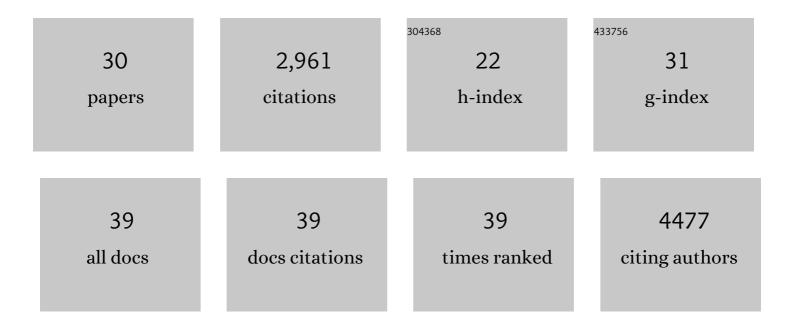
## Lin Jiang

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/8635924/publications.pdf

Version: 2024-02-01



LINHANC

#	Article	IF	CITATIONS
1	Structure of the toxic core of $\hat{I}\pm$ -synuclein from invisible crystals. Nature, 2015, 525, 486-490.	13.7	528
2	Cryo-EM of full-length α-synuclein reveals fibril polymorphs with a common structural kernel. Nature Communications, 2018, 9, 3609.	5.8	468
3	A Designed Inhibitor of p53 Aggregation Rescues p53 Tumor Suppression in Ovarian Carcinomas. Cancer Cell, 2016, 29, 90-103.	7.7	273
4	Out-of-register Î <sup>2</sup> -sheets suggest a pathway to toxic amyloid aggregates. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, 20913-20918.	3.3	184
5	Structural basis for reversible amyloids of hnRNPA1 elucidates their role in stress granule assembly. Nature Communications, 2019, 10, 2006.	5.8	157
6	Structures of fibrils formed by α-synuclein hereditary disease mutant H50Q reveal new polymorphs. Nature Structural and Molecular Biology, 2019, 26, 1044-1052.	3.6	127
7	The α-synuclein hereditary mutation E46K unlocks a more stable, pathogenic fibril structure. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 3592-3602.	3.3	122
8	Uncovering the Mechanism of Aggregation of Human Transthyretin. Journal of Biological Chemistry, 2015, 290, 28932-28943.	1.6	117
9	Atomic structures of fibrillar segments of hIAPP suggest tightly mated β-sheets are important for cytotoxicity. ELife, 2017, 6, .	2.8	95
10	Structure-based discovery of fiber-binding compounds that reduce the cytotoxicity of amyloid beta. ELife, 2013, 2, e00857.	2.8	94
11	NELL-1 in the treatment of osteoporotic bone loss. Nature Communications, 2015, 6, 7362.	5.8	93
12	Precise and Reversible Protein-Microtubule-Like Structure with Helicity Driven by Dual Supramolecular Interactions. Journal of the American Chemical Society, 2016, 138, 1932-1937.	6.6	85
13	Antiparallel Triple-strand Architecture for Prefibrillar Aβ42 Oligomers. Journal of Biological Chemistry, 2014, 289, 27300-27313.	1.6	60
14	Amyloid β-protein oligomers promote the uptake of tau fibril seeds potentiating intracellular tau aggregation. Alzheimer's Research and Therapy, 2019, 11, 86.	3.0	59
15	Mechanistic basis for receptor-mediated pathological α-synuclein fibril cell-to-cell transmission in Parkinson's disease. Proceedings of the National Academy of Sciences of the United States of America, 2021, 118, .	3.3	59
16	Structure-Based Peptide Inhibitor Design of Amyloid-β Aggregation. Frontiers in Molecular Neuroscience, 2019, 12, 54.	1.4	58
17	Inhibition of synucleinopathic seeding by rationally designed inhibitors. ELife, 2020, 9, .	2.8	54
18	A new structural model of Alzheimer's Aβ42 fibrils based on electron paramagnetic resonance data and Rosetta modeling. Journal of Structural Biology, 2016, 194, 61-67.	1.3	50

Lin Jiang

#	Article	IF	CITATIONS
19	Common fibrillar spines of amyloid-β and human islet amyloid polypeptide revealed by microelectron diffraction and structure-based inhibitors. Journal of Biological Chemistry, 2018, 293, 2888-2902.	1.6	50
20	Inhibiting amyloid-Î <sup>2</sup> cytotoxicity through its interaction with the cell surface receptor LilrB2 by structure-based design. Nature Chemistry, 2018, 10, 1213-1221.	6.6	46
21	A Proposed Mechanism for the Promotion of Prion Conversion Involving a Strictly Conserved Tyrosine Residue in the β2-α2 Loop of PrPC. Journal of Biological Chemistry, 2014, 289, 10660-10667.	1.6	37
22	Inhibiting amyloid βâ€protein assembly: Size–activity relationships among grape seedâ€derived polyphenols. Journal of Neurochemistry, 2015, 135, 416-430.	2.1	28
23	Asparagine and glutamine ladders promote cross-species prion conversion. Journal of Biological Chemistry, 2017, 292, 19076-19086.	1.6	23
24	Toward the Atomic Structure of PrP <sup>Sc</sup> . Cold Spring Harbor Perspectives in Biology, 2017, 9, a031336.	2.3	21
25	Ischemic axonal injury up-regulates MARK4 in cortical neurons and primes tau phosphorylation and aggregation. Acta Neuropathologica Communications, 2019, 7, 135.	2.4	21
26	Nicotinamide mononucleotide adenylyltransferase uses its NAD+ substrate-binding site to chaperone phosphorylated Tau. ELife, 2020, 9, .	2.8	18
27	Different Amyloid-β Self-Assemblies Have Distinct Effects on Intracellular Tau Aggregation. Frontiers in Molecular Neuroscience, 2019, 12, 268.	1.4	13
28	Annotating Protein Functional Residues by Coupling High-Throughput Fitness Profile and Homologous-Structure Analysis. MBio, 2016, 7, .	1.8	11
29	O4â€01â€01: INHIBITING AMYLOIDâ€BETA CYTOTOXICITY THROUGH ITS INTERACTION WITH THE CELL SURFACEÂRECEPTOR LILRB2 BY STRUCTUREâ€BASED DESIGN. Alzheimer's and Dementia, 2019, 15, P1229.	0.4	1
30	The mouse nicotinamide mononucleotide adenylyltransferase chaperones diverse pathological amyloid client proteins. Journal of Biological Chemistry, 2022, 298, 101912.	1.6	1

3