Lisa M Ellerby

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	PNA microprobe for label-free detection of expanded trinucleotide repeats. RSC Advances, 2022, 12, 7757-7761.	3.6	1
2	Pluripotent stem cell-derived models of neurological diseases reveal early transcriptional heterogeneity. Genome Biology, 2021, 22, 73.	8.8	6
3	Modulating FKBP5/FKBP51 and autophagy lowers HTT (huntingtin) levels. Autophagy, 2021, 17, 4119-4140.	9.1	27
4	Neuronal intranuclear inclusion disease: Polyglycine protein is the culprit. Neuron, 2021, 109, 1757-1760.	8.1	6
5	Unbiased identification of novel transcription factors in striatal compartmentation and striosome maturation. ELife, 2021, 10, .	6.0	9
6	Characterization and application of fluidic properties of trinucleotide repeat sequences by wax-on-plastic microfluidics. Journal of Materials Chemistry B, 2020, 8, 743-751.	5.8	9
7	FOXO3 targets are reprogrammed as Huntington's disease neural cells and striatal neurons face senescence with p16 ^{INK4a} increase. Aging Cell, 2020, 19, e13226.	6.7	17
8	Insulin-like growth factor 2 (IGF2) protects against Huntington's disease through the extracellular disposal of protein aggregates. Acta Neuropathologica, 2020, 140, 737-764.	7.7	43
9	Modeling Polyglutamine Expansion Diseases with Induced Pluripotent Stem Cells. Neurotherapeutics, 2019, 16, 979-998.	4.4	21
10	Repeat Expansion Disorders: Mechanisms and Therapeutics. Neurotherapeutics, 2019, 16, 924-927.	4.4	20
11	Nuclear Receptor Nr4a1 Regulates Striatal Striosome Development and Dopamine D ₁ Receptor Signaling. ENeuro, 2019, 6, ENEURO.0305-19.2019.	1.9	17
12	Mutant huntingtin impairs PNKP and ATXN3, disrupting DNA repair and transcription. ELife, 2019, 8, .	6.0	83
13	Altered Expression of Matrix Metalloproteinases and Their Endogenous Inhibitors in a Human Isogenic Stem Cell Model of Huntington's Disease. Frontiers in Neuroscience, 2017, 11, 736.	2.8	22
14	Using Genome Engineering to Understand Huntington's Disease. Research and Perspectives in Neurosciences, 2017, , 87-101.	0.4	5
15	iPSC-based drug screening for Huntington׳s disease. Brain Research, 2016, 1638, 42-56.	2.2	26
16	Genomic Analysis Reveals Disruption of Striatal Neuronal Development and Therapeutic Targets in Human Huntington's Disease Neural Stem Cells. Stem Cell Reports, 2015, 5, 1023-1038.	4.8	117
17	Proteolytic cleavage of ataxin-7 promotes SCA7 retinal degeneration and neurological dysfunction. Human Molecular Genetics, 2015, 24, 3908-3917.	2.9	22
18	Integration-independent Transgenic Huntington Disease Fragment Mouse Models Reveal Distinct Phenotypes and Life Span in Vivo. Journal of Biological Chemistry, 2015, 290, 19287-19306.	3.4	20

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19	Polyglutamine Disease Modeling: Epitope Based Screen for Homologous Recombination using CRISPR/Cas9 System. PLOS Currents, 2014, 6, .	1.4	52
20	Histone deacetylase-3 interacts with ataxin-7 and is altered in a spinocerebellar ataxia type 7 mouse model. Molecular Neurodegeneration, 2013, 8, 42.	10.8	24
21	A Genome-Scale RNA–Interference Screen Identifies RRAS Signaling as a Pathologic Feature of Huntington's Disease. PLoS Genetics, 2012, 8, e1003042.	3.5	41
22	Inhibition of Lipid Signaling Enzyme Diacylglycerol Kinase ϵ Attenuates Mutant Huntingtin Toxicity. Journal of Biological Chemistry, 2012, 287, 21204-21213.	3.4	26
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. Journal of Neuroscience, 2012, 32, 7454-7465.	3.6	46
24	Genetic Correction of Huntington's Disease Phenotypes in Induced Pluripotent Stem Cells. Cell Stem Cell, 2012, 11, 253-263.	11.1	336
25	Pizotifen Activates ERK and Provides Neuroprotection in vitro and in vivo in Models of Huntington's Disease. Journal of Huntington's Disease, 2012, 1, 195-210.	1.9	25
26	Mass Spectrometric Identification of Novel Lysine Acetylation Sites in Huntingtin. Molecular and Cellular Proteomics, 2011, 10, M111.009829.	3.8	34
27	Identification and Evaluation of Small Molecule Pan-Caspase Inhibitors in Huntington's Disease Models. Chemistry and Biology, 2010, 17, 1189-1200.	6.0	50
28	Proteolysis of Mutant Huntingtin Produces an Exon 1 Fragment That Accumulates as an Aggregated Protein in Neuronal Nuclei in Huntington Disease. Journal of Biological Chemistry, 2010, 285, 8808-8823.	3.4	259
29	Autophagy: PolyQ toxic fragment turnover. Autophagy, 2010, 6, 312-314.	9.1	14
30	Matrix Metalloproteinases Are Modifiers of Huntingtin Proteolysis and Toxicity in Huntington's Disease. Neuron, 2010, 67, 199-212.	8.1	152
31	Characterization of Human Huntington's Disease Cell Model from Induced Pluripotent Stem Cells. PLOS Currents, 2010, 2, RRN1193.	1.4	216
32	Polyglutamine-Expanded Androgen Receptor Truncation Fragments Activate a Bax-Dependent Apoptotic Cascade Mediated by DP5/Hrk. Journal of Neuroscience, 2009, 29, 1987-1997.	3.6	56
33	Posttranslational Modification of Ataxin-7 at Lysine 257 Prevents Autophagy-Mediated Turnover of an N-Terminal Caspase-7 Cleavage Fragment. Journal of Neuroscience, 2009, 29, 15134-15144.	3.6	47
34	Proteolytic Cleavage of Ataxin-7 by Caspase-7 Modulates Cellular Toxicity and Transcriptional Dysregulation. Journal of Biological Chemistry, 2007, 282, 30150-30160.	3.4	69
35	Huntingtin Interacting Proteins Are Genetic Modifiers of Neurodegeneration. PLoS Genetics, 2007, 3, e82.	3.5	368

36 Cut to the chase. Nature, 2006, 442, 641-642.

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37	Progressive phenotype and nuclear accumulation of an amino-terminal cleavage fragment in a transgenic mouse model with inducible expression of full-length mutant huntingtin. Neurobiology of Disease, 2006, 21, 381-391.	4.4	59
38	Huntingtin Phosphorylation Sites Mapped by Mass Spectrometry. Journal of Biological Chemistry, 2006, 281, 23686-23697.	3.4	131
39	Ataxin-7 Can Export from the Nucleus via a Conserved Exportin-dependent Signal. Journal of Biological Chemistry, 2006, 281, 2730-2739.	3.4	38
40	Inhibition of Calpain Cleavage of Huntingtin Reduces Toxicity. Journal of Biological Chemistry, 2004, 279, 20211-20220.	3.4	242
41	Nuclear Localization of a Non-caspase Truncation Product of Atrophin-1, with an Expanded Polyglutamine Repeat, Increases Cellular Toxicity. Journal of Biological Chemistry, 2003, 278, 13047-13055.	3.4	78
42	Kennedy's Disease. Journal of Biological Chemistry, 2003, 278, 34918-34924.	3.4	67
43	Calpain Activation in Huntington's Disease. Journal of Neuroscience, 2002, 22, 4842-4849.	3.6	282
44	Caspase Cleavage of Mutant Huntingtin Precedes Neurodegeneration in Huntington's Disease. Journal of Neuroscience, 2002, 22, 7862-7872.	3.6	344
45	Cleavage of Atrophin-1 at Caspase Site Aspartic Acid 109 Modulates Cytotoxicity. Journal of Biological Chemistry, 1999, 274, 8730-8736.	3.4	99
46	Kennedy's Disease. Journal of Neurochemistry, 1999, 72, 185-195.	3.9	211
47	Caspase Cleavage of Gene Products Associated with Triplet Expansion Disorders Generates Truncated Fragments Containing the Polyglutamine Tract. Journal of Biological Chemistry, 19 <u>98</u> , 273, 9158-9167.	3.4	499