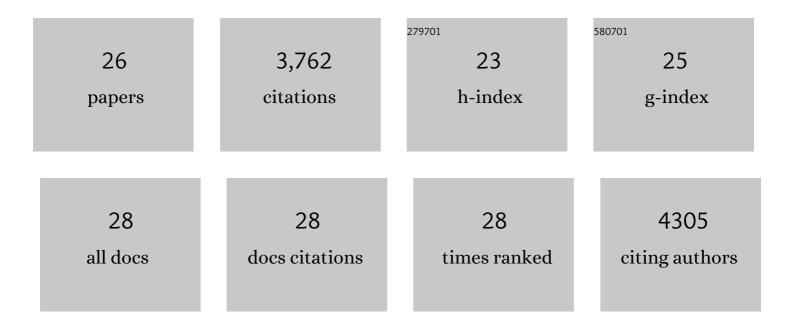
## Anatoli Meriin

List of Publications by Year in descending order

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ANATOLI MEDUN

#	Article	IF	CITATIONS
1	The Chaperone Function of hsp70 Is Required for Protection against Stress-Induced Apoptosis. Molecular and Cellular Biology, 2000, 20, 7146-7159.	1.1	646
2	Hsp70 Prevents Activation of Stress Kinases. Journal of Biological Chemistry, 1997, 272, 18033-18037.	1.6	473
3	Huntingtin toxicity in yeast model depends on polyglutamine aggregation mediated by a prion-like protein Rnq1. Journal of Cell Biology, 2002, 157, 997-1004.	2.3	348
4	Proteasome Inhibitors Activate Stress Kinases and Induce Hsp72. Journal of Biological Chemistry, 1998, 273, 6373-6379.	1.6	280
5	A potent small molecule inhibits polyglutamine aggregation in Huntington's disease neurons and suppresses neurodegeneration in vivo. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 892-897.	3.3	257
6	Protein-Damaging Stresses Activate c-Jun N-Terminal Kinase via Inhibition of Its Dephosphorylation: a Novel Pathway Controlled by HSP72. Molecular and Cellular Biology, 1999, 19, 2547-2555.	1.1	234
7	Role of Hsp70 in regulation of stress-kinase JNK: implications in apoptosis and aging. FEBS Letters, 1998, 438, 1-4.	1.3	215
8	Hsp72-Mediated Suppression of c-Jun N-Terminal Kinase Is Implicated in Development of Tolerance to Caspase-Independent Cell Death. Molecular and Cellular Biology, 2000, 20, 6826-6836.	1.1	154
9	Abnormal proteins can form aggresome in yeast: aggresomeâ€ŧargeting signals and components of the machinery. FASEB Journal, 2009, 23, 451-463.	0.2	150
10	Role of molecular chaperones in neurodegenerative disorders. International Journal of Hyperthermia, 2005, 21, 403-419.	1.1	111
11	Suppression of Stress Kinase JNK Is Involved in HSP72-mediated Protection of Myogenic Cells from Transient Energy Deprivation. Journal of Biological Chemistry, 2000, 275, 38088-38094.	1.6	101
12	Aggregation of Expanded Polyglutamine Domain in Yeast Leads to Defects in Endocytosis. Molecular and Cellular Biology, 2003, 23, 7554-7565.	1.1	98
13	A first order phase transition mechanism underlies protein aggregation in mammalian cells. ELife, 2019, 8, .	2.8	80
14	Triggering Aggresome Formation. Journal of Biological Chemistry, 2008, 283, 27575-27584.	1.6	75
15	The Function of HSP72 in Suppression of c-Jun N-terminal Kinase Activation Can Be Dissociated from Its Role in Prevention of Protein Damage. Journal of Biological Chemistry, 1999, 274, 20223-20228.	1.6	71
16	The heat shock transcription factor Hsf1 is downregulated in DNA damage–associated senescence, contributing to the maintenance of senescence phenotype. Aging Cell, 2012, 11, 617-627.	3.0	66
17	Endocytosis machinery is involved in aggregation of proteins with expanded polyglutamine domains. FASEB Journal, 2007, 21, 1915-1925.	0.2	63
18	Proteasome Failure Promotes Positioning of Lysosomes around the Aggresome via Local Block of Microtubule-Dependent Transport. Molecular and Cellular Biology, 2014, 34, 1336-1348.	1.1	62

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#	Article	IF	CITATIONS
19	Hsp70–Bag3 complex is a hub for proteotoxicity-induced signaling that controls protein aggregation. Proceedings of the National Academy of Sciences of the United States of America, 2018, 115, E7043-E7052.	3.3	55
20	Intracellular Aggregation of Polypeptides with Expanded Polyglutamine Domain Is Stimulated by Stress-Activated Kinase Mekk1. Journal of Cell Biology, 2001, 153, 851-864.	2.3	54
21	Characterization of Proteins Associated with Polyglutamine Aggregates. Prion, 2007, 1, 128-135.	0.9	48
22	RuvbL1 and RuvbL2 enhance aggresome formation and disaggregate amyloid fibrils. EMBO Journal, 2015, 34, 2363-2382.	3.5	47
23	Association of translation factor eEF1A with defective ribosomal products generates a signal for aggresome formation Journal of Cell Science, 2012, 125, 2665-74.	1.2	28
24	A Novel Approach to Recovery of Function of Mutant Proteins by Slowing Down Translation. Journal of Biological Chemistry, 2012, 287, 34264-34272.	1.6	22
25	Insulin-responsive amino peptidase follows the Clut4 pathway but is dispensable for the formation and translocation of insulin-responsive vesicles. Molecular Biology of the Cell, 2019, 30, 1536-1543.	0.9	17
26	HEAT SHOCK PROTEIN 70 PROTECTS FROM CASPASE-INDEPENDENT PROGRAMMED CELL DEATH VIA SUPPRESSION OF STRESS KINASE JNK. Scientific World Journal, The, 2001, 1, 36-36.	0.8	0