Girish C Melkani

List of Publications by Year
in descending order

60 papers	1,356 citations	567144 15 h-index	360920 35 g-index
63 all docs	63 docs citations	63 times ranked	1933 citing authors

#	Article	IF	Citations
1	Time-Restricted Eating to Prevent and Manage Chronic Metabolic Diseases. Annual Review of Nutrition, 2019, 39, 291-315.	4.3	239
2	Time-restricted feeding attenuates age-related cardiac decline in Drosophila. Science, 2015, 347, 1265-1269.	6.0	223
3	Timeâ€restricted feeding for prevention and treatment of cardiometabolic disorders. Journal of Physiology, 2017, 595, 3691-3700.	1.3	117
4	Time-restricted feeding restores muscle function in Drosophila models of obesity and circadian-rhythm disruption. Nature Communications, 2019, 10, 2700.	5.8	85
5	Using Drosophila as an integrated model to study mild repetitive traumatic brain injury. Scientific Reports, 2016, 6, 25252.	1.6	76
6	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
7	The UNC-45 Myosin Chaperone. International Review of Cell and Molecular Biology, 2014, 313, 103-144.	1.6	56
8	Î±B-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
9	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
10	DrosophilaUNC-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
11	Increasing autophagy and blocking Nrf2 suppress laminopathyâ€induced ageâ€dependent cardiac dysfunction and shortened lifespan. Aging Cell, 2018, 17, e12747.	3.0	33
12	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
13	Prolonged cross-bridge binding triggers muscle dysfunction in a Drosophila model of myosin-based hypertrophic cardiomyopathy. ELife, 2018, 7, .	2.8	26
14	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
15	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
16	The Relay/Converter Interface Influences Hydrolysis of ATP by Skeletal Muscle Myosin II. Journal of Biological Chemistry, 2016, 291, 1763-1773.	1.6	18
17	Alternative Relay and Converter Domains Tune Native Muscle Myosin Isoform Function in Drosophila. Journal of Molecular Biology, 2012, 416, 543-557.	2.0	17
18	Suppression of myopathic lamin mutations by muscle-specific activation ofAMPKand modulation of downstream signaling. Human Molecular Genetics, 2019, 28, 351-371.	1.4	16

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19	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
20	TRiC/CCT chaperonins are essential for maintaining myofibril organization, cardiac physiological rhythm, and lifespan. FEBS Letters, 2017, 591, 3447-3458.	1.3	15
21	Mapping Interactions between Myosin Relay and Converter Domains That Power Muscle Function. Journal of Biological Chemistry, 2014, 289, 12779-12790.	1.6	14
22	Mutating the Converterâ€“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
23	On the chaperonin activity of GroEL at heat-shock temperature. International Journal of Biochemistry and Cell Biology, 2005, 37, 1375-1385.	1.2	12
24	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
25	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
26	Transgenic expression and purification of myosin isoforms using the Drosophila melanogaster indirect flight muscle system. Methods, 2012, 56, 25-32.	1.9	10
27	GroEL interacts transiently with oxidatively inactivated rhodanese facilitating its reactivation. Biochemical and Biophysical Research Communications, 2002, 294, 893-899.	1.0	9
28	Oxidized GroEL can function as a chaperonin. Frontiers in Bioscience - Landmark, 2004, 9, 724.	3.0	8
29	Protection of GroEL by its methionine residues against oxidation by hydrogen peroxide. Biochemical and Biophysical Research Communications, 2006, 347, 534-539.	1.0	8
30	A Failure to Communicate. Journal of Biological Chemistry, 2015, 290, 29270-29280.	1.6	8
31	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
32	Huntingtonâ€™s Disease-Induced Cardiac Disorders Affect Multiple Cellular Pathways. , 2016, 2, 325-338.		6
33	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
34	Divalent cations stabilize GroEL under conditions of oxidative stress. Biochemical and Biophysical Research Communications, 2008, 368, 625-630.	1.0	4
35	Drosophila as a potential model to ameliorate mutant Huntington-mediated cardiac amyloidosis. Rare Diseases (Austin, Tex), 2014, 2, e968003.	1.8	4
36	Interaction of oxidized chaperonin GroEL with an unfolded protein at low temperatures. Bioscience Reports, 2012, 32, 299-303.	1.1	1

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37	Exploration and Suppression of Tau-Induced Cardiac and Skeletal Muscle Defects in a Drosophila Model. Biophysical Journal, 2013, 104, 486a.	0.2	1
38	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
39	Kinetics Of Two Single Point Mutants Of Drosophila Myosin S1. Biophysical Journal, 2009, 96, 496a-497a.	0.2	0
40	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
41	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
42	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
43	Drosophila as a Model for Amyloid Induced Cardiac Dysfunction. Biophysical Journal, 2011, 100, 294a.	0.2	0
44	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
45	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	0
46	Kinetic Characterization of Converter and Relay Loop Domain Interaction in Drosophila Myosin Sub-Fragment 1. Biophysical Journal, 2012, 102, 149a.	0.2	0
47	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
48	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
49	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
50	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	0
51	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
52	X-Ray Structure Determination of the First Insect Skeletal Muscle MyosinÂII. Biophysical Journal, 2014, 106, 45a.	0.2	0
53	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
54	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

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55	A R146N Hypertrophic Cardiomyopathy Myosin Mutation Disrupts Myosin Function, Myofibrillar Structure, and Cardiac Contraction in Drosophila. Biophysical Journal, 2017, 112, 264a.	0.2	0
56	X-Ray Crystallography Structures of Drosophila Striated Muscle MyosinÂII. Biophysical Journal, 2017, 112, 266a.	0.2	0
57	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
58	Cardiac amyloidosis and its suppression in a Huntington's disease model in the Drosophila heart. FASEB Journal, 2012, 26, 1135.6.	0.2	0
59	Transgenic Expression and Purification of Myosin Isoforms Using the Drosophila melanogaster Indirect Flight Muscle System. FASEB Journal, 2012, 26, lb204.	0.2	0
60	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