## **Yvon Trottier**

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/1067381/publications.pdf Version: 2024-02-01

		279798	434195
31	5,924	23	31
papers	citations	h-index	g-index
33	33	33	5298
all docs	docs citations	times ranked	citing authors

#	Article	IF	CITATIONS
1	SCA7 Mouse Cerebellar Pathology Reveals Preferential Downregulation of Key Purkinje Cell-Identity Genes and Shared Disease Signature with SCA1 and SCA2. Journal of Neuroscience, 2021, 41, 4910-4936.	3.6	25
2	Gene Deregulation and Underlying Mechanisms in Spinocerebellar Ataxias With Polyglutamine Expansion. Frontiers in Neuroscience, 2020, 14, 571.	2.8	18
3	Molecular Targets and Therapeutic Strategies in Spinocerebellar Ataxia Type 7. Neurotherapeutics, 2019, 16, 1074-1096.	4.4	29
4	Loss of zebrafish Ataxin-7, a SAGA subunit responsible for SCA7 retinopathy, causes ocular coloboma and malformation of photoreceptors. Human Molecular Genetics, 2019, 28, 912-927.	2.9	12
5	Molecular Mechanisms and Therapeutic Strategies in Spinocerebellar Ataxia Type 7. Advances in Experimental Medicine and Biology, 2018, 1049, 197-218.	1.6	17
6	SynAggreg: A Multifunctional High-Throughput Technology for Precision Study of Amyloid Aggregation and Systematic Discovery of Synergistic Inhibitor Compounds. Journal of Molecular Biology, 2018, 430, 5257-5279.	4.2	3
7	A novel function of Huntingtin in the cilium and retinal ciliopathy in Huntington's disease mice. Neurobiology of Disease, 2015, 80, 15-28.	4.4	31
8	Linear and extended: a common polyglutamine conformation recognized by the three antibodies MW1, 1C2 and 3B5H10. Human Molecular Genetics, 2013, 22, 4215-4223.	2.9	37
9	Huntingtin affinity for partners is not changed by polyglutamine length: aggregation itself triggers aberrant interactions. Human Molecular Genetics, 2011, 20, 2795-2806.	2.9	28
10	Polyglutamine toxicity induces rod photoreceptor division, morphological transformation or death in Spinocerebellar ataxia 7 mouse retina. Neurobiology of Disease, 2010, 40, 311-324.	4.4	20
11	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
12	Transcriptional Activation of REST by Sp1 in Huntington's Disease Models. PLoS ONE, 2010, 5, e14311.	2.5	54
13	Stoichiometry of Base Excision Repair Proteins Correlates with Increased Somatic CAG Instability in Striatum over Cerebellum in Huntington's Disease Transgenic Mice. PLoS Genetics, 2009, 5, e1000749.	3.5	124
14	Pathogenic and Non-pathogenic Polyglutamine Tracts Have Similar Structural Properties: Towards a Length-dependent Toxicity Gradient. Journal of Molecular Biology, 2007, 371, 235-244.	4.2	86
15	Preventing polyglutamine-induced activation of c-Jun delays neuronal dysfunction in a mouse model of SCA7 retinopathy. Neurobiology of Disease, 2007, 25, 571-581.	4.4	15
16	Mapping of the epitope of monoclonal antibody 2B4 to the proline-rich region of human Huntingtin, a region critical for aggregation and toxicity. Biotechnology Journal, 2007, 2, 559-564.	3.5	14
17	Polyglutamine expansion causes neurodegeneration by altering the neuronal differentiation program. Human Molecular Genetics, 2006, 15, 691-703.	2.9	67
18	Glutamine-Expanded Ataxin-7 Alters TFTC/STAGA Recruitment and Chromatin Structure Leading to Photoreceptor Dysfunction. PLoS Biology, 2006, 4, e67.	5.6	143

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19	PML clastosomes prevent nuclear accumulation of mutant ataxin-7 and other polyglutamine proteins. Journal of Cell Biology, 2006, 174, 65-76.	5.2	82
20	Disease Progression Despite Early Loss of Polyglutamine Protein Expression in SCA7 Mouse Model. Journal of Neuroscience, 2004, 24, 1881-1887.	3.6	42
21	Hsp70 and Hsp40 Chaperones Do Not Modulate Retinal Phenotype in SCA7 Mice. Journal of Biological Chemistry, 2004, 279, 55969-55977.	3.4	30
22	Domain architecture of the polyglutamine protein ataxin-3: a globular domain followed by a flexible tail. FEBS Letters, 2003, 549, 21-25.	2.8	103
23	PML nuclear bodies and neuronal intranuclear inclusion in polyglutamine diseases. Neurobiology of Disease, 2003, 13, 230-237.	4.4	57
24	Polyglutamine Expansion Induces a Protein-damaging Stress Connecting Heat Shock Protein 70 to the JNK Pathway. Journal of Biological Chemistry, 2003, 278, 16957-16967.	3.4	67
25	Solution structure of polyglutamine tracts in GST-polyglutamine fusion proteins. FEBS Letters, 2002, 513, 267-272.	2.8	140
26	Proteases Acting on Mutant Huntingtin Generate Cleaved Products that Differentially Build Up Cytoplasmic and Nuclear Inclusions. Molecular Cell, 2002, 10, 259-269.	9.7	356
27	Two populations of neuronal intranuclear inclusions in SCA7 differ in size and promyelocytic leukaemia protein content. Brain, 2002, 125, 1534-1543.	7.6	61
28	Differential distribution of the normal and mutated forms of huntingtin in the human brain. Annals of Neurology, 1997, 42, 712-719.	5.3	48
29	Exon 1 of the HD Gene with an Expanded CAG Repeat Is Sufficient to Cause a Progressive Neurological Phenotype in Transgenic Mice. Cell, 1996, 87, 493-506.	28.9	2,892
30	Cellular localization of the Huntington's disease protein and discrimination of the normal and mutated form. Nature Genetics, 1995, 10, 104-110.	21.4	431
31	Polyglutamine expansion as a pathological epitope in Huntington's disease and four dominant cerebellar ataxias. Nature, 1995, 378, 403-406.	27.8	632