Ronald Wetzel

List of Publications by Year
in descending order

103	14,203	20036	35168
papers	citations	63 h-index	102 g-index

105	105	105	11424
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Correlative light and electron microscopy suggests that mutant huntingtin dysregulates the endolysosomal pathway in presymptomatic Huntingtonâ€™s disease. Acta Neuropathologica Communications, 2021, 9, 70.	2.4	7
2	Exploding the Repeat Length Paradigm while Exploring Amyloid Toxicity in Huntingtonâ€™s Disease. Accounts of Chemical Research, 2020, 53, 2347-2357.	7.6	25
3	RAD52 is required for RNA-templated recombination repair in post-mitotic neurons. Journal of Biological Chemistry, 2018, 293, 1353-1362.	1.6	69
4	Mutational analysis implicates the amyloid fibril as the toxic entity in Huntington's disease. Neurobiology of Disease, 2018, 120, 126-138.	2.1	37
5	An Aggregate Weight-Normalized Thioflavin-T Measurement Scale for Characterizing Polymorphic Amyloids and Assembly Intermediates. Methods in Molecular Biology, 2018, 1777, 121-144.	0.4	23
6	Fibril polymorphism affects immobilized non-amyloid flanking domains of huntingtin exon1 rather than its polyglutamine core. Nature Communications, 2017, 8, 15462.	5.8	81
7	Backbone Engineering within a Latent Î²-Hairpin Structure to Design Inhibitors of Polyglutamine Amyloid Formation. Journal of Molecular Biology, 2017, 429, 308-323.	2.0	21
8	Rapid Î±-oligomer formation mediated by the AÎ² C terminus initiates an amyloid assembly pathway. Nature Communications, 2016, 7, 12419.	5.8	51
9	Huntingtin exon 1 fibrils feature an interdigitated Î²-hairpinâ€“based polyglutamine core. Proceedings of the National Academy of Sciences of the United States of America, 2016, 113, 1546-1551.	3.3	143
10	Fluorescence Correlation Spectroscopy: A Tool to Study Protein Oligomerization and Aggregation In Vitro and In Vivo. Methods in Molecular Biology, 2016, 1345, 67-87.	0.4	19
11	C-Terminal Threonine Reduces AÎ²43 Amyloidogenicity Compared with AÎ²42. Journal of Molecular Biology, 2016, 428, 274-291.	2.0	20
12	Folding Landscape of Mutant Huntingtin Exon1: Diffusible Multimers, Oligomers and Fibrils, and No Detectable Monomer. PLoS ONE, 2016, 11, e0155747.	1.1	48
13	Huntington disease. Nature Reviews Disease Primers, 2015, 1, 15005.	18.1	1,031
14	Triosephosphate isomerase I170V alters catalytic site, enhances stability and induces pathology in a Drosophila model of TPI deficiency. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2015, 1852, 61-69.	1.8	29
15	Direct visualization of HIV-enhancing endogenous amyloid fibrils in human semen. Nature Communications, 2014, 5, 3508.	5.8	95
16	Biophysical Underpinnings of the Repeat Length Dependence of Polyglutamine Amyloid Formation. Journal of Biological Chemistry, 2014, 289, 10254-10260.	1.6	34
17	Improved chemical synthesis of hydrophobic AÎ² peptides using addition of Câ€terminal lysines later removed by carboxypeptidase B. Biopolymers, 2014, 102, 206-221.	1.2	18
18	Aggregation Behavior of Chemically Synthesized, Full-Length Huntingtin Exon1. Biochemistry, 2014, 53, 3897-3907.	1.2	37

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19	Polyglutamine Amyloid Core Boundaries and Flanking Domain Dynamics in Huntingtin Fragment Fibrils Determined by Solid-State Nuclear Magnetic Resonance. Biochemistry, 2014, 53, 6653-6666.	1.2	74
20	d-Polyglutamine Amyloid Recruits l-Polyglutamine Monomers and Kills Cells. Journal of Molecular Biology, 2014, 426, 816-829.	2.0	36
21	Structural Biology. , 2014, , .		2
22	Levels of supramolecular chirality of polyglutamine aggregates revealed by vibrational circular dichroism. FEBS Letters, 2013, 587, 1638-1643.	1.3	31
23	Î²-Hairpin-Mediated Nucleation of Polyglutamine Amyloid Formation. Journal of Molecular Biology, 2013, 425, 1183-1197.	2.0	91
24	A serendipitous survey of prediction algorithms for amyloidogenicity. Biopolymers, 2013, 100, 780-789.	1.2	21
25	Slow Amyloid Nucleation via Î±-Helix-Rich Oligomeric Intermediates in Short Polyglutamine-Containing Huntingtin Fragments. Journal of Molecular Biology, 2012, 415, 881-899.	2.0	166
26	Inhibiting the Nucleation of Amyloid Structure in a Huntingtin Fragment by Targeting Î±-Helix-Rich Oligomeric Intermediates. Journal of Molecular Biology, 2012, 415, 900-917.	2.0	76
27	Physical Chemistry of Polyglutamine: Intriguing Tales of a Monotonous Sequence. Journal of Molecular Biology, 2012, 421, 466-490.	2.0	155
28	Serine Phosphorylation Suppresses Huntingtin Amyloid Accumulation by Altering Protein Aggregation Properties. Journal of Molecular Biology, 2012, 424, 1-14.	2.0	76
29	Kinetically Competing Huntingtin Aggregation Pathways Control Amyloid Polymorphism and Properties. Biochemistry, 2012, 51, 2706-2716.	1.2	66
30	The Aggregation-Enhancing Huntingtin N-Terminus Is Helical in Amyloid Fibrils. Journal of the American Chemical Society, 2011, 133, 4558-4566.	6.6	158
31	Assays for studying nucleated aggregation of polyglutamine proteins. Methods, 2011, 53, 246-254.	1.9	29
32	Critical nucleus size for disease-related polyglutamine aggregation is repeat-length dependent. Nature Structural and Molecular Biology, 2011, 18, 328-336.	3.6	187
33	Apolipoprotein A-I Deficiency Increases Cerebral Amyloid Angiopathy and Cognitive Deficits in APP/PS1Î”E9 Mice. Journal of Biological Chemistry, 2010, 285, 36945-36957.	1.6	106
34	Structural Variations in the Cross-Î² Core of Amyloid Î² Fibrils Revealed by Deep UV Resonance Raman Spectroscopy. Journal of the American Chemical Society, 2010, 132, 6324-6328.	6.6	65
35	AÎ²(1â€“40) Forms Five Distinct Amyloid Structures whose Î²-Sheet Contents and Fibril Stabilities Are Correlated. Journal of Molecular Biology, 2010, 401, 503-517.	2.0	206
36	The impact of ataxin-1-like histidine insertions on polyglutamine aggregation. Protein Engineering, Design and Selection, 2009, 22, 469-478.	1.0	32

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37	Polyglutamine disruption of the huntingtin exon 1 N terminus triggers a complex aggregation mechanism. Nature Structural and Molecular Biology, 2009, 16, 380-389.	3.6	384
38	Serines 13 and 16 Are Critical Determinants of Full-Length Human Mutant Huntingtin Induced Disease Pathogenesis in HD Mice. Neuron, 2009, 64, 828-840.	3.8	288
39	Molecular basis for passive immunotherapy of Alzheimer's disease. Proceedings of the National Academy of Sciences of the United States of America, 2007, 104, 15659-15664.	3.3	99
40	In-cell Aggregation of a Polyglutamine-containing Chimera Is a Multistep Process Initiated by the Flanking Sequence. Journal of Biological Chemistry, 2007, 282, 36736-36743.	1.6	58
41	Plasticity of Amyloid Fibrilsâ€. Biochemistry, 2007, 46, 1-10.	1.2	115
42	Amyloid adhesins are abundant in natural biofilms. Environmental Microbiology, 2007, 9, 3077-3090.	1.8	291
43	Polymorphism in the intermediates and products of amyloid assembly. Current Opinion in Structural Biology, 2007, 17, 48-57.	2.6	349
44	Kinetics and Thermodynamics of Amyloid Assembly Using a Highâ€Performance Liquid Chromatographyâ€“Based Sedimentation Assay. Methods in Enzymology, 2006, 413, 34-74.	0.4	131
45	Hydrogen/Deuterium Exchange Mass Spectrometry Analysis of Protein Aggregates. Methods in Enzymology, 2006, 413, 140-166.	0.4	28
46	Kinetics and Thermodynamics of Amyloid Fibril Assembly. Accounts of Chemical Research, 2006, 39, 671-679.	7.6	259
47	Hydrogen/Deuterium Exchange Mass SpectrometryA Window into Amyloid Structure. Accounts of Chemical Research, 2006, 39, 584-593.	7.6	65
48	Oligoproline Effects on Polyglutamine Conformation and Aggregation. Journal of Molecular Biology, 2006, 355, 524-535.	2.0	235
49	Alanine Scanning Mutagenesis of AÎ²(1-40) Amyloid Fibril Stability. Journal of Molecular Biology, 2006, 357, 1283-1294.	2.0	154
50	Structural Differences in AÎ² Amyloid Protofibrils and Fibrils Mapped by Hydrogen Exchange â€“ Mass Spectrometry with On-line Proteolytic Fragmentation. Journal of Molecular Biology, 2006, 361, 785-795.	2.0	130
51	Nucleation of huntingtin aggregation in cells. Nature Chemical Biology, 2006, 2, 297-298.	3.9	21
52	Imaging Polyglutamine Deposits in Brain Tissue. Methods in Enzymology, 2006, 412, 106-122.	0.4	40
53	Fluorescence correlation spectroscopy shows that monomeric polyglutamine molecules form collapsed structures in aqueous solutions. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103, 16764-16769.	3.3	265
54	Scanning Cysteine Mutagenesis Analysis of AÎ²-(1-40) Amyloid Fibrils. Journal of Biological Chemistry, 2006, 281, 993-1000.	1.6	69

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55	Analysis of Amyloid Fibril Structure by Scanning Cysteine Mutagenesis. Methods in Enzymology, 2006, 413, 182-198.	0.4	9
56	Normal-repeat-length polyglutamine peptides accelerate aggregation nucleation and cytotoxicity of expanded polyglutamine proteins. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103, 14367-14372.	3.3	73
57	CA150 Expression Delays Striatal Cell Death in Overexpression and Knock-In Conditions for Mutant Huntingtin Neurotoxicity. Journal of Neuroscience, 2006, 26, 4649-4659.	1.7	48
58	Behavioral abnormalities precede neuropathological markers in rats transgenic for Huntington's disease. Human Molecular Genetics, 2006, 15, 3177-3194.	1.4	109
59	Polyglutamine homopolymers having 8-45 residues form slablike Î²-crystallite assemblies. Proteins: Structure, Function and Bioinformatics, 2005, 61, 398-411.	1.5	106
60	Structural properties of AÂ protofibrils stabilized by a small molecule. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 7115-7120.	3.3	135
61	Absence of behavioral abnormalities and neurodegeneration in vivo despite widespread neuronal huntingtin inclusions. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 11402-11407.	3.3	247
62	Polyglutamine aggregation nucleation: Thermodynamics of a highly unfavorable protein folding reaction. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 15400-15405.	3.3	154
63	Thermodynamics of AÎ²(1â˜'40) Amyloid Fibril Elongation. Biochemistry, 2005, 44, 12709-12718.	1.2	210
64	Hydrogenâ˜'Deuterium (H/D) Exchange Mapping of AÎ²1-40Amyloid Fibril Secondary Structure Using Nuclear Magnetic Resonance Spectroscopyâ€. Biochemistry, 2005, 44, 4434-4441.	1.2	124
65	Seeding Specificity in Amyloid Growth Induced by Heterologous Fibrils. Journal of Biological Chemistry, 2004, 279, 17490-17499.	1.6	377
66	Inhibition of polyglutamine aggregate cytotoxicity by a structureâ€based elongation inhibitor. FASEB Journal, 2004, 18, 923-925.	0.2	40
67	Molecular modeling of the core of AÎ² amyloid fibrils. Proteins: Structure, Function and Bioinformatics, 2004, 57, 357-364.	1.5	59
68	Kinetic analysis of beta-amyloid fibril elongation. Analytical Biochemistry, 2004, 328, 67-75.	1.1	183
69	An Intersheet Packing Interaction in AÎ² Fibrils Mapped by Disulfide Cross-Linkingâ€. Biochemistry, 2004, 43, 15310-15317.	1.2	65
70	Mapping AÎ² Amyloid Fibril Secondary Structure Using Scanning Proline Mutagenesis. Journal of Molecular Biology, 2004, 335, 833-842.	2.0	377
71	Eukaryotic Proteasomes Cannot Digest Polyglutamine Sequences and Release Them during Degradation of Polyglutamine-Containing Proteins. Molecular Cell, 2004, 14, 95-104.	4.5	363
72	Enhanced correction methods for hydrogen exchange-mass spectrometric studies of amyloid fibrils. Protein Science, 2003, 12, 635-643.	3.1	39

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73	AÎ² Protofibrils Possess a Stable Core Structure Resistant to Hydrogen Exchangeâ€. Biochemistry, 2003, 42, 14092-14098.	1.2	127
74	Conformational Abs recognizing a generic amyloid fibril epitope. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 1485-1490.	3.3	311
75	Huntington's disease age-of-onset linked to polyglutamine aggregation nucleation. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 11884-11889.	3.3	496
76	Aggregated polyglutamine peptides delivered to nuclei are toxic to mammalian cells. Human Molecular Genetics, 2002, 11, 2905-2917.	1.4	321
77	Amyloid-like Features of Polyglutamine Aggregates and Their Assembly Kineticsâ€. Biochemistry, 2002, 41, 7391-7399.	1.2	315
78	Ideas of Order for Amyloid Fibril Structure. Structure, 2002, 10, 1031-1036.	1.6	132
79	Mutational analysis of the structural organization of polyglutamine aggregates. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 17014-17019.	3.3	200
80	Structural Features of the AÎ² Amyloid Fibril Elucidated by Limited Proteolysisâ€. Biochemistry, 2001, 40, 11757-11767.	1.2	217
81	Polyglutamine aggregation behavior in vitro supports a recruitment mechanism of cytotoxicity. Journal of Molecular Biology, 2001, 311, 173-182.	2.0	310
82	A Microtiter Plate Assay for Polyglutamine Aggregate Extension. Analytical Biochemistry, 2001, 295, 227-236.	1.1	29
83	Solubilization and disaggregation of polyglutamine peptides. Protein Science, 2001, 10, 887-891.	3.1	169
84	Seeding of AÎ² Fibril Formation Is Inhibited by All Three Isotypes of Apolipoprotein E. Biochemistry, 1996, 35, 12623-12628.	1.2	117
85	Physical, Morphological and Functional Differences between pH 5.8 and 7.4 Aggregates of the Alzheimer's Amyloid Peptide A Î². Journal of Molecular Biology, 1996, 256, 870-877.	2.0	358
86	Specificity of Abnormal Assembly in Immunoglobulin Light Chain Deposition Disease and Amyloidosis. Journal of Molecular Biology, 1996, 257, 77-86.	2.0	105
87	For Protein Misassembly, It's the â€œlâ€•Decade. Cell, 1996, 86, 699-702.	13.5	161
88	Selective Inhibition of AÎ² Fibril Formation. Journal of Biological Chemistry, 1996, 271, 4086-4092.	1.6	175
89	Destabilizing loop swaps in the CDRs of an immunoglobulin V_L domain. Protein Science, 1995, 4, 2073-2081.	3.1	44
90	Prolines and Aamyloidogenicity in Fragments of the Alzheimer's Peptide .beta./A4. Biochemistry, 1995, 34, 724-730.	1.2	337

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91	Aggregation State and Neurotoxic Properties of Alzheimer Beta-Amyloid Peptide. Experimental Neurology, 1995, 4, 23-32.	1.7	178
92	Recombinant immunoglobulin variable domains generated from synthetic genes provide a system for in vitro characterization of lightâ€chain amyloid proteins. Protein Science, 1995, 4, 421-432.	3.1	98
93	Mutations and off-pathway aggregation of proteins. Trends in Biotechnology, 1994, 12, 193-198.	4.9	260
94	Inclusion body formation by interleukin-1Î² depends on the thermal sensitivity of a folding intermediate. FEBS Letters, 1994, 350, 245-248.	1.3	37
95	Breakdown in the relationship between thermal and thermodynamic stability in an interleukin-1Î² point mutant modified in a surface loop. Protein Engineering, Design and Selection, 1993, 6, 733-738.	1.0	40
96	Simplification of high-energy collision spectra of peptides by amino-terminal derivatization. Analytical Chemistry, 1993, 65, 1703-1708.	3.2	64
97	Strong inhibition of fibrinogen binding to platelet receptor Î±IIbÎ²3 by RGD sequences installed into a presentation scaffold. Protein Engineering, Design and Selection, 1993, 6, 745-754.	1.0	34
98	A general method for highly selective crosslinking of unprotected polypeptides via pH-controlled modification of N-terminal .alpha.-amino groups. Bioconjugate Chemistry, 1990, 1, 114-122.	1.8	72
99	Harnessing disulfide bonds using protein engineering. Trends in Biochemical Sciences, 1987, 12, 478-482.	3.7	120
100	Expression in escherichia coli of a chemically synthesized gene for a â€œmini-câ€•analog of human proinsulin. Gene, 1981, 16, 63-71.	1.0	51
101	Assignment of the disulphide bonds of leukocyte interferon. Nature, 1981, 289, 606-607.	13.7	108
102	Properties of a Human Alpha-Interferon Purified fromE. ColiExtracts. Journal of Interferon Research, 1981, 1, 381-390.	1.2	64
103	Production of biologically active N.alpha.-deacetylthymosin .alpha.1 in Escherichia coli through expression of a chemically synthesized gene. Biochemistry, 1980, 19, 6096-6104.	1.2	86