

# Hermann C Altmeppen

## List of Publications by Year in descending order

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Version: 2024-02-01

42  
papers

1,597  
citations

331259

21  
h-index

315357

38  
g-index

48  
all docs

48  
docs citations

48  
times ranked

2206  
citing authors

#	ARTICLE	IF	CITATIONS
1	Anchorless risk or released benefit? An updated view on the ADAM10-mediated shedding of the prion protein. <i>Cell and Tissue Research</i> , 2023, 392, 215-234.	1.5	4
2	NeuroCOVID: Insights into Neuroinvasion and Pathophysiology. <i>Clinical and Translational Neuroscience</i> , 2022, 6, 10.	0.4	1
3	The prion protein and its ligands: Insights into structure-function relationships. <i>Biochimica Et Biophysica Acta - Molecular Cell Research</i> , 2022, 1869, 119240.	1.9	10
4	Autosomal dominantly inherited myopathy likely caused by the <i>TNNT1</i> variant p.(Asp65Ala). <i>Human Mutation</i> , 2022, 43, 1224-1233.	1.1	0
5	Multiplexed mRNA analysis of brain-derived extracellular vesicles upon experimental stroke in mice reveals increased mRNA content with potential relevance to inflammation and recovery processes. <i>Cellular and Molecular Life Sciences</i> , 2022, 79, .	2.4	6
6	Pharmacological inactivation of the prion protein by targeting a folding intermediate. <i>Communications Biology</i> , 2021, 4, 62.	2.0	30
7	Disordered structure and flexible roles: using the prion protein N1 fragment for neuroprotective and regenerative therapy. <i>Neural Regeneration Research</i> , 2021, 16, 1431.	1.6	3
8	Brain-Derived Extracellular Vesicles in Health and Disease: A Methodological Perspective. <i>International Journal of Molecular Sciences</i> , 2021, 22, 1365.	1.8	17
9	Prion protein oligomers cause neuronal cytoskeletal damage in rapidly progressive Alzheimer's disease. <i>Molecular Neurodegeneration</i> , 2021, 16, 11.	4.4	15
10	The SARS-CoV-2 main protease Mpro causes microvascular brain pathology by cleaving NEMO in brain endothelial cells. <i>Nature Neuroscience</i> , 2021, 24, 1522-1533.	7.1	164
11	Ligands binding to the prion protein induce its proteolytic release with therapeutic potential in neurodegenerative proteinopathies. <i>Science Advances</i> , 2021, 7, eabj1826.	4.7	18
12	Shortening heparan sulfate chains prolongs survival and reduces parenchymal plaques in prion disease caused by mobile, ADAM10-cleaved prions. <i>Acta Neuropathologica</i> , 2020, 139, 527-546.	3.9	23
13	Characterization of brain-derived extracellular vesicles reveals changes in cellular origin after stroke and enrichment of the prion protein with a potential role in cellular uptake. <i>Journal of Extracellular Vesicles</i> , 2020, 9, 1809065.	5.5	47
14	Prion protein post-translational modifications modulate heparan sulfate binding and limit aggregate size in prion disease. <i>Neurobiology of Disease</i> , 2020, 142, 104955.	2.1	5
15	Show Me Your Friends and I Tell You Who You Are: The Many Facets of Prion Protein in Stroke. <i>Cells</i> , 2020, 9, 1609.	1.8	6
16	Transgenic Overexpression of the Disordered Prion Protein N1 Fragment in Mice Does Not Protect Against Neurodegenerative Diseases Due to Impaired ER Translocation. <i>Molecular Neurobiology</i> , 2020, 57, 2812-2829.	1.9	17
17	Prion protein glycans reduce intracerebral fibril formation and spongiosis in prion disease. <i>Journal of Clinical Investigation</i> , 2020, 130, 1350-1362.	3.9	32
18	The cellular prion protein and its derived fragments in human prion diseases and their role as potential biomarkers. <i>Expert Review of Molecular Diagnostics</i> , 2019, 19, 1007-1018.	1.5	5

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19	GPI-anchor signal sequence influences PrPC sorting, shedding and signalling, and impacts on different pathomechanistic aspects of prion disease in mice. <i>PLoS Pathogens</i> , 2019, 15, e1007520.	2.1	34
20	Infectious prions do not induce A $\beta$ deposition in an in vivo seeding model. <i>Acta Neuropathologica</i> , 2018, 135, 965-967.	3.9	8
21	Structural and mechanistic aspects influencing the ADAM10-mediated shedding of the prion protein. <i>Molecular Neurodegeneration</i> , 2018, 13, 18.	4.4	45
22	In vivo regulation of the A disintegrin and metalloproteinase 10 (ADAM10) by the tetraspanin 15. <i>Cellular and Molecular Life Sciences</i> , 2018, 75, 3251-3267.	2.4	37
23	Muskelin Coordinates PrPC Lysosome versus Exosome Targeting and Impacts Prion Disease Progression. <i>Neuron</i> , 2018, 99, 1155-1169.e9.	3.8	39
24	Diverse functions of the prion protein â€œ Does proteolytic processing hold the key?. <i>Biochimica Et Biophysica Acta - Molecular Cell Research</i> , 2017, 1864, 2128-2137.	1.9	60
25	Tetraspanin 3: A central endocytic membrane component regulating the expression of ADAM10, presenilin and the amyloid precursor protein. <i>Biochimica Et Biophysica Acta - Molecular Cell Research</i> , 2017, 1864, 217-230.	1.9	26
26	Exosomes in Prion Diseases. <i>Neuromethods</i> , 2017, , 197-207.	0.2	0
27	Exosomal cellular prion protein drives fibrillization of amyloid beta and counteracts amyloid betaâ€‘mediated neurotoxicity. <i>Journal of Neurochemistry</i> , 2016, 137, 88-100.	2.1	117
28	Misfolding leads the way to unraveling signaling pathways in the pathophysiology of prion diseases. <i>Prion</i> , 2016, 10, 434-443.	0.9	2
29	Secretory pathway retention of mutant prion protein induces p38-MAPK activation and lethal disease in mice. <i>Scientific Reports</i> , 2016, 6, 24970.	1.6	22
30	Generation of aggregation prone N-terminally truncated amyloid $\beta$ peptides by meprin $\beta$ depends on the sequence specificity at the cleavage site. <i>Molecular Neurodegeneration</i> , 2016, 11, 19.	4.4	65
31	Shedding light on prion disease. <i>Prion</i> , 2015, 9, 244-256.	0.9	17
32	The sheddase ADAM10 is a potent modulator of prion disease. <i>ELife</i> , 2015, 4, .	2.8	66
33	The GPI-anchoring of PrP. <i>Prion</i> , 2014, 8, 11-18.	0.9	49
34	LIMP-2 expression is critical for $\beta$ -glucocerebrosidase activity and $\alpha$ -synuclein clearance. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2014, 111, 15573-15578.	3.3	109
35	High molecular mass assemblies of amyloid- $\beta$ oligomers bind prion protein in patients with Alzheimerâ€™s disease. <i>Brain</i> , 2014, 137, 873-886.	3.7	96
36	Postnatal Disruption of the Disintegrin/Metalloproteinase ADAM10 in Brain Causes Epileptic Seizures, Learning Deficits, Altered Spine Morphology, and Defective Synaptic Functions. <i>Journal of Neuroscience</i> , 2013, 33, 12915-12928.	1.7	107

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37	Roles of endoproteolytic cleavage and shedding of the prion protein in neurodegeneration. FEBS Journal, 2013, 280, 4338-4347.	2.2	48
38	Activation of the Nipah Virus Fusion Protein in MDCK Cells Is Mediated by Cathepsin B within the Endosome-Recycling Compartment. Journal of Virology, 2012, 86, 3736-3745.	1.5	58
39	Proteolytic processing of the prion protein in health and disease. American Journal of Neurodegenerative Disease, 2012, 1, 15-31.	0.1	58
40	Lack of a-disintegrin-and-metalloproteinase ADAM10 leads to intracellular accumulation and loss of shedding of the cellular prion protein in vivo. Molecular Neurodegeneration, 2011, 6, 36.	4.4	93
41	N-Glycans and Glycosylphosphatidylinositol-Anchor Act on Polarized Sorting of Mouse PrPC in Madin-Darby Canine Kidney Cells. PLoS ONE, 2011, 6, e24624.	1.1	19
42	Young COVID-19 Patients Show a Higher Degree of Microglial Activation When Compared to Controls. Frontiers in Neurology, 0, 13, .	1.1	7