James Shorter

List of Publications by Year
in descending order

175 papers	20,667 citations	13865 67 h-index	11939 134 g-index
190 all docs	190 docs citations	190 times ranked	16831 citing authors

#	Article	IF	Citations
1	Heterozygous variants of CLPB are a cause of severe congenital neutropenia. Blood, 2022, 139, 779-791.	1.4	25
2	Nuclear-Import Receptors Counter Deleterious Phase Transitions in Neurodegenerative Disease. Journal of Molecular Biology, 2022, 434, 167220.	4.2	13
3	TDP-43 represses cryptic exon inclusion in the FTDâ€“ALS gene UNC13A. Nature, 2022, 603, 124-130.	27.8	193
4	Poly(ADP-ribose) drives condensation of FUS via a transient interaction. Molecular Cell, 2022, 82, 969-985.e11.	9.7	41
5	AAA+ proteins: one motor, multiple ways to work. Biochemical Society Transactions, 2022, 50, 895-906.	3.4	13
6	Aggregates of TDP-43 protein spiral into view. Nature, 2022, 601, 29-30.	27.8	4
7	Flying under the radar: TMEM106B(120â€“254) fibrils break out in diverse neurodegenerative disorders. Cell, 2022, 185, 1290-1292.	28.9	3
8	Heterozygous frameshift variants in HNRNPA2B1 cause early-onset oculopharyngeal muscular dystrophy. Nature Communications, 2022, 13, 2306.	12.8	20
9	Developing RNA Therapeutics for TDPâ€43 Proteinopathy in ALS/FTD. FASEB Journal, 2022, 36, .	0.5	0
10	Increased Nuclear Localization of Engineered Hsp104 Variants Mitigates aS, FUS, and TDPâ€43 Toxicity in Yeast. FASEB Journal, 2022, 36, .	0.5	0
11	Identifying Therapeutic Inhibitors of TDP43 Phaseâ€Separation. FASEB Journal, 2022, 36, .	0.5	0
12	Sequestration of TDP-43²¹⁶⁻⁴¹⁴ Aggregates by Cytoplasmic Expression of the proSAAS Chaperone. ACS Chemical Neuroscience, 2022, 13, 1651-1665.	3.5	6
13	Elucidating the mechanism of potentiated Hsp104 NBD2 variants against proteotoxicity. FASEB Journal, 2022, 36, .	0.5	0
14	Developing therapeutic protein disaggregases for Neurodegenerative Disease. FASEB Journal, 2022, 36, .	0.5	0
15	Heat shock protein Grp78/BiP/HspA5 binds directly to TDP-43 and mitigates toxicity associated with disease pathology. Scientific Reports, 2022, 12, 8140.	3.3	12
16	Sexually dimorphic RNA helicases DDX3X and DDX3Y differentially regulate RNA metabolism through phase separation. Molecular Cell, 2022, 82, 2588-2603.e9.	9.7	24
17	Combating deleterious phase transitions in neurodegenerative disease. Biochimica Et Biophysica Acta - Molecular Cell Research, 2021, 1868, 118984.	4.1	52
18	Tau heckles speckles: A pathogenic mechanism in tauopathy?. Neuron, 2021, 109, 1585-1587.	8.1	3

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19	(Dis)Solving the problem of aberrant protein states. DMM Disease Models and Mechanisms, 2021, 14, .	2.4	23
20	Open Access: A Role for p53 in c9ALS/FTD?. Trends in Genetics, 2021, 37, 404-406.	6.7	3
21	Biochemical Timekeeping Via Reentrant Phase Transitions. Journal of Molecular Biology, 2021, 433, 166794.	4.2	22
22	Higher-order organization of biomolecular condensates. Open Biology, 2021, 11, 210137.	3.6	96
23	Characterization of HNRNPA1 mutations defines diversity in pathogenic mechanisms and clinical presentation. JCI Insight, 2021, 6, .	5.0	38
24	FUS and TDP-43 Phases in Health and Disease. Trends in Biochemical Sciences, 2021, 46, 550-563.	7.5	154
25	DAXX represents a new type of protein-folding enabler. Nature, 2021, 597, 132-137.	27.8	54
26	TDP-43 condensation properties specify its RNA-binding and regulatory repertoire. Cell, 2021, 184, 4680-4696.e22.	28.9	121
27	N-alpha-acetylation of Huntingtin protein increases its propensity to aggregate. Journal of Biological Chemistry, 2021, 297, 101363.	3.4	9
28	Loss of Dynamic RNA Interaction and Aberrant Phase Separation Induced by Two Distinct Types of ALS/FTD-Linked FUS Mutations. Molecular Cell, 2020, 77, 82-94.e4.	9.7	119
29	Switching Condensates: The CTD Code Goes Liquid. Trends in Biochemical Sciences, 2020, 45, 1-3.	7.5	16
30	Emerging small-molecule therapeutic approaches for amyotrophic lateral sclerosis and frontotemporal dementia. Bioorganic and Medicinal Chemistry Letters, 2020, 30, 126942.	2.2	31
31	TRIM11 Prevents and Reverses Protein Aggregation and Rescues a Mouse Model of Parkinsonâ€™s Disease. Cell Reports, 2020, 33, 108418.	6.4	39
32	Atomic Structures of Amyloid-Î² Oligomers Illuminate a Neurotoxic Mechanism. Trends in Neurosciences, 2020, 43, 740-743.	8.6	10
33	C9orf72 poly(GR) aggregation induces TDP-43 proteinopathy. Science Translational Medicine, 2020, 12, .	12.4	115
34	Supramolecular Mechanism of Viral Envelope Disruption by Molecular Tweezers. Journal of the American Chemical Society, 2020, 142, 17024-17038.	13.7	31
35	Nuclear Import Receptors Directly Bind to Arginine-Rich Dipeptide Repeat Proteins and Suppress Their Pathological Interactions. Cell Reports, 2020, 33, 108538.	6.4	69
36	ALS/FTLD-Linked Mutations in FUS Glycine Residues Cause Accelerated Gelation and Reduced Interactions with Wild-Type FUS. Molecular Cell, 2020, 80, 666-681.e8.	9.7	62

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37	Structural and mechanistic insights into Hsp104 function revealed by synchrotron X-ray footprinting. Journal of Biological Chemistry, 2020, 295, 1517-1538.	3.4	16
38	The clinical trial landscape in amyotrophic lateral sclerosisâ€"Past, present, and future. Medicinal Research Reviews, 2020, 40, 1352-1384.	10.5	61
39	The Sense of Targeting Nonsense-Mediated Decay in C9-ALS/FTD. Neuron, 2020, 106, 6-9.	8.1	1
40	Structural and kinetic basis for the regulation and potentiation of Hsp104 function. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 9384-9392.	7.1	16
41	Conformational plasticity of the ClpAP AAA+ protease couples protein unfolding and proteolysis. Nature Structural and Molecular Biology, 2020, 27, 406-416.	8.2	51
42	Just Took a DNA Test, Turns Out 100% Not That Phase. Molecular Cell, 2020, 78, 193-194.	9.7	10
43	Arginine-rich dipeptide-repeat proteins as phase disruptors in C9-ALS/FTD. Emerging Topics in Life Sciences, 2020, 4, 293-305.	2.6	26
44	The extent of Ssa1/Ssa2 Hsp70 chaperone involvement in nuclear protein quality control degradation varies with the substrate. Molecular Biology of the Cell, 2020, 31, 221-233.	2.1	18
45	Skd3 (human ClpB) is a potent mitochondrial protein disaggregase that is inactivated by 3-methylglutaconic aciduria-linked mutations. ELife, 2020, 9, .	6.0	44
46	Therapeutic genetic variation revealed in diverse Hsp104 homologs. ELife, 2020, 9, .	6.0	17
47	Karyopherin-Î²2 Inhibits and Reverses Aggregation and Liquid-liquid Phase Separation of the ALS/FTD-Associated Protein FUS. Bio-protocol, 2020, 10, e3725.	0.4	3
48	Expression and Purification of Recombinant Skd3 (Human ClpB) Protein and Tobacco Etch Virus (TEV) Protease from Escherichia coli. Bio-protocol, 2020, 10, e3858.	0.4	11
49	The molecular language of membraneless organelles. Journal of Biological Chemistry, 2019, 294, 7115-7127.	3.4	515
50	Mining Disaggregase Sequence Space to Safely Counter TDP-43, FUS, and Î±-Synuclein Proteotoxicity. Cell Reports, 2019, 28, 2080-2095.e6.	6.4	36
51	CRISPR-Cas9 Screens Identify the RNA Helicase DDX3X as a Repressor of C9ORF72 (GGGGCC)n Repeat-Associated Non-AUG Translation. Neuron, 2019, 104, 885-898.e8.	8.1	107
52	Engineered protein disaggregases mitigate toxicity of aberrant prion-like fusion proteins underlying sarcoma. Journal of Biological Chemistry, 2019, 294, 11286-11296.	3.4	31
53	Structural basis for substrate gripping and translocation by the ClpB AAA+ disaggregase. Nature Communications, 2019, 10, 2393.	12.8	88
54	Hsp104 and Potentiated Variants Can Operate as Distinct Nonprocessive Translocases. Biophysical Journal, 2019, 116, 1856-1872.	0.5	17

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55	Cytoplasmic TDP-43 De-mixing Independent of Stress Granules Drives Inhibition of Nuclear Import, Loss of Nuclear TDP-43, and Cell Death. Neuron, 2019, 102, 339-357.e7.	8.1	331
56	Hydrogen exchange reveals Hsp104 architecture, structural dynamics, and energetics in physiological solution. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 7333-7342.	7.1	22
57	AAA+ Protein-Based Technologies to Counter Neurodegenerative Disease. Biophysical Journal, 2019, 116, 1380-1385.	0.5	17
58	Phase separation of RNA-binding proteins in physiology and disease: An introduction to the JBC Reviews thematic series. Journal of Biological Chemistry, 2019, 294, 7113-7114.	3.4	39
59	Spiraling in Control: Structures and Mechanisms of the Hsp104 Disaggregase. Cold Spring Harbor Perspectives in Biology, 2019, 11, a034033.	5.5	77
60	RNA Binding Antagonizes Neurotoxic Phase Transitions of TDP-43. Neuron, 2019, 102, 321-338.e8.	8.1	365
61	Heterochromatin anomalies and double-stranded RNA accumulation underlie C9orf72 poly(PR) toxicity. Science, 2019, 363, .	12.6	181
62	TDP-43 shapeshifts to encipher FTD severity. Nature Neuroscience, 2019, 22, 3-5.	14.8	7
63	Structure of Calcarisporiella thermophila Hsp104 Disaggregase that Antagonizes Diverse Proteotoxic Misfolding Events. Structure, 2019, 27, 449-463.e7.	3.3	29
64	Therapeutic Dissolution of Aberrant Phases by Nuclear-Import Receptors. Trends in Cell Biology, 2019, 29, 308-322.	7.9	55
65	FUS Regulates Activity of MicroRNA-Mediated Gene Silencing. Molecular Cell, 2018, 69, 787-801.e8.	9.7	76
66	Amyloid assembly and disassembly. Journal of Cell Science, 2018, 131, .	2.0	138
67	Nuclear-Import Receptors Reverse Aberrant Phase Transitions of RNA-Binding Proteins with Prion-like Domains. Cell, 2018, 173, 677-692.e20.	28.9	376
68	Ubiquilin 2: Shuttling Clients Out of Phase?. Molecular Cell, 2018, 69, 919-921.	9.7	5
69	Protein Phase Separation: A New Phase in Cell Biology. Trends in Cell Biology, 2018, 28, 420-435.	7.9	1,439
70	3â€² UTRs in the Eye of the TIGER. Developmental Cell, 2018, 47, 544-546.	7.0	2
71	Poly(ADP-ribose) Engages the TDP-43 Nuclear-Localization Sequence to Regulate Granulo-Filamentous Aggregation. Biochemistry, 2018, 57, 6923-6926.	2.5	28
72	TDP-43 and RNA form amyloid-like myo-granules in regenerating muscle. Nature, 2018, 563, 508-513.	27.8	163

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73	Editorial: The Role of AAA+ Proteins in Protein Repair and Degradation. Frontiers in Molecular Biosciences, 2018, 5, 85.	3.5	12
74	Potentiating Hsp104 activity via phosphomimetic mutations in the middle domain. FEMS Yeast Research, 2018, 18, .	2.3	37
75	Enhancement of Ebola virus infection by seminal amyloid fibrils. Proceedings of the National Academy of Sciences of the United States of America, 2018, 115, 7410-7415.	7.1	21
76	Molecular Dissection of FUS Points at Synergistic Effect of Low-Complexity Domains in Toxicity. Cell Reports, 2018, 24, 529-537.e4.	6.4	74
77	Poly(ADP-Ribose) Prevents Pathological Phase Separation of TDP-43 by Promoting Liquid Demixing and Stress Granule Localization. Molecular Cell, 2018, 71, 703-717.e9.	9.7	309
78	Designer protein disaggregases to counter neurodegenerative disease. Current Opinion in Genetics and Development, 2017, 44, 1-8.	3.3	68
79	Avidity for Polypeptide Binding by Nucleotide-Bound Hsp104 Structures. Biochemistry, 2017, 56, 2071-2075.	2.5	14
80	Ratchet-like polypeptide translocation mechanism of the AAA+ disaggregase Hsp104. Science, 2017, 357, 273-279.	12.6	241
81	FUS inclusions disrupt RNA localization by sequestering kinesin-1 and inhibiting microtubule detyrosination. Journal of Cell Biology, 2017, 216, 1015-1034.	5.2	92
82	Susan Lee Lindquist (1949â€“2016). Trends in Biochemical Sciences, 2017, 42, 169-170.	7.5	0
83	RNA-binding proteins with prion-like domains in health and disease. Biochemical Journal, 2017, 474, 1417-1438.	3.7	347
84	Biology and Pathobiology of TDP-43 and Emergent Therapeutic Strategies. Cold Spring Harbor Perspectives in Medicine, 2017, 7, a024554.	6.2	56
85	Liquidizing <scp>FUS</scp> via prionâ€like domain phosphorylation. EMBO Journal, 2017, 36, 2925-2927.	7.8	17
86	Prion-like Domains Program Ewingâ€™s Sarcoma. Cell, 2017, 171, 30-31.	28.9	15
87	Protein-Remodeling Factors As Potential Therapeutics for Neurodegenerative Disease. Frontiers in Neuroscience, 2017, 11, 99.	2.8	27
88	Neurodegenerative disease: models, mechanisms, and a new hope. DMM Disease Models and Mechanisms, 2017, 10, 499-502.	2.4	508
89	Engineering and Evolution of Molecular Chaperones and Protein Disaggregases with Enhanced Activity. Frontiers in Molecular Biosciences, 2016, 3, 8.	3.5	44
90	Susan Lee Lindquist (1949â€“2016). Nature, 2016, 540, 40-40.	27.8	2

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91	Phasing in and out. Nature Chemistry, 2016, 8, 528-530.	13.6	28
92	Engineering therapeutic protein disaggregases. Molecular Biology of the Cell, 2016, 27, 1556-1560.	2.1	48
93	Spiral architecture of the Hsp104 disaggregase reveals the basis for polypeptide translocation. Nature Structural and Molecular Biology, 2016, 23, 830-837.	8.2	102
94	Mechanistic Insights into Hsp104 Potentiation. Journal of Biological Chemistry, 2016, 291, 5101-5115.	3.4	37
95	Prion-like domains as epigenetic regulators, scaffolds for subcellular organization, and drivers of neurodegenerative disease. Brain Research, 2016, 1647, 9-18.	2.2	195
96	Mechanistic and Structural Insights into the Prion-Disaggregase Activity of Hsp104. Journal of Molecular Biology, 2016, 428, 1870-1885.	4.2	80
97	Epigallocatechin-3-gallate rapidly remodels PAP85-120, SEM1(45-107), and SEM2(49-107) seminal amyloid fibrils. Biology Open, 2015, 4, 1206-1212.	1.2	20
98	The Hsp104 N-Terminal Domain Enables Disaggregase Plasticity and Potentiation. Molecular Cell, 2015, 57, 836-849.	9.7	83
99	Discovery and Characterization of an Endogenous CXCR4 Antagonist. Cell Reports, 2015, 11, 737-747.	6.4	80
100	Engineering enhanced protein disaggregases for neurodegenerative disease. Prion, 2015, 9, 90-109.	1.8	68
101	Itâ€™s Raining Liquids: RNA Tunes Viscoelasticity and Dynamics of Membraneless Organelles. Molecular Cell, 2015, 60, 189-192.	9.7	121
102	Disparate Mutations Confer Therapeutic Gain of Hsp104 Function. ACS Chemical Biology, 2015, 10, 2672-2679.	3.4	38
103	Chaperones in Neurodegeneration. Journal of Neuroscience, 2015, 35, 13853-13859.	3.6	81
104	Fleeting Amyloid-like Forms of Rim4 Ensure Meiotic Fidelity. Cell, 2015, 163, 275-276.	28.9	7
105	Repurposing Hsp104 to Antagonize Seminal Amyloid and Counter HIV Infection. Chemistry and Biology, 2015, 22, 1074-1086.	6.0	34
106	A molecular tweezer antagonizes seminal amyloids and HIV infection. ELife, 2015, 4, .	6.0	71
107	Suramin Inhibits Hsp104 ATPase and Disaggregase Activity. PLoS ONE, 2014, 9, e110115.	2.5	16
108	Reversing deleterious protein aggregation with re-engineered protein disaggregases. Cell Cycle, 2014, 13, 1379-1383.	2.6	37

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109	Conserved Distal Loop Residues in the Hsp104 and ClpB Middle Domain Contact Nucleotide-binding Domain 2 and Enable Hsp70-dependent Protein Disaggregation. Journal of Biological Chemistry, 2014, 289, 848-867.	3.4	42
110	Potentiated Hsp104 variants suppress toxicity of diverse neurodegenerative disease-linked proteins. DMM Disease Models and Mechanisms, 2014, 7, 1175-84.	2.4	74
111	Counteracting Semen-mediated Enhancement of HIV Infection and Enveloped Virus Infection by a Lysine-specific Molecular Tweezer. AIDS Research and Human Retroviruses, 2014, 30, A263-A263.	1.1	0
112	Potentiated Hsp104 Variants Antagonize Diverse Proteotoxic Misfolding Events. Cell, 2014, 156, 170-182.	28.9	205
113	Specific aromatic foldamers potently inhibit spontaneous and seeded AÎ²42 and AÎ²43 fibril assembly. Biochemical Journal, 2014, 464, 85-98.	3.7	13
114	A Cellular System that Degrades Misfolded Proteins and Protects against Neurodegeneration. Molecular Cell, 2014, 55, 15-30.	9.7	157
115	Isolating Potentiated Hsp104 Variants Using Yeast Proteinopathy Models. Journal of Visualized Experiments, 2014, , e52089.	0.3	13
116	ALS-associated mutation FUS-R521C causes DNA damage and RNA splicing defects. Journal of Clinical Investigation, 2014, 124, 981-999.	8.2	225
117	Fission Yeast Does Not Age under Favorable Conditions, but Does So after Stress. Current Biology, 2013, 23, 1844-1852.	3.9	83
118	Mutations in prion-like domains in hnRNPA2B1 and hnRNPA1 cause multisystem proteinopathy and ALS. Nature, 2013, 495, 467-473.	27.8	1,249
119	The metazoan protein disaggregase and amyloid depolymerase system. Prion, 2013, 7, 457-463.	1.8	67
120	Hsp104 Suppresses Polyglutamine-Induced Degeneration Post Onset in a Drosophila MJD/SCA3 Model. PLoS Genetics, 2013, 9, e1003781.	3.5	73
121	Stress granules as crucibles of ALS pathogenesis. Journal of Cell Biology, 2013, 201, 361-372.	5.2	756
122	Disease mutations in the prion-like domains of hnRNPA1 and hnRNPA2/B1 introduce potent steric zippers that drive excess RNP granule assembly. Rare Diseases (Austin, Tex), 2013, 1, e25200.	1.8	38
123	Small Heat Shock Proteins Potentiate Amyloid Dissolution by Protein Disaggregases from Yeast and Humans. PLoS Biology, 2012, 10, e1001346.	5.6	167
124	RNA-Binding Proteins in Amyotrophic Lateral Sclerosis and Neurodegeneration. Neurology Research International, 2012, 2012, 1-5.	1.3	26
125	Evaluating the role of the FUS/TLS-related gene EWSR1 in amyotrophic lateral sclerosis. Human Molecular Genetics, 2012, 21, 2899-2911.	2.9	246
126	Hsp104 Drives â€œProtein-Onlyâ€•Positive Selection of Sup35 Prion Strains Encoding Strong [PSI]. Chemistry and Biology, 2012, 19, 1400-1410.	6.0	40

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127	Operational Plasticity Enables Hsp104 to Disaggregate Diverse Amyloid and Nonamyloid Clients. Cell, 2012, 151, 778-793.	28.9	162
128	The Surprising Role of Amyloid Fibrils in HIV Infection. Biology, 2012, 1, 58-80.	2.8	56
129	Inhibition of RNA lariat debranching enzyme suppresses TDP-43 toxicity in ALS disease models. Nature Genetics, 2012, 44, 1302-1309.	21.4	214
130	The elusive middle domain of Hsp104 and ClpB: Location and function. Biochimica Et Biophysica Acta - Molecular Cell Research, 2012, 1823, 29-39.	4.1	68
131	The tip of the iceberg: RNA-binding proteins with prion-like domains in neurodegenerative disease. Brain Research, 2012, 1462, 61-80.	2.2	572
132	The Mammalian Disaggregase Machinery: Hsp110 Synergizes with Hsp70 and Hsp40 to Catalyze Protein Disaggregation and Reactivation in a Cell-Free System. PLoS ONE, 2011, 6, e26319.	2.5	282
133	Purification of Hsp104, a Protein Disaggregase. Journal of Visualized Experiments, 2011, , .	0.3	15
134	RNA-binding proteins with prion-like domains in ALS and FTLD-U. Prion, 2011, 5, 179-187.	1.8	140
135	A yeast functional screen predicts new candidate ALS disease genes. Proceedings of the National Academy of Sciences of the United States of America, 2011, 108, 20881-20890.	7.1	365
136	Molecular Determinants and Genetic Modifiers of Aggregation and Toxicity for the ALS Disease Protein FUS/TLS. PLoS Biology, 2011, 9, e1000614.	5.6	396
137	Shock and awe: unleashing the heat shock response to treat Huntington disease. Journal of Clinical Investigation, 2011, 121, 2972-2975.	8.2	12
138	Emergence and natural selection of drug-resistant prions. Molecular BioSystems, 2010, 6, 1115.	2.9	48
139	Countering amyloid polymorphism and drug resistance with minimal drug cocktails. Prion, 2010, 4, 244-251.	1.8	18
140	Prion-like disorders: blurring the divide between transmissibility and infectivity. Journal of Cell Science, 2010, 123, 1191-1201.	2.0	268
141	Applying Hsp104 to protein-misfolding disordersThis paper is one of a selection of papers published in this special issue entitled 8th International Conference on AAA Proteins and has undergone the Journal's usual peer review process.. Biochemistry and Cell Biology, 2010, 88, 1-13.	2.0	73
142	N-terminal Domains Elicit Formation of Functional Pmel17 Amyloid Fibrils. Journal of Biological Chemistry, 2009, 284, 35543-35555.	3.4	101
143	TDP-43 Is Intrinsically Aggregation-prone, and Amyotrophic Lateral Sclerosis-linked Mutations Accelerate Aggregation and Increase Toxicity. Journal of Biological Chemistry, 2009, 284, 20329-20339.	3.4	651
144	TDP-43 is intrinsically aggregation-prone, and amyotrophic lateral sclerosis-linked mutations accelerate aggregation and increase toxicity.. Journal of Biological Chemistry, 2009, 284, 25459.	3.4	11

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145	A synergistic small-molecule combination directly eradicates diverse prion strain structures. Nature Chemical Biology, 2009, 5, 936-946.	8.0	93
146	Motor Mechanism for Protein Threading through Hsp104. Molecular Cell, 2009, 34, 81-92.	9.7	84
147	A PDZâ€Binding Motif Controls Basolateral Targeting of Syndecanâ€1 Along the Biosynthetic Pathway in Polarized Epithelial Cells. Traffic, 2008, 9, 1915-1924.	2.7	62
148	Hsp104, Hsp70 and Hsp40 interplay regulates formation, growth and elimination of Sup35 prions. EMBO Journal, 2008, 27, 2712-2724.	7.8	153
149	Escaping amyloid fate. Nature Structural and Molecular Biology, 2008, 15, 544-546.	8.2	37
150	Hsp104 antagonizes Î±-synuclein aggregation and reduces dopaminergic degeneration in a rat model of Parkinson disease. Journal of Clinical Investigation, 2008, 118, 3087-3097.	8.2	184
151	The Parkinson's disease protein Î±-synuclein disrupts cellular Rab homeostasis. Proceedings of the National Academy of Sciences of the United States of America, 2008, 105, 145-150.	7.1	479
152	The Mad2 partial unfolding model: regulating mitosis through Mad2 conformational switching. Journal of Cell Biology, 2008, 183, 761-768.	5.2	51
153	Prion proteostasis. Prion, 2008, 2, 135-140.	1.8	42
154	Hsp104: A Weapon to Combat Diverse Neurodegenerative Disorders. NeuroSignals, 2008, 16, 63-74.	0.9	108
155	Direct and selective elimination of specific prions and amyloids by 4,5-dianilinophthalimide and analogs. Proceedings of the National Academy of Sciences of the United States of America, 2008, 105, 7159-7164.	7.1	53
156	Hsp110 Chaperones Regulate Prion Formation and Propagation in S. cerevisiae by Two Discrete Activities. PLoS ONE, 2008, 3, e1763.	2.5	69
157	Atypical AAA+ Subunit Packing Creates an Expanded Cavity for Disaggregation by the Protein-Remodeling Factor Hsp104. Cell, 2007, 131, 1366-1377.	28.9	107
158	Prime Time for Â-Synuclein. Journal of Neuroscience, 2007, 27, 2433-2434.	3.6	48
159	Asymmetric deceleration of ClpB or Hsp104 ATPase activity unleashes protein-remodeling activity. Nature Structural and Molecular Biology, 2007, 14, 114-122.	8.2	139
160	Destruction or Potentiation of Different Prions Catalyzed by Similar Hsp104 Remodeling Activities. Molecular Cell, 2006, 23, 425-438.	9.7	197
161	Navigating the ClpB channel to solution. Nature Structural and Molecular Biology, 2005, 12, 4-6.	8.2	28
162	Prions as adaptive conduits of memory and inheritance. Nature Reviews Genetics, 2005, 6, 435-450.	16.3	500

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163	A Cryptic Rab1-binding Site in the p115 TetheringProtein. Journal of Biological Chemistry, 2005, 280, 25840-25848.	3.4	95
164	Hsp104 Catalyzes Formation and Elimination of Self-Replicating Sup35 Prion Conformers. Science, 2004, 304, 1793-1797.	12.6	454
165	A direct role for GRASP65 as a mitotically regulated Golgi stacking factor. EMBO Journal, 2003, 22, 3279-3290.	7.8	169
166	Sequential tethering of Golgins and catalysis of SNAREpin assembly by the vesicle-tethering protein p115. Journal of Cell Biology, 2002, 157, 45-62.	5.2	188
167	Sequential SNARE disassembly and GATE-16â€“GOS-28 complex assembly mediated by distinct NSF activities drives Golgi membrane fusion. Journal of Cell Biology, 2002, 157, 1161-1173.	5.2	83
168	Golgi Architecture and Inheritance. Annual Review of Cell and Developmental Biology, 2002, 18, 379-420.	9.4	337
169	Membrane traffic: Do cones mark sites of fission?. Current Biology, 2000, 10, R141-R144.	3.9	32
170	A complex of mammalian Ufd1 and Npl4 links the AAA-ATPase, p97, to ubiquitin and nuclear transport pathways. EMBO Journal, 2000, 19, 2181-2192.	7.8	404
171	The Amino-terminal Domain of the Golgi Protein Giantin Interacts Directly with the Vesicle-tethering Protein p115. Journal of Biological Chemistry, 2000, 275, 2831-2836.	3.4	74
172	Phosphorylation of the Vesicle-Tethering Protein P115 by a Casein Kinase IIâ€“Like Enzyme Is Required for Golgi Reassembly from Isolated Mitotic Fragments. Journal of Cell Biology, 2000, 150, 475-488.	5.2	69
173	A Role for the Vesicle Tethering Protein, P115, in the Post-Mitotic Stacking of Reassembling Golgi Cisternae in a Cell-Free System. Journal of Cell Biology, 1999, 146, 57-70.	5.2	149
174	An NSF function distinct from ATPase-dependent SNARE disassembly is essential for Golgi membrane fusion. Nature Cell Biology, 1999, 1, 335-340.	10.3	58
175	GRASP55, a second mammalian GRASP protein involved in the stacking of Golgi cisternae in a cell-free system. EMBO Journal, 1999, 18, 4949-4960.	7.8	287