

Michael R Nichols

List of Publications by Year in descending order

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33
papers

1,706
citations

471509

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454955

30
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34
all docs

34
docs citations

34
times ranked

2936
citing authors

#	ARTICLE	IF	CITATIONS
1	Development of a Simple and Effective Lipid-A Antagonist Based on Computational Prediction. ACS Infectious Diseases, 2022, 8, 1171-1178.	3.8	1
2	Inhibition of matrix metalloproteinase-9 secretion by dimethyl sulfoxide and cyclic adenosine monophosphate in human monocytes. World Journal of Biological Chemistry, 2021, 12, 1-14.	4.3	2
3	The intricate biophysical puzzle of caspase-1 activation. Archives of Biochemistry and Biophysics, 2021, 699, 108753.	3.0	13
4	Expression of NLRP3 inflammasome proteins in ExpiCHO mammalian cells reveals oligomerization properties that are highly sensitive to solution conditions. Biotechnology Progress, 2021, 37, e3153.	2.6	0
5	Disentangling aggregation-prone proteins: a new method for isolating β -synuclein species. Journal of Neurochemistry, 2020, 153, 7-9.	3.9	0
6	Human and mouse single-nucleus transcriptomics reveal TREM2-dependent and TREM2-independent cellular responses in Alzheimer's disease. Nature Medicine, 2020, 26, 131-142.	30.7	641
7	Inflammatory mechanisms in neurodegeneration. Journal of Neurochemistry, 2019, 149, 562-581.	3.9	85
8	A β 242 Protofibrils Interact with and Are Trafficked through Microglial-Derived Microvesicles. ACS Chemical Neuroscience, 2018, 9, 1416-1425.	3.5	32
9	The conformational epitope for a new A β 242 protofibril-selective antibody partially overlaps with the peptide N-terminal region. Journal of Neurochemistry, 2017, 143, 736-749.	3.9	22
10	A β 240 has a subtle effect on A β 242 protofibril formation, but to a lesser degree than A β 242 concentration, in A β 242/A β 240 mixtures. Archives of Biochemistry and Biophysics, 2016, 597, 1-11.	3.0	17
11	APP Regulates Microglial Phenotype in a Mouse Model of Alzheimer's Disease. Journal of Neuroscience, 2016, 36, 8471-8486.	3.6	55
12	Amyloid- β 242 protofibrils are internalized by microglia more extensively than monomers. Brain Research, 2016, 1648, 485-495.	2.2	26
13	Biophysical Comparison of Soluble Amyloid- β 2(1-42) Protofibrils, Oligomers, and Protofilaments. Biochemistry, 2015, 54, 2193-2204.	2.5	41
14	CD47 does not mediate amyloid- β 2(1-42) protofibril-stimulated microglial cytokine release. Biochemical and Biophysical Research Communications, 2014, 454, 239-244.	2.1	9
15	The influence of gold surface texture on microglia morphology and activation. Biomaterials Science, 2014, 2, 110-120.	5.4	26
16	Amyloid- β 2(1-42) protofibrils stimulate a quantum of secreted IL-1 β despite significant intracellular IL-1 β accumulation in microglia. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2014, 1842, 2276-2285.	3.8	32
17	Amyloid- β 2(1-42) Protofibrils Formed in Modified Artificial Cerebrospinal Fluid Bind and Activate Microglia. Journal of Neuroimmune Pharmacology, 2013, 8, 312-322.	4.1	32
18	Stability of early-stage amyloid- β 2(1-42) aggregation species. Biochimica Et Biophysica Acta - Proteins and Proteomics, 2013, 1834, 65-70.	2.3	17

#	ARTICLE	IF	CITATIONS
19	A comparative first-principles study of structural and electronic properties among memantine, amantadine and rimantadine. <i>Molecular Physics</i> , 2012, 110, 685-689.	1.7	2
20	Introduction. <i>Life Sciences</i> , 2012, 91, 1140.	4.3	1
21	Isolated Amyloid- β (1-42) Protofibrils, But Not Isolated Fibrils, Are Robust Stimulators of Microglia. <i>ACS Chemical Neuroscience</i> , 2012, 3, 302-311.	3.5	62
22	Development of LPS antagonistic therapeutics: synthesis and evaluation of glucopyranoside-spacer-amino acid motifs. <i>RSC Advances</i> , 2011, 1, 83.	3.6	10
23	Substituted tryptophans at amyloid- β (1-40) residues 19 and 20 experience different environments after fibril formation. <i>Archives of Biochemistry and Biophysics</i> , 2011, 514, 27-32.	3.0	6
24	Special issue on Alzheimer's disease:. <i>Life Sciences</i> , 2011, 89, 288.	4.3	0
25	Probing the amyloid- β (1-40) fibril environment with substituted tryptophan residues. <i>Archives of Biochemistry and Biophysics</i> , 2010, 494, 192-197.	3.0	13
26	Oligomeric amyloid- β (1-42) induces THP-1 human monocyte adhesion and maturation. <i>Brain Research</i> , 2009, 1254, 109-119.	2.2	13
27	Amyloid- β (1-42) Fibrillar Precursors Are Optimal for Inducing Tumor Necrosis Factor- α Production in the THP-1 Human Monocytic Cell Line. <i>Biochemistry</i> , 2009, 48, 9011-9021.	2.5	19
28	Toll-like receptors 2 and 4 mediate A β (1-42) activation of the innate immune response in a human monocytic cell line. <i>Journal of Neurochemistry</i> , 2008, 104, 524-533.	3.9	146
29	Amyloid- β aggregates formed at polar-nonpolar interfaces differ from amyloid- β protofibrils produced in aqueous buffers. <i>Microscopy Research and Technique</i> , 2005, 67, 164-174.	2.2	34
30	Amyloid- β Protofibrils Differ from Amyloid- β Aggregates Induced in Dilute Hexafluoroisopropanol in Stability and Morphology. <i>Journal of Biological Chemistry</i> , 2005, 280, 2471-2480.	3.4	100
31	Rapid Assembly of Amyloid- β Peptide at a Liquid/Liquid Interface Produces Unstable β -Sheet Fibers. <i>Biochemistry</i> , 2005, 44, 165-173.	2.5	40
32	The Peptide KLVFF-K6 Promotes β -Amyloid(1-40) Protofibril Growth by Association but Does Not Alter Protofibril Effects on Cellular Reduction of 3-(4,5-Dimethylthiazol-2-yl)-2,5-diphenyltetrazolium Bromide (MTT). <i>Molecular Pharmacology</i> , 2003, 64, 1160-1168.	2.3	23
33	Growth of β -Amyloid(1-40) Protofibrils by Monomer Elongation and Lateral Association. Characterization of Distinct Products by Light Scattering and Atomic Force Microscopy. <i>Biochemistry</i> , 2002, 41, 6115-6127.	2.5	180