flight Muscle System. FASEB Journal, 2012, 26, lb204.	0.2	0
40	Drosophila as a Model for Amyloid Induced Cardiac Dysfunction. Biophysical Journal, 2011, 100, 294a.	0.2	0
41	The E706K IBM3 Myosin Mutation Depresses the Chemomechanical Properties and Increases the Lability of the Molecular Motor. Biophysical Journal, 2011, 100, 129a.	0.2	0
42	The UNC-45 Chaperone Is Critical for Establishing Myosin-Based Myofibrillar Organization and Cardiac Contractility in the Drosophila Heart Model. PLoS ONE, 2011, 6, e22579.	1.1	44
43	Two Drosophila Myosin Transducer Mutants with Distinct Cardiomyopathies Have Divergent ADP and Actin Affinities. Journal of Biological Chemistry, 2011, 286, 28435-28443.	1.6	12
44	<i>Drosophila</i> UNC-45 accumulates in embryonic blastoderm and in muscles, and is essential for muscle myosin stability. Journal of Cell Science, 2011, 124, 699-705.	1.2	36
45	Drosophila UNC-45 accumulates in embryonic blastoderm and in muscles, and is essential for muscle myosin stability. Development (Cambridge), 2011, 138, e1-e1.	1.2	0
46	UNC-45 Knock-Down in Drosophila Heart Targets Myosin Accumulation and Yields Severe Myofibrillar Disarray and Cardiac Dysfunction. Biophysical Journal, 2010, 98, 7a.	0.2	0
47	A Single Amino Acid Mutation in the Drosophila Myosin SH1 Domain Severely Affects Muscle Function, Myofibril Structure, Myosin Enzymatic Activity, and Actin Sliding Velocity. Biophysical Journal, 2010, 98, 144a.	0.2	0
48	Mutating the Converter–Relay Interface of Drosophila Myosin Perturbs ATPase Activity, Actin Motility, Myofibril Stability and Flight Ability. Journal of Molecular Biology, 2010, 398, 625-632.	2.0	13
49	Drosophila UNC-45 prevents heat-induced aggregation of skeletal muscle myosin and facilitates refolding of citrate synthase. Biochemical and Biophysical Research Communications, 2010, 396, 317-322.	1.0	32
50	Converter Domain Residue R759 Interaction with Relay Loop Residue N509 in Drosophila Muscle Myosin is Critical for Motor Function, Myofibril Stability and Flight Ability. Biophysical Journal, 2010, 98, 215a.	0.2	0
51	Alternative Exon 9-Encoded Relay Domains Affect More than One Communication Pathway in the Drosophila Myosin Head. Journal of Molecular Biology, 2009, 389, 707-721.	2.0	18
52	Kinetics Of Two Single Point Mutants Of Drosophila Myosin S1. Biophysical Journal, 2009, 96, 496a-497a.	0.2	0
53	Divalent cations stabilize GroEL under conditions of oxidative stress. Biochemical and Biophysical Research Communications, 2008, 368, 625-630.	1.0	4
54	Protection of GroEL by its methionine residues against oxidation by hydrogen peroxide. Biochemical and Biophysical Research Communications, 2006, 347, 534-539.	1.0	8

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55	αB-Crystallin Maintains Skeletal Muscle Myosin Enzymatic Activity and Prevents its Aggregation under Heat-shock Stress. Journal of Molecular Biology, 2006, 358, 635-645.	2.0	54
56	On the chaperonin activity of GroEL at heat-shock temperature. International Journal of Biochemistry and Cell Biology, 2005, 37, 1375-1385.	1.2	12
57	Oxidized GroEL can function as a chaperonin. Frontiers in Bioscience - Landmark, 2004, 9, 724.	3.0	8
58	Hydrogen peroxide induces the dissociation of GroEL into monomers that can facilitate the reactivation of oxidatively inactivated rhodanese. International Journal of Biochemistry and Cell Biology, 2004, 36, 505-518.	1.2	11
59	The ATPase activity of GroEL is supported at high temperatures by divalent cations that stabilize its structure. BioMetals, 2003, 16, 479-484.	1.8	22
60	GroEL interacts transiently with oxidatively inactivated rhodanese facilitating its reactivation. Biochemical and Biophysical Research Communications, 2002, 294, 893-899.	1.0	9