## Michael Willem

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Glitter in the Darkness? Nonfibrillar β-Amyloid Plaque Components Significantly Impact the β-Amyloid PET Signal in Mouse Models of Alzheimer Disease. Journal of Nuclear Medicine, 2022, 63, 117-124.	5.0	14
2	A molecular view of human amyloid-β folds. Science, 2022, 375, 147-148.	12.6	7
3	Chronic PPARÎ <sup>3</sup> Stimulation Shifts Amyloidosis to Higher Fibrillarity but Improves Cognition. Frontiers in Aging Neuroscience, 2022, 14, 854031.	3.4	5
4	Novel App knock-in mouse model shows key features of amyloid pathology and reveals profound metabolic dysregulation of microglia. Molecular Neurodegeneration, 2022, 17, .	10.8	26
5	The β-Secretase BACE1 in Alzheimer's Disease. Biological Psychiatry, 2021, 89, 745-756.	1.3	336
6	Pre-therapeutic microglia activation and sex determine therapy effects of chronic immunomodulation. Theranostics, 2021, 11, 8964-8976.	10.0	12
7	Microglial activation in the right amygdala-entorhinal-hippocampal complex is associated with preserved spatial learning in App mice. Neurolmage, 2021, 230, 117707.	4.2	16
8	Microglial activation states drive glucose uptake and FDG-PET alterations in neurodegenerative diseases. Science Translational Medicine, 2021, 13, eabe5640.	12.4	108
9	Asymmetry of Fibrillar Plaque Burden in Amyloid Mouse Models. Journal of Nuclear Medicine, 2020, 61, 1825-1831.	5.0	19
10	Enhancing protective microglial activities with a dual function <scp>TREM</scp> 2 antibody to the stalk region. EMBO Molecular Medicine, 2020, 12, e11227.	6.9	155
11	Transgenic Overexpression of the Disordered Prion Protein N1 Fragment in Mice Does Not Protect Against Neurodegenerative Diseases Due to Impaired ER Translocation. Molecular Neurobiology, 2020, 57, 2812-2829.	4.0	17
12	Fibrillar AÎ <sup>2</sup> triggers microglial proteome alterations and dysfunction in Alzheimer mouse models. ELife, 2020, 9, .	6.0	80
13	Aβ-induced acceleration of Alzheimer-related τ-pathology spreading and its association with prion protein. Acta Neuropathologica, 2019, 138, 913-941.	7.7	75
14	Lack of β-amyloid cleaving enzyme-1 (BACE1) impairs long-term synaptic plasticity but enhances granule cell excitability and oscillatory activity in the dentate gyrus in vivo. Brain Structure and Function, 2019, 224, 1279-1290.	2.3	9
15	Secreted APP Modulates Synaptic Activity: A Novel Target for Therapeutic Intervention?. Neuron, 2019, 101, 557-559.	8.1	14
16	Loss of TREM2 function increases amyloid seeding but reduces plaque-associated ApoE. Nature Neuroscience, 2019, 22, 191-204.	14.8	358
17	Amyloid precursor protein-fragments-containing inclusions in cardiomyocytes with basophilic degeneration and its association with cerebral amyloid angiopathy and myocardial fibrosis. Scientific Reports, 2018, 8, 16594.	3.3	11
18	Efficacy of chronic BACE1 inhibition in PS2APP mice depends on the regional AÎ <sup>2</sup> deposition rate and plaque burden at treatment initiation. Theranostics, 2018, 8, 4957-4968.	10.0	22

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19	Young microglia restore amyloid plaque clearance of aged microglia. EMBO Journal, 2017, 36, 583-603.	7.8	124
20	Tetraspanin 3: A central endocytic membrane component regulating the expression of ADAM10, presenilin and the amyloid precursor protein. Biochimica Et Biophysica Acta - Molecular Cell Research, 2017, 1864, 217-230.	4.1	26
21	Physiological and pathophysiological control of synaptic GluN2B-NMDA receptors by the C-terminal domain of amyloid precursor protein. ELife, 2017, 6, .	6.0	29
22	Proteolytic processing of Neuregulin-1. Brain Research Bulletin, 2016, 126, 178-182.	3.0	37
23	Specific Inhibition of β-Secretase Processing of the Alzheimer Disease Amyloid Precursor Protein. Cell Reports, 2016, 14, 2127-2141.	6.4	87
24	Proteolytic Processing of Neuregulin 1 Type III by Three Intramembrane-cleaving Proteases. Journal of Biological Chemistry, 2016, 291, 318-333.	3.4	42
25	ÎSecretase processing of APP inhibits neuronal activity in the hippocampus. Nature, 2015, 526, 443-447.	27.8	308
26	TREM2 mutations implicated in neurodegeneration impair cell surface transport and phagocytosis. Science Translational Medicine, 2014, 6, 243ra86.	12.4	600
27	Postnatal Disruption of the Disintegrin/Metalloproteinase ADAM10 in Brain Causes Epileptic Seizures, Learning Deficits, Altered Spine Morphology, and Defective Synaptic Functions. Journal of Neuroscience, 2013, 33, 12915-12928.	3.6	107
28	Loss of Bace2 in zebrafish affects melanocyte migration and is distinct from Bace1 knock out phenotypes. Journal of Neurochemistry, 2013, 127, 471-481.	3.9	56
29	Dual Cleavage of Neuregulin 1 Type III by BACE1 and ADAM17 Liberates Its EGF-Like Domain and Allows Paracrine Signaling. Journal of Neuroscience, 2013, 33, 7856-7869.	3.6	104
30	Bace1 and Neuregulin-1 cooperate to control formation and maintenance of muscle spindles. EMBO Journal, 2013, 32, 2015-2028.	7.8	122
31	BACE1 Dependent Neuregulin Processing: Review. Current Alzheimer Research, 2012, 9, 178-183.	1.4	62
32	Mitochondrion-Derived Reactive Oxygen Species Lead to Enhanced Amyloid Beta Formation. Antioxidants and Redox Signaling, 2012, 16, 1421-1433.	5.4	273
33	Secretome protein enrichment identifies physiological BACE1 protease substrates in neurons. EMBO Journal, 2012, 31, 3157-3168.	7.8	279
34	Bace1 processing of NRG1 type III produces a myelinâ€inducing signal but is not essential for the stimulation of myelination. Glia, 2012, 60, 203-217.	4.9	73
35	PPARÎ <sup>3</sup> Co-Activator-1α (PGC-1α) Reduces Amyloid-Î <sup>2</sup> Generation Through a PPARÎ <sup>3</sup> -Dependent Mechanism. Journal of Alzheimer's Disease, 2011, 25, 151-162.	2.6	104
36	Translational Repression of the Disintegrin and Metalloprotease ADAM10 by a Stable G-quadruplex Secondary Structure in Its 5â€2-Untranslated Region. Journal of Biological Chemistry, 2011, 286, 45063-45072.	3.4	68

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37	Rescue of Progranulin Deficiency Associated with Frontotemporal Lobar Degeneration by Alkalizing Reagents and Inhibition of Vacuolar ATPase. Journal of Neuroscience, 2011, 31, 1885-1894.	3.6	121
38	Destruxin E Decreases Beta-Amyloid Generation by Reducing Colocalization of Beta-Amyloid-Cleaving Enzyme 1 and Beta-Amyloid Protein Precursor. Neurodegenerative Diseases, 2009, 6, 230-239.	1.4	9
39	Function, regulation and therapeutic properties of β-secretase (BACE1). Seminars in Cell and Developmental Biology, 2009, 20, 175-182.	5.0	73
40	Macrocyclic Statineâ€Based Inhibitors of BACEâ€1. ChemBioChem, 2007, 8, 2078-2091.	2.6	22
41	Amyloid precursor protein intracellular domain modulates cellular calcium homeostasis and ATP content. Journal of Neurochemistry, 2007, 102, 1264-1275.	3.9	56
42	Control of Peripheral Nerve Myelination by the ß-Secretase BACE1. Science, 2006, 314, 664-666.	12.6	652
43	Amyloid Precursor Protein and Notch Intracellular Domains are Generated after Transport of their Precursors to the Cell Surface. Traffic, 2006, 7, 408-415.	2.7	133
44	Absence of α7 integrin in dystrophin-deficient mice causes a myopathy similar to Duchenne muscular dystrophy. Human Molecular Genetics, 2006, 15, 989-998.	2.9	97
45	Basement Membrane–Dependent Survival of Retinal Ganglion Cells. , 2005, 46, 1000.		70
46	Dimerization of β-Site β-Amyloid Precursor Protein-cleaving Enzyme. Journal of Biological Chemistry, 2004, 279, 53205-53212.	3.4	103
47	Identification of a β-Secretase Activity, Which Truncates Amyloid β-Peptide after Its Presenilin-dependent Generation. Journal of Biological Chemistry, 2003, 278, 5531-5538.	3.4	62
48	Defective integrin switch and matrix composition at alpha 7-deficient myotendinous junctions precede the onset of muscular dystrophy in mice. Human Molecular Genetics, 2003, 12, 483-495.	2.9	42
49	Constitutive properties, not molecular adaptations, mediate extraocular muscle sparing in dystrophicmdxmice. FASEB Journal, 2003, 17, 1-27.	0.5	66
50	Insulin-degrading Enzyme Rapidly Removes the β-Amyloid Precursor Protein Intracellular Domain (AICD). Journal of Biological Chemistry, 2002, 277, 13389-13393.	3.4	185
51	A Critical Function of the Pial Basement Membrane in Cortical Histogenesis. Journal of Neuroscience, 2002, 22, 6029-6040.	3.6	261
52	A nonâ€amyloidogenic function of BACEâ€2 in the secretory pathway. Journal of Neurochemistry, 2002, 81, 1011-1020.	3.9	99
53	A γâ€secretase inhibitor blocks Notch signaling <i>in vivo</i> and causes a severe neurogenic phenotype in zebrafish. EMBO Reports, 2002, 3, 688-694.	4.5	459
54	Migratory Activity and Functional Changes of Green Fluorescent Effector Cells before and during Experimental Autoimmune Encephalomyelitis. Immunity, 2001, 14, 547-560.	14.3	428

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55	Phosphorylation Regulates Intracellular Trafficking of β-Secretase. Journal of Biological Chemistry, 2001, 276, 14634-14641.	3.4	248
56	Maturation and Pro-peptide Cleavage of β-Secretase. Journal of Biological Chemistry, 2000, 275, 30849-30854.	3.4	229
57	Gene transfer into CD4+ T lymphocytes: Green fluorescent protein-engineered, encephalitogenic T cells illuminate brain autoimmune responses. Nature Medicine, 1999, 5, 843-847.	30.7	135