Maurizio Molinari

List of Publications by Year
in descending order

99 papers	15,136 citations	41258 49 h-index	38300 95 g-index
154 all docs	154 docs citations	154 times ranked	22528 citing authors

#	Article	IF	Citations
1	Quantitative and time-resolved monitoring of organelle and protein delivery to the lysosome with a tandem fluorescent Halo-GFP reporter. Molecular Biology of the Cell, 2022, 33, mbcE21100526.	0.9	7
2	ER-phagy: mechanisms, regulation, and diseases connected to the lysosomal clearance of the endoplasmic reticulum. Physiological Reviews, 2022, 102, 1393-1448.	13.1	53
3	Tandem fluorescent Halo-GFP reporter for quantitative and time-resolved monitoring of organelle and protein delivery to lysosomes. , 2022, 1, 187-191.		1
4	Proteasomal and Lysosomal Degradation of Misfolded Proteins From the Endoplasmic Reticulum. , 2022, , .		0
5	Protein Turnover | Endoplasmic Reticulum-Associated Protein Degradation. , 2021, , 225-228.		0
6	ER-phagy responses in yeast, plants, and mammalian cells and their crosstalk with UPR and ERAD. Developmental Cell, 2021, 56, 949-966.	3.1	72
7	Nâ€glycan processing selects ERADâ€resistant misfolded proteins for ERâ€toâ€lysosomeâ€associated degradation. EMBO Journal, 2021, 40, e107240.	3.5	30
8	Endoplasmic Reticulum (ER) and ER-Phagy. Progress in Molecular and Subcellular Biology, 2021, 59, 99-114.	0.9	4
9	Thioredoxin-Related Transmembrane Proteins: TMX1 and Little Brothers TMX2, TMX3, TMX4 and TMX5. Cells, 2020, 9, 2000.	1.8	10
10	Deep learning approach for quantification of organelles and misfolded polypeptide delivery within degradative compartments. Molecular Biology of the Cell, 2020, 31, 1512-1524.	0.9	20
11	ER-phagy: Eating the Factory. Molecular Cell, 2020, 78, 811-813.	4.5	13
12	Identification of signal peptide features for substrate specificity in human Sec62/Sec63â€dependent ER protein import. FEBS Journal, 2020, 287, 4612-4640.	2.2	40
13	Mechanistic insights in recov-ER-phagy: micro-ER-phagy to recover from stress. Autophagy, 2020, 16, 385-386.	4.3	28
14	ESCRT-III-driven piecemeal micro-ER-phagy remodels the ER during recovery from ER stress. Nature Communications, 2019, 10, 5058.	5.8	94
15	Proteasomal and lysosomal clearance of faulty secretory proteins: ER-associated degradation (ERAD) and ER-to-lysosome-associated degradation (ERLAD) pathways. Critical Reviews in Biochemistry and Molecular Biology, 2019, 54, 153-163.	2.3	110
16	Schwann cells ER-associated degradation contributes to myelin maintenance in adult nerves and limits demyelination in CMT1B mice. PLoS Genetics, 2019, 15, e1008069.	1.5	18
17	A selective <scp>ER</scp> â€phagy exerts procollagen quality control via a Calnexinâ€•<scp>FAM</scp> 134B complex. EMBO Journal, 2019, 38, .	3.5	178
18	Chemical stresses fail to mimic the unfolded protein response resulting from luminal load with unfolded polypeptides. Journal of Biological Chemistry, 2018, 293, 5600-5612.	1.6	53

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19	The reductase TMX1 contributes to ERAD by preferentially acting on membrane-associated folding-defective polypeptides. Biochemical and Biophysical Research Communications, 2018, 503, 938-943.	1.0	9
20	<scp>ER</scp> â€toâ€lysosomeâ€associated degradation of proteasomeâ€resistant <scp>ATZ</scp> polymers occurs via receptorâ€mediated vesicular transport. EMBO Journal, 2018, 37, .	3.5	144
21	Three branches to rule them all? UPR signalling in response to chemically versus misfolded proteinsâ€induced ER stress. Biology of the Cell, 2018, 110, 197-204.	0.7	29
22	Eat it right: ER-phagy and recovER-phagy. Biochemical Society Transactions, 2018, 46, 699-706.	1.6	39
23	Endoplasmic reticulum turnover: ER-phagy and other flavors in selective and non-selective ER clearance. F1000Research, 2018, 7, 454.	0.8	57
24	Role of SEC62 in ER maintenance: A link with ER stress tolerance in SEC62-overexpressing tumors?. Molecular and Cellular Oncology, 2017, 4, e1264351.	0.3	24
25	Translocon component Sec62 acts in endoplasmic reticulum turnover during stress recovery. Nature Cell Biology, 2016, 18, 1173-1184.	4.6	350
26	Five Questions (with their Answers) on <scp>ER</scp>â€Associated Degradation. Traffic, 2016, 17, 341-350.	1.3	31
27	Guidelines for the use and interpretation of assays for monitoring autophagy (3rd edition). Autophagy, 2016, 12, 1-222.	4.3	4,701
28	Quality control mechanisms of protein biogenesis: proteostasis dies hard. AIMS Biophysics, 2016, 3, 456-478.	0.3	4
29	A novel UGGT1 and p97-dependent checkpoint for native ectodomains with ionizable intramembrane residue. Molecular Biology of the Cell, 2015, 26, 1532-1542.	0.9	14
30	Glycoprotein maturation and quality control. Seminars in Cell and Developmental Biology, 2015, 41, 70.	2.3	9
31	The Protein-disulfide Isomerase ERp57 Regulates the Steady-state Levels of the Prion Protein. Journal of Biological Chemistry, 2015, 290, 23631-23645.	1.6	48
32	Division of labor among oxidoreductases: TMX1 preferentially acts on transmembrane polypeptides. Molecular Biology of the Cell, 2015, 26, 3390-3400.	0.9	24
33	N-linked sugar-regulated protein folding and quality control in the ER. Seminars in Cell and Developmental Biology, 2015, 41, 79-89.	2.3	194
34	RESETting proteostasis. Nature Chemical Biology, 2014, 10, 881-882.	3.9	5
35	How Viruses Hijack the ERAD Tuning Machinery. Journal of Virology, 2014, 88, 10272-10275.	1.5	40
36	Non-Lipidated LC3 is Essential for Mouse Hepatitis Virus Infection. , 2014, , 129-136.		1

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37	Proteostasis: Bad news and good news from the endoplasmic reticulum. Swiss Medical Weekly, 2014, 144, w14001.	0.8	13
38	Transgenic expression of Î²1 antibody in brain neurons impairs age-dependent amyloid deposition in APP23 mice. Neurobiology of Aging, 2013, 34, 2866-2878.	1.5	4
39	Specificity and Regulation of the Endoplasmic Reticulumâ€Associated Degradation Machinery. Traffic, 2013, 14, 767-777.	1.3	50
40	Endoplasmic Reticulum-Associated Protein Degradation. , 2013, , 200-203.		0
41	UDP-glucose:glycoprotein glucosyltransferase (UGGT1) promotes substrate solubility in the endoplasmic reticulum. Molecular Biology of the Cell, 2013, 24, 2597-2608.	0.9	40
42	Unconventional roles of nonlipidated LC3 in ERAD tuning and coronavirus infection. Autophagy, 2012, 8, 1534-1536.	4.3	17
43	Flagging and docking: dual roles for N-glycans in protein quality control and cellular proteostasis. Trends in Biochemical Sciences, 2012, 37, 404-410.	3.7	81
44	Unconventional Use of LC3 by Coronaviruses through the Alleged Subversion of the ERAD Tuning Pathway. Viruses, 2011, 3, 1610-1623.	1.5	21
45	Malectin Participates in a Backup Glycoprotein Quality Control Pathway in the Mammalian ER. PLoS ONE, 2011, 6, e16304.	1.1	70
46	Chronic Delivery of Antibody Fragments Using Immunoisolated Cell Implants as a Passive Vaccination Tool. PLoS ONE, 2011, 6, e18268.	1.1	7
47	ERAD and ERAD tuning: disposal of cargo and of ERAD regulators from the mammalian ER. Current Opinion in Cell Biology, 2011, 23, 176-183.	2.6	115
48	N-glycan structures: recognition and processing in the ER. Trends in Biochemical Sciences, 2010, 35, 74-82.	3.7	404
49	Stringent requirement for HRD1, SEL1L, and OS-9/XTP3-B for disposal of ERAD-LS substrates. Journal of Cell Biology, 2010, 188, 223-235.	2.3	163
50	Autophagy-independent LC3 function in vesicular traffic. Autophagy, 2010, 6, 994-996.	4.3	25
51	Coronaviruses Hijack the LC3-I-Positive EDEMosomes, ER-Derived Vesicles Exporting Short-Lived ERAD Regulators, for Replication. Cell Host and Microbe, 2010, 7, 500-508.	5.1	332
52	ERAD substrates: Which way out?. Seminars in Cell and Developmental Biology, 2010, 21, 526-532.	2.3	102
53	Cyclosporine A-Sensitive, Cyclophilin B-Dependent Endoplasmic Reticulum-Associated Degradation. PLoS ONE, 2010, 5, e13008.	1.1	45
54	Segregation and rapid turnover of EDEM1 by an autophagy-like mechanism modulates standard ERAD and folding activities. Biochemical and Biophysical Research Communications, 2008, 371, 405-410.	1.0	111

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55	The Endoplasmic Reticulum: Crossroads for Newly Synthesized Polypeptide Chains. Progress in Molecular Biology and Translational Science, 2008, 83, 135-179.	0.9	18
56	Consequences of Individual N-glycan Deletions and of Proteasomal Inhibition on Secretion of Active BACE. Molecular Biology of the Cell, 2008, 19, 4086-4098.	0.9	25
57	A Dual Task for the Xbp1-responsive OS-9 Variants in the Mammalian Endoplasmic Reticulum. Journal of Biological Chemistry, 2008, 283, 16446-16454.	1.6	107
58	Substrate-Specific Requirements for UGT1-Dependent Release from Calnexin. Molecular Cell, 2007, 27, 238-249.	4.5	77
59	Glycoprotein folding and the role of EDEM1, EDEM2 and EDEM3 in degradation of folding-defective glycoproteins. FEBS Letters, 2007, 581, 3658-3664.	1.3	119
60	In and Out of the ER: Protein Folding, Quality Control, Degradation, and Related Human Diseases. Physiological Reviews, 2007, 87, 1377-1408.	13.1	563
61	N-glycan structure dictates extension of protein folding or onset of disposal. Nature Chemical Biology, 2007, 3, 313-320.	3.9	258
62	EDEM1 regulates ER-associated degradation by accelerating de-mannosylation of folding-defective polypeptides and by inhibiting their covalent aggregation. Biochemical and Biophysical Research Communications, 2006, 349, 1278-1284.	1.0	154
63	N-glycan processing in ER quality control. Journal of Cell Science, 2006, 119, 4373-4380.	1.2	266
64	Death of a chaperone. Nature, 2006, 443, 511-512.	13.7	9
65	N-linked glycan recognition and processing: the molecular basis of endoplasmic reticulum quality control. Current Opinion in Structural Biology, 2006, 16, 592-599.	2.6	111
66	Consequences of ERp57 Deletion on Oxidative Folding of Obligate and Facultative Clients of the Calnexin Cycle. Journal of Biological Chemistry, 2006, 281, 6219-6226.	1.6	102
67	Analyzing folding and degradation of metabolically labelled polypeptides by conventional and diagonal sodium dodecyl sulfate-polyacrylamide gel electrophoresis. Biological Procedures Online, 2005, 7, 136-143.	1.4	4
68	The Use of Calnexin and Calreticulin by Cellular and Viral Glycoproteins. Journal of Biological Chemistry, 2005, 280, 28265-28271.	1.6	56
69	A Novel Stress-induced EDEM Variant Regulating Endoplasmic Reticulum-associated Glycoprotein Degradation. Journal of Biological Chemistry, 2005, 280, 2424-2428.	1.6	143
70	Degradation of Trafficking-defective Long QT Syndrome Type II Mutant Channels by the Ubiquitin-Proteasome Pathway. Journal of Biological Chemistry, 2005, 280, 19419-19425.	1.6	99
71	The glycan code of the endoplasmic reticulum: asparagine-linked carbohydrates as protein maturation and quality-control tags. Trends in Cell Biology, 2005, 15, 364-370.	3.6	227
72	Î²-site specific intrabodies to decrease and prevent generation of Alzheimer's AÎ² peptide. Journal of Cell Biology, 2005, 168, 863-868.	2.3	98

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73	Persistent Glycoprotein Misfolding Activates the Glucosidase II/UGT1-Driven Calnexin Cycle to Delay Aggregation and Loss of Folding Competence. Molecular Cell, 2005, 20, 503-512.	4.5	109
74	The Secretory Capacity of a Cell Depends on the Efficiency of Endoplasmic Reticulum-Associated Degradation. , 2005, 300, 1-15.		41
75	Endoplasmic Reticulum-Associated Protein Degradation. , 2004, , 20-23.		0
76	EDEM Contributes to Maintenance of Protein Folding Efficiency and Secretory Capacity. Journal of Biological Chemistry, 2004, 279, 44600-44605.	1.6	40
77	Contrasting Functions of Calreticulin and Calnexin in Glycoprotein Folding and ER Quality Control. Molecular Cell, 2004, 13, 125-135.	4.5	196
78	Role of EDEM in the Release of Misfolded Glycoproteins from the Calnexin Cycle. Science, 2003, 299, 1397-1400.	6.0	431
79	Early Postnatal Death and Motor Disorders in Mice Congenitally Deficient in Calnexin Expression. Molecular and Cellular Biology, 2002, 22, 7398-7404.	1.1	125
80	[4] Analyzing cotranslational protein folding and disulfide formation by diagonal sodium dodecyl sulfate-polyacrylamide gel electrophoresis. Methods in Enzymology, 2002, 348, 35-42.	0.4	15
81	Sequential assistance of molecular chaperones and transient formation of covalent complexes during protein degradation from the ER. Journal of Cell Biology, 2002, 158, 247-257.	2.3	204
82	The disulphide bonds in the catalytic domain of BACE are critical but not essential for amyloid precursor protein processing activity. Journal of Neurochemistry, 2002, 80, 1079-1088.	2.1	31
83	Folding of viral glycoproteins in the endoplasmic reticulum. Virus Research, 2001, 82, 83-86.	1.1	4
84	Chaperone Selection During Glycoprotein Translocation into the Endoplasmic Reticulum. Science, 2000, 288, 331-333.	6.0	315
85	The Helicobacter pylori neutrophil-activating protein is an iron-binding protein with dodecameric structure. Molecular Microbiology, 1999, 34, 238-246.	1.2	159
86	Glycoproteins form mixed disulphides with oxidoreductases during folding in living cells. Nature, 1999, 402, 90-93.	13.7	294
87	Setting the Standards: Quality Control in the Secretory Pathway. Science, 1999, 286, 1882-1888.	6.0	1,142
88	Action site and cellular effects of cytotoxin VacA produced byHelicobacter pylori. Folia Microbiologica, 1998, 43, 279-284.	1.1	14
89	Calpain: A Protease in Search of a Function?. Biochemical and Biophysical Research Communications, 1998, 247, 193-203.	1.0	352
90	The Acid Activation ofHelicobacter pyloriToxin VacA: Structural and Membrane Binding Studies. Biochemical and Biophysical Research Communications, 1998, 248, 334-340.	1.0	84

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91	Selective Inhibition of Ii-dependent Antigen Presentation by Helicobacter pylori Toxin VacA. Journal of Experimental Medicine, 1998, 187, 135-140.	4.2	270
92	Vacuoles Induced by Helicobacter pylori Toxin Contain Both Late Endosomal and Lysosomal Markers. Journal of Biological Chemistry, 1997, 272, 25339-25344.	1.6	174
93	Proteolysis by Calpains: a Possible Contribution to Degradation of p53. Molecular and Cellular Biology, 1997, 17, 2806-2815.	1.1	163
94	Calpain: A Cytosolic Proteinase Active at the Membranes. Journal of Membrane Biology, 1997, 156, 1-8.	1.0	146
95	Purification of Active Calpain by Affinity Chromatography on an Immobilized Peptide Inhibitor. FEBS Journal, 1996, 241, 948-954.	0.2	24
96	Purification of Î¼-Calpain by a Novel Affinity Chromatography Approach. NEW INSIGHTS INTO THE MECHANISM OF THE INTERACTION OF THE PROTEASE WITH TARGETS. Journal of Biological Chemistry, 1995, 270, 14576-14581.	1.6	34
97	PEST Sequences Do Not Influence Substrate Susceptibility to Calpain Proteolysis. Journal of Biological Chemistry, 1995, 270, 2032-2035.	1.6	57
98	Ca(2+)-activated neutral protease is active in the erythrocyte membrane in its nonautolyzed 80-kDa form.. Journal of Biological Chemistry, 1994, 269, 27992-27995.	1.6	85
99	Ca(2+)-activated neutral protease is active in the erythrocyte membrane in its nonautolyzed 80-kDa form. Journal of Biological Chemistry, 1994, 269, 27992-5.	1.6	69