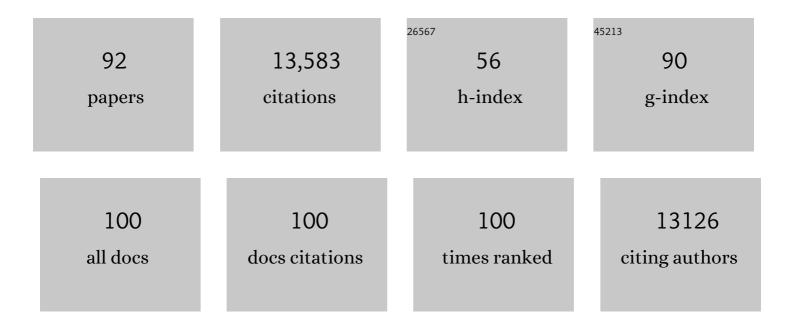
Dieter Edbauer

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/5834869/publications.pdf Version: 2024-02-01



NIETED FORALIED

#	Article	IF	CITATIONS
1	Diseaseâ€linked TDPâ€43 hyperphosphorylation suppresses TDPâ€43 condensation and aggregation. EMBO Journal, 2022, 41, e108443.	3.5	68
2	Gelâ€like inclusions of Câ€terminal fragments of TDPâ€43 sequester stalled proteasomes in neurons. EMBO Reports, 2022, 23, e53890.	2.0	28
3	Drug screen in iPSC-Neurons identifies nucleoside analogs as inhibitors of (G4C2)n expression in C9orf72 ALS/FTD. Cell Reports, 2022, 39, 110913.	2.9	7
4	Multi-omics profiling identifies a deregulated FUS-MAP1B axis in ALS/FTD–associated UBQLN2 mutants. Life Science Alliance, 2022, 5, e202101327.	1.3	6
5	Chronic T cell proliferation in brains after stroke could interfere with the efficacy of immunotherapies. Journal of Experimental Medicine, 2021, 218, .	4.2	26
6	Low-degree trisomy 21 mosaicism promotes early-onset Alzheimer disease. Neurobiology of Aging, 2021, 103, 147.e1-147.e5.	1.5	4
7	The porphyrin TMPyP4 inhibits elongation during the noncanonical translation of the FTLD/ALS-associated GGGGCC repeat in the C9orf72 gene. Journal of Biological Chemistry, 2021, 297, 101120.	1.6	17
8	Poly-glycine–alanine exacerbates C9orf72 repeat expansion-mediated DNA damage via sequestration of phosphorylated ATM and loss of nuclear hnRNPA3. Acta Neuropathologica, 2020, 139, 99-118.	3.9	49
9	Active polyâ€GA vaccination prevents microglia activation and motor deficits in a <i>C9orf72</i> mouse model. EMBO Molecular Medicine, 2020, 12, e10919.	3.3	39
10	Nuclear Import Receptors Directly Bind to Arginine-Rich Dipeptide Repeat Proteins and Suppress Their Pathological Interactions. Cell Reports, 2020, 33, 108538.	2.9	69
11	Congenic expression of poly-GA but not poly-PR in mice triggers selective neuron loss and interferon responses found in C9orf72 ALS. Acta Neuropathologica, 2020, 140, 121-142.	3.9	44
12	Cellâ€ŧoâ€eell transmission of <i>C9orf72</i> polyâ€(Glyâ€Ala) triggers key features of <scp>ALS</scp> / <scp>FTD</scp> . EMBO Journal, 2020, 39, e102811.	3.5	51
13	Synaptic dysfunction induced by glycineâ€alanine dipeptides in C9orf72― <scp>ALS</scp> / <scp>FTD</scp> is rescued by <scp>SV</scp> 2 replenishment. EMBO Molecular Medicine, 2020, 12, e10722.	3.3	38
14	Knocking out C9ORF72 Exacerbates Axonal Trafficking Defects Associated with Hexanucleotide Repeat Expansion and Reduces Levels of Heat Shock Proteins. Stem Cell Reports, 2020, 14, 390-405.	2.3	48
15	Loss of <scp>TMEM</scp> 106B potentiates lysosomal and <scp>FTLD</scp> â€like pathology in progranulinâ€deficient mice. EMBO Reports, 2020, 21, e50241.	2.0	37
16	FDG-PET underscores the key role of the thalamus in frontotemporal lobar degeneration caused by C9ORF72 mutations. Translational Psychiatry, 2019, 9, 54.	2.4	28
17	Genome-wide analyses as part of the international FTLD-TDP whole-genome sequencing consortium reveals novel disease risk factors and increases support for immune dysfunction in FTLD. Acta Neuropathologica, 2019, 137, 879-899.	3.9	90
18	Loss of TREM2 function increases amyloid seeding but reduces plaque-associated ApoE. Nature Neuroscience, 2019, 22, 191-204.	7.1	358

#	Article	IF	CITATIONS
19	RNA-Dependent Intergenerational Inheritance of Enhanced Synaptic Plasticity after Environmental Enrichment. Cell Reports, 2018, 23, 546-554.	2.9	113
20	RNA versus protein toxicity in C9orf72 ALS/FTLD. Acta Neuropathologica, 2018, 135, 475-479.	3.9	8
21	In Situ Structure of Neuronal C9orf72 Poly-GA Aggregates Reveals Proteasome Recruitment. Cell, 2018, 172, 696-705.e12.	13.5	311
22	Targeting RNA Gâ€quadruplexes as new treatment strategy for C9orf72 ALS / FTD. EMBO Molecular Medicine, 2018, 10, 4-6.	3.3	10
23	A novel CHCHD10 mutation implicates a Mia40â€dependent mitochondrial import deficit in ALS. EMBO Molecular Medicine, 2018, 10, .	3.3	43
24	Novel antibodies reveal presynaptic localization of C9orf72 protein and reduced protein levels in C9orf72 mutation carriers. Acta Neuropathologica Communications, 2018, 6, 72.	2.4	87
25	Atrophy in the Thalamus But Not Cerebellum Is Specific for C9orf72 FTD and ALS Patients – An Atlas-Based Volumetric MRI Study. Frontiers in Aging Neuroscience, 2018, 10, 45.	1.7	40
26	Proteomics and <i>C9orf72</i> neuropathology identify ribosomes as poly-GR/PR interactors driving toxicity. Life Science Alliance, 2018, 1, e201800070.	1.3	88
27	Cytoplasmic poly-GA aggregates impair nuclear import of TDP-43 in <i>C9orf72</i> ALS/FTLD. Human Molecular Genetics, 2017, 26, ddw432.	1.4	82
28	Glycine-alanine dipeptide repeat protein contributes to toxicity in a zebrafish model of C9orf72 associated neurodegeneration. Molecular Neurodegeneration, 2017, 12, 6.	4.4	57
29	Spinal poly-GA inclusions in a C9orf72 mouse model trigger motor deficits and inflammation without neuron loss. Acta Neuropathologica, 2017, 134, 241-254.	3.9	99
30	Polyâ€ <scp>GP</scp> in cerebrospinal fluid links <i>C9orf72</i> â€associated dipeptide repeat expression to the asymptomatic phase of <scp>ALS</scp> / <scp>FTD</scp> . EMBO Molecular Medicine, 2017, 9, 859-868.	3.3	90
31	Antibodies inhibit transmission and aggregation of <i>C9orf72</i> poly― <scp>GA</scp> dipeptide repeat proteins. EMBO Molecular Medicine, 2017, 9, 687-702.	3.3	70
32	[S5–01–03]: C9ORF72 TRANSLATION AND DISEASE. Alzheimer's and Dementia, 2017, 13, P1444.	0.4	0
33	TDPâ€43 loss of function inhibits endosomal trafficking and alters trophic signaling in neurons. EMBO Journal, 2016, 35, 2350-2370.	3.5	76
34	Reduced hn <scp>RNPA</scp> 3 increases <i>C9orf72</i> repeat <scp>RNA</scp> levels and dipeptideâ€repeat protein deposition. EMBO Reports, 2016, 17, 1314-1325.	2.0	39
35	Gain of Toxicity from ALS/FTD-Linked Repeat Expansions in C9ORF72 Is Alleviated by Antisense Oligonucleotides Targeting GGGGCC-Containing RNAs. Neuron, 2016, 90, 535-550.	3.8	437
36	C9ORF72 poly(GA) aggregates sequester and impair HR23 and nucleocytoplasmic transport proteins. Nature Neuroscience, 2016, 19, 668-677.	7.1	268

#	Article	IF	CITATIONS
37	Monomethylated and unmethylated FUS exhibit increased binding to Transportin and distinguish FTLD-FUS from ALS-FUS. Acta Neuropathologica, 2016, 131, 587-604.	3.9	76
38	An amyloid-like cascade hypothesis for C9orf72 ALS/FTD. Current Opinion in Neurobiology, 2016, 36, 99-106.	2.0	59
39	Proteolytic Processing of Neuregulin 1 Type III by Three Intramembrane-cleaving Proteases. Journal of Biological Chemistry, 2016, 291, 318-333.	1.6	42
40	The C9orf72 repeat size correlates with onset age of disease, DNA methylation and transcriptional downregulation of the promoter. Molecular Psychiatry, 2016, 21, 1112-1124.	4.1	201
41	Distribution of dipeptide repeat proteins in cellular models and C9orf72 mutation cases suggests link to transcriptional silencing. Acta Neuropathologica, 2015, 130, 537-555.	3.9	157
42	<i>C9ORF72</i> repeat expansions in mice cause TDP-43 pathology, neuronal loss, and behavioral deficits. Science, 2015, 348, 1151-1154.	6.0	332
43	Editorial. Journal of Neural Transmission, 2015, 122, 933-936.	1.4	Ο
44	Novel clinical associations with specific C9ORF72 transcripts in patients with repeat expansions in C9ORF72. Acta Neuropathologica, 2015, 130, 863-876.	3.9	104
45	Cerebellar c9RAN proteins associate with clinical and neuropathological characteristics of C9ORF72 repeat expansion carriers. Acta Neuropathologica, 2015, 130, 559-573.	3.9	89
46	Quantitative analysis and clinico-pathological correlations of different dipeptide repeat protein pathologies in C9ORF72 mutation carriers. Acta Neuropathologica, 2015, 130, 845-861.	3.9	204
47	Three novel presenilin 1 mutations marking the wide spectrum of age at onset and clinical patterns in familial Alzheimer's disease. Journal of Neural Transmission, 2015, 122, 1715-1719.	1.4	8
48	C9orf72 FTLD/ALS-associated Gly-Ala dipeptide repeat proteins cause neuronal toxicity and Unc119 sequestration. Acta Neuropathologica, 2014, 128, 485-503.	3.9	300
49	The ER under rapid fire. EMBO Journal, 2014, 33, 1195-7.	3.5	4
50	Sequestration of multiple RNA recognition motif-containing proteins by C9orf72 repeat expansions. Brain, 2014, 137, 2040-2051.	3.7	253
51	Micro <scp>RNA</scp> â€125b induces tau hyperphosphorylation and cognitive deficits in Alzheimer's disease. EMBO Journal, 2014, 33, 1667-1680.	3.5	257
52	Early dipeptide repeat pathology in a frontotemporal dementia kindred with C9ORF72 mutation and intellectual disability. Acta Neuropathologica, 2014, 127, 451-458.	3.9	67
53	Good guy or bad guy: the opposing roles of microRNA 125b in cancer. Cell Communication and Signaling, 2014, 12, 30.	2.7	144
54	Promoter DNA methylation regulates progranulin expression and is altered in FTLD. Acta Neuropathologica Communications, 2013, 1, 16.	2.4	43

#	Article	IF	CITATIONS
55	Bidirectional transcripts of the expanded C9orf72 hexanucleotide repeat are translated into aggregating dipeptide repeat proteins. Acta Neuropathologica, 2013, 126, 881-893.	3.9	427
56	FUS-mediated alternative splicing in the nervous system: consequences for ALS and FTLD. Journal of Molecular Medicine, 2013, 91, 1343-1354.	1.7	39
57	Staufen2 Regulates Neuronal Target RNAs. Cell Reports, 2013, 5, 1511-1518.	2.9	78
58	The <i>C9orf72</i> GGGGCC Repeat Is Translated into Aggregating Dipeptide-Repeat Proteins in FTLD/ALS. Science, 2013, 339, 1335-1338.	6.0	1,095
59	hnRNP A3 binds to GGGGCC repeats and is a constituent of p62-positive/TDP43-negative inclusions in the hippocampus of patients with C9orf72 mutations. Acta Neuropathologica, 2013, 125, 413-423.	3.9	302
60	Loss of ALS-associated TDP-43 in zebrafish causes muscle degeneration, vascular dysfunction, and reduced motor neuron axon outgrowth. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, 4986-4991.	3.3	126
61	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.	1.7	104
62	Dipeptide repeat protein pathology in C9ORF72 mutation cases: clinico-pathological correlations. Acta Neuropathologica, 2013, 126, 859-879.	3.9	298
63	The FTLD risk factor TMEM106B and MAP6 control dendritic trafficking of lysosomes. EMBO Journal, 2013, 33, n/a-n/a.	3.5	122
64	MicroRNA-132 dysregulation in schizophrenia has implications for both neurodevelopment and adult brain function. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, 3125-3130.	3.3	277
65	Membrane Orientation and Subcellular Localization of Transmembrane Protein 106B (TMEM106B), a Major Risk Factor for Frontotemporal Lobar Degeneration. Journal of Biological Chemistry, 2012, 287, 19355-19365.	1.6	126
66	Loss of fused in sarcoma (FUS) promotes pathological Tau splicing. EMBO Reports, 2012, 13, 759-764.	2.0	73
67	miR-132, an experience-dependent microRNA, is essential for visual cortex plasticity. Nature Neuroscience, 2011, 14, 1240-1242.	7.1	167
68	microRNA-34c is a novel target to treat dementias. EMBO Journal, 2011, 30, 4299-4308.	3.5	302
69	ALS-associated fused in sarcoma (FUS) mutations disrupt Transportin-mediated nuclear import. EMBO Journal, 2010, 29, 2841-2857.	3.5	717
70	Distinct Roles of NR2A and NR2B Cytoplasmic Tails in Long-Term Potentiation. Journal of Neuroscience, 2010, 30, 2676-2685.	1.7	184
71	Regulation of Synaptic Structure and Function by FMRP-Associated MicroRNAs miR-125b and miR-132. Neuron, 2010, 65, 373-384.	3.8	657
72	Regulation of Synaptic Structure and Function by FMRP-Associated MicroRNAs miR-125b and miR-132. Neuron, 2010, 68, 161.	3.8	4

#	Article	IF	CITATIONS
73	Identification and Characterization of Neuronal Mitogen-activated Protein Kinase Substrates Using a Specific Phosphomotif Antibody. Molecular and Cellular Proteomics, 2009, 8, 681-695.	2.5	35
74	Role of Septin Cytoskeleton in Spine Morphogenesis and Dendrite Development in Neurons. Current Biology, 2007, 17, 1752-1758.	1.8	255
75	Co-expression of Nicastrin and Presenilin Rescues a Loss of Function Mutant of APH-1. Journal of Biological Chemistry, 2004, 279, 37311-37315.	1.6	25
76	Requirement of PEN-2 for Stabilization of the Presenilin N-/C-terminal Fragment Heterodimer within the Î ³ -Secretase Complex. Journal of Biological Chemistry, 2004, 279, 23255-23261.	1.6	107
77	Identification of Distinct γ-Secretase Complexes with Different APH-1 Variants. Journal of Biological Chemistry, 2004, 279, 41340-41345.	1.6	149
78	Immature nicastrin stabilizes APHâ€1 independent of PENâ€2 and presenilin: identification of nicastrin mutants that selectively interact with APHâ€1. Journal of Neurochemistry, 2004, 89, 1520-1527.	2.1	60
79	The presenilin C-terminus is required for ER-retention, nicastrin-binding and Î ³ -secretase activity. EMBO Journal, 2004, 23, 4738-4748.	3.5	91
80	P4-192 Structure-function analysis of the gamma-secretase complex subunit PEN-2. Neurobiology of Aging, 2004, 25, S530.	1.5	0
81	O4-04-06 Î ² -Amyloid precursor protein intracellular domain (AICD) strongly enhances resting free cytosolic calcium levels. Neurobiology of Aging, 2004, 25, S81.	1.5	0
82	Genetic modifications of the adeno-associated virus type 2 capsid reduce the affinity and the neutralizing effects of human serum antibodies. Gene Therapy, 2003, 10, 2139-2147.	2.3	112
83	Reconstitution of \hat{I}^3 -secretase activity. Nature Cell Biology, 2003, 5, 486-488.	4.6	850
84	Î ³ -Secretase Activity Is Associated with a Conformational Change of Nicastrin. Journal of Biological Chemistry, 2003, 278, 16474-16477.	1.6	89
85	Nicastrin Interacts with Î ³ -Secretase Complex Components via the N-terminal Part of Its Transmembrane Domain. Journal of Biological Chemistry, 2003, 278, 52519-52523.	1.6	54
86	Presenilin-dependent Intramembrane Proteolysis of CD44 Leads to the Liberation of Its Intracellular Domain and the Secretion of an Aβ-like Peptide. Journal of Biological Chemistry, 2002, 277, 44754-44759.	1.6	253
87	Insulin-degrading Enzyme Rapidly Removes the β-Amyloid Precursor Protein Intracellular Domain (AICD). Journal of Biological Chemistry, 2002, 277, 13389-13393.	1.6	185
88	Presenilin-1 mutations of leucine 166 equally affect the generation of the Notch and APP intracellular domains independent of their effect on AÂ42 production. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 8025-8030.	3.3	265
89	Presenilin and nicastrin regulate each other and determine amyloid Â-peptide production via complex formation. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 8666-8671.	3.3	229
90	Presenilin-1 affects trafficking and processing of βAPP and is targeted in a complex with nicastrin to the plasma membrane. Journal of Cell Biology, 2002, 158, 551-561.	2.3	179

#	Article	IF	CITATIONS
91	PEN-2 Is an Integral Component of the \hat{I}^3 -Secretase Complex Required for Coordinated Expression of Presenilin and Nicastrin. Journal of Biological Chemistry, 2002, 277, 39062-39065.	1.6	244
92	Strategies to Generate Molecular and Cellular Tumor Vaccines for Low Grade Non-Hodgkin's Lymphoma. Arzneimittelforschung, 1999, 49, 171-171.	0.5	0