

# Lisa M Ellerby

## List of Publications by Year in descending order

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47  
papers

4,350  
citations

201674

27  
h-index

223800

46  
g-index

53  
all docs

53  
docs citations

53  
times ranked

4089  
citing authors

#	ARTICLE	IF	CITATIONS
1	Caspase Cleavage of Gene Products Associated with Triplet Expansion Disorders Generates Truncated Fragments Containing the Polyglutamine Tract. <i>Journal of Biological Chemistry</i> , 1998, 273, 9158-9167.	3.4	499
2	Huntingtin Interacting Proteins Are Genetic Modifiers of Neurodegeneration. <i>PLoS Genetics</i> , 2007, 3, e82.	3.5	368
3	Caspase Cleavage of Mutant Huntingtin Precedes Neurodegeneration in Huntington's Disease. <i>Journal of Neuroscience</i> , 2002, 22, 7862-7872.	3.6	344
4	Genetic Correction of Huntington's Disease Phenotypes in Induced Pluripotent Stem Cells. <i>Cell Stem Cell</i> , 2012, 11, 253-263.	11.1	336
5	Calpain Activation in Huntington's Disease. <i>Journal of Neuroscience</i> , 2002, 22, 4842-4849.	3.6	282
6	Proteolysis of Mutant Huntingtin Produces an Exon 1 Fragment That Accumulates as an Aggregated Protein in Neuronal Nuclei in Huntington Disease. <i>Journal of Biological Chemistry</i> , 2010, 285, 8808-8823.	3.4	259
7	Inhibition of Calpain Cleavage of Huntingtin Reduces Toxicity. <i>Journal of Biological Chemistry</i> , 2004, 279, 20211-20220.	3.4	242
8	Characterization of Human Huntington's Disease Cell Model from Induced Pluripotent Stem Cells. <i>PLOS Currents</i> , 2010, 2, RRN1193.	1.4	216
9	Kennedy's Disease. <i>Journal of Neurochemistry</i> , 1999, 72, 185-195.	3.9	211
10	Matrix Metalloproteinases Are Modifiers of Huntingtin Proteolysis and Toxicity in Huntington's Disease. <i>Neuron</i> , 2010, 67, 199-212.	8.1	152
11	Huntingtin Phosphorylation Sites Mapped by Mass Spectrometry. <i>Journal of Biological Chemistry</i> , 2006, 281, 23686-23697.	3.4	131
12	Genomic Analysis Reveals Disruption of Striatal Neuronal Development and Therapeutic Targets in Human Huntington's Disease Neural Stem Cells. <i>Stem Cell Reports</i> , 2015, 5, 1023-1038.	4.8	117
13	Cleavage of Atrophin-1 at Caspase Site Aspartic Acid 109 Modulates Cytotoxicity. <i>Journal of Biological Chemistry</i> , 1999, 274, 8730-8736.	3.4	99
14	Mutant huntingtin impairs PNKP and ATXN3, disrupting DNA repair and transcription. <i>ELife</i> , 2019, 8, .	6.0	83
15	Nuclear Localization of a Non-caspase Truncation Product of Atrophin-1, with an Expanded Polyglutamine Repeat, Increases Cellular Toxicity. <i>Journal of Biological Chemistry</i> , 2003, 278, 13047-13055.	3.4	78
16	Proteolytic Cleavage of Ataxin-7 by Caspase-7 Modulates Cellular Toxicity and Transcriptional Dysregulation. <i>Journal of Biological Chemistry</i> , 2007, 282, 30150-30160.	3.4	69
17	Kennedy's Disease. <i>Journal of Biological Chemistry</i> , 2003, 278, 34918-34924.	3.4	67
18	Progressive phenotype and nuclear accumulation of an amino-terminal cleavage fragment in a transgenic mouse model with inducible expression of full-length mutant huntingtin. <i>Neurobiology of Disease</i> , 2006, 21, 381-391.	4.4	59

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19	Polyglutamine-Expanded Androgen Receptor Truncation Fragments Activate a Bax-Dependent Apoptotic Cascade Mediated by DP5/Hrk. <i>Journal of Neuroscience</i> , 2009, 29, 1987-1997.	3.6	56
20	Polyglutamine Disease Modeling: Epitope Based Screen for Homologous Recombination using CRISPR/Cas9 System. <i>PLOS Currents</i> , 2014, 6, .	1.4	52
21	Identification and Evaluation of Small Molecule Pan-Caspase Inhibitors in Huntington's Disease Models. <i>Chemistry and Biology</i> , 2010, 17, 1189-1200.	6.0	50
22	Posttranslational Modification of Ataxin-7 at Lysine 257 Prevents Autophagy-Mediated Turnover of an N-Terminal Caspase-7 Cleavage Fragment. <i>Journal of Neuroscience</i> , 2009, 29, 15134-15144.	3.6	47
23	Caspase-6 Activity in a BACHD Mouse Modulates Steady-State Levels of Mutant Huntingtin Protein But Is Not Necessary for Production of a 586 Amino Acid Proteolytic Fragment. <i>Journal of Neuroscience</i> , 2012, 32, 7454-7465.	3.6	46
24	Insulin-like growth factor 2 (IGF2) protects against Huntington's disease through the extracellular disposal of protein aggregates. <i>Acta Neuropathologica</i> , 2020, 140, 737-764.	7.7	43
25	A Genome-Scale RNA Interference Screen Identifies RRAS Signaling as a Pathologic Feature of Huntington's Disease. <i>PLoS Genetics</i> , 2012, 8, e1003042.	3.5	41
26	Ataxin-7 Can Export from the Nucleus via a Conserved Exportin-dependent Signal. <i>Journal of Biological Chemistry</i> , 2006, 281, 2730-2739.	3.4	38
27	Mass Spectrometric Identification of Novel Lysine Acetylation Sites in Huntingtin. <i>Molecular and Cellular Proteomics</i> , 2011, 10, M111.009829.	3.8	34
28	Modulating FKBP5/FKBP51 and autophagy lowers HTT (huntingtin) levels. <i>Autophagy</i> , 2021, 17, 4119-4140.	9.1	27
29	Inhibition of Lipid Signaling Enzyme Diacylglycerol Kinase $\beta$ Attenuates Mutant Huntingtin Toxicity. <i>Journal of Biological Chemistry</i> , 2012, 287, 21204-21213.	3.4	26
30	iPSC-based drug screening for Huntington's disease. <i>Brain Research</i> , 2016, 1638, 42-56.	2.2	26
31	Pizotifen Activates ERK and Provides Neuroprotection in vitro and in vivo in Models of Huntington's Disease. <i>Journal of Huntington's Disease</i> , 2012, 1, 195-210.	1.9	25
32	Histone deacetylase-3 interacts with ataxin-7 and is altered in a spinocerebellar ataxia type 7 mouse model. <i>Molecular Neurodegeneration</i> , 2013, 8, 42.	10.8	24
33	Proteolytic cleavage of ataxin-7 promotes SCA7 retinal degeneration and neurological dysfunction. <i>Human Molecular Genetics</i> , 2015, 24, 3908-3917.	2.9	22
34	Altered Expression of Matrix Metalloproteinases and Their Endogenous Inhibitors in a Human Isogenic Stem Cell Model of Huntington's Disease. <i>Frontiers in Neuroscience</i> , 2017, 11, 736.	2.8	22
35	Modeling Polyglutamine Expansion Diseases with Induced Pluripotent Stem Cells. <i>Neurotherapeutics</i> , 2019, 16, 979-998.	4.4	21
36	Integration-independent Transgenic Huntington Disease Fragment Mouse Models Reveal Distinct Phenotypes and Life Span in Vivo. <i>Journal of Biological Chemistry</i> , 2015, 290, 19287-19306.	3.4	20

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37	Repeat Expansion Disorders: Mechanisms and Therapeutics. <i>Neurotherapeutics</i> , 2019, 16, 924-927.	4.4	20
38	FOXO3 targets are reprogrammed as Huntington's disease neural cells and striatal neurons face senescence with p16 <sup>INK4a</sup> increase. <i>Aging Cell</i> , 2020, 19, e13226.	6.7	17
39	Nuclear Receptor Nr4a1 Regulates Striatal Striosome Development and Dopamine D <sub>1</sub> Receptor Signaling. <i>ENeuro</i> , 2019, 6, ENEURO.0305-19.2019.	1.9	17
40	Autophagy: PolyQ toxic fragment turnover. <i>Autophagy</i> , 2010, 6, 312-314.	9.1	14
41	Cut to the chase. <i>Nature</i> , 2006, 442, 641-642.	27.8	12
42	Characterization and application of fluidic properties of trinucleotide repeat sequences by wax-on-plastic microfluidics. <i>Journal of Materials Chemistry B</i> , 2020, 8, 743-751.	5.8	9
43	Unbiased identification of novel transcription factors in striatal compartmentation and striosome maturation. <i>ELife</i> , 2021, 10, .	6.0	9
44	Pluripotent stem cell-derived models of neurological diseases reveal early transcriptional heterogeneity. <i>Genome Biology</i> , 2021, 22, 73.	8.8	6
45	Neuronal intranuclear inclusion disease: Polyglycine protein is the culprit. <i>Neuron</i> , 2021, 109, 1757-1760.	8.1	6
46	Using Genome Engineering to Understand Huntington's Disease. <i>Research and Perspectives in Neurosciences</i> , 2017, , 87-101.	0.4	5
47	PNA microprobe for label-free detection of expanded trinucleotide repeats. <i>RSC Advances</i> , 2022, 12, 7757-7761.	3.6	1