K Ulrich Bayer

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Guidelines for the use and interpretation of assays for monitoring autophagy (3rd edition). Autophagy, 2016, 12, 1-222.	9.1	4,701
2	CaMKII regulation in information processing and storage. Trends in Neurosciences, 2012, 35, 607-618.	8.6	281
3	Transition from Reversible to Persistent Binding of CaMKII to Postsynaptic Sites and NR2B. Journal of Neuroscience, 2006, 26, 1164-1174.	3.6	223
4	Role of the CaMKII/NMDA Receptor Complex in the Maintenance of Synaptic Strength. Journal of Neuroscience, 2011, 31, 9170-9178.	3.6	220
5	CaM Kinase: Still Inspiring at 40. Neuron, 2019, 103, 380-394.	8.1	220
6	Autonomous CaMKII Mediates Both LTP and LTD Using a Mechanism for Differential Substrate Site Selection. Cell Reports, 2014, 6, 431-437.	6.4	173
7	Dual Mechanism of a Natural CaMKII Inhibitor. Molecular Biology of the Cell, 2007, 18, 5024-5033.	2.1	162
8	CaMKII "Autonomy" Is Required for Initiating But Not for Maintaining Neuronal Long-Term Information Storage. Journal of Neuroscience, 2010, 30, 8214-8220.	3.6	141
9	Calcium/Calmodulin-dependent Protein Kinase II Binds to Raf-1 and Modulates Integrin-stimulated ERK Activation. Journal of Biological Chemistry, 2003, 278, 45101-45108.	3.4	135
10	Selective translocation of Ca ²⁺ /calmodulin protein kinase Ilα (CaMKIIα) to inhibitory synapses. Proceedings of the National Academy of Sciences of the United States of America, 2010, 107, 20559-20564.	7.1	125
11	CaMKII in cerebral ischemia. Acta Pharmacologica Sinica, 2011, 32, 861-872.	6.1	114
12	Effective Post-insult Neuroprotection by a Novel Ca2+/ Calmodulin-dependent Protein Kinase II (CaMKII) Inhibitor. Journal of Biological Chemistry, 2010, 285, 20675-20682.	3.4	109
13	CaMKIIÎ ² Association with the Actin Cytoskeleton Is Regulated by Alternative Splicing. Molecular Biology of the Cell, 2006, 17, 4656-4665.	2.1	101
14	The CaMKII holoenzyme structure in activation-competent conformations. Nature Communications, 2017, 8, 15742.	12.8	100
15	Alternative splicing modulates the frequency-dependent response of CaMKII to Ca2+ oscillations. EMBO Journal, 2002, 21, 3590-3597.	7.8	99
16	NMDA Receptor Activation Strengthens Weak Electrical Coupling in Mammalian Brain. Neuron, 2014, 81, 1375-1388.	8.1	90
17	CaMKII Autonomy Is Substrate-dependent and Further Stimulated by Ca2+/Calmodulin. Journal of Biological Chemistry, 2010, 285, 17930-17937.	3.4	85
18	DAPK1 Mediates LTD by Making CaMKII/GluN2B Binding LTP Specific. Cell Reports, 2017, 19, 2231-2243.	6.4	73

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19	Nucleotides and Phosphorylation Bi-directionally Modulate Ca2+/Calmodulin-dependent Protein Kinase II (CaMKII) Binding to the N-Methyl-d-aspartate (NMDA) Receptor Subunit GluN2B. Journal of Biological Chemistry, 2011, 286, 31272-31281.	3.4	63
20	Nitric Oxide Induces Ca2+-independent Activity of the Ca2+/Calmodulin-dependent Protein Kinase II (CaMKII). Journal of Biological Chemistry, 2014, 289, 19458-19465.	3.4	63
21	The CaMKII/GluN2B Protein Interaction Maintains Synaptic Strength. Journal of Biological Chemistry, 2016, 291, 16082-16089.	3.4	63
22	CaMKII Metaplasticity Drives AÎ ² Oligomer-Mediated Synaptotoxicity. Cell Reports, 2018, 23, 3137-3145.	6.4	61
23	Excitotoxic glutamate insults block autophagic flux in hippocampal neurons. Brain Research, 2014, 1542, 12-19.	2.2	60
24	CaMKII versus DAPK1 Binding to GluN2B in Ischemic Neuronal Cell Death after Resuscitation from Cardiac Arrest. Cell Reports, 2020, 30, 1-8.e4.	6.4	46
25	Autonomous CaMKII Activity as a Drug Target for Histological and Functional Neuroprotection after Resuscitation from Cardiac Arrest. Cell Reports, 2017, 18, 1109-1117.	6.4	45
26	Autonomous CaMKII requires further stimulation by Ca ²⁺ /calmodulin for enhancing synaptic strength. FASEB Journal, 2014, 28, 3810-3819.	0.5	44
27	Analysis of the CaMKIIα and β splice-variant distribution among brain regions reveals isoform-specific differences in holoenzyme formation. Scientific Reports, 2018, 8, 5448.	3.3	43
28	CaMKII regulates the depalmitoylation and synaptic removal of the scaffold protein AKAP79/150 to mediate structural long-term depression. Journal of Biological Chemistry, 2018, 293, 1551-1567.	3.4	43
29	CaMKII holoenzyme mechanisms that govern the LTP versus LTD decision. Science Advances, 2021, 7, .	10.3	42
30	Persistent Reversal of Enhanced Amphetamine Intake by Transient CaMKII Inhibition. Journal of Neuroscience, 2013, 33, 1411-1416.	3.6	41
31	Simultaneous Live Imaging of Multiple Endogenous Proteins Reveals a Mechanism for Alzheimer's-Related Plasticity Impairment. Cell Reports, 2019, 27, 658-665.e4.	6.4	39
32	Improving a Natural CaMKII Inhibitor by Random and Rational Design. PLoS ONE, 2011, 6, e25245.	2.5	37
33	A Significant but Rather Mild Contribution of T286 Autophosphorylation to Ca2+/CaM-Stimulated CaMKII Activity. PLoS ONE, 2012, 7, e37176.	2.5	32
34	Multiple CaMKII Binding Modes to the Actin Cytoskeleton Revealed by Single-Molecule Imaging. Biophysical Journal, 2016, 111, 395-408.	0.5	29
35	CaMKII Activity in the Ventral Tegmental Area Gates Cocaine-Induced Synaptic Plasticity in the Nucleus Accumbens. Neuropsychopharmacology, 2014, 39, 989-999.	5.4	28
36	Enzymatic Activity of CaMKII Is Not Required for Its Interaction with the Glutamate Receptor Subunit GluN2B. Molecular Pharmacology, 2013, 84, 834-843.	2.3	23

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37	Ca2+/Calmodulin-Dependent Protein Kinase II (CaMKII). Neuromethods, 2012, , 49-72.	0.3	21
38	Necessary, but not sufficient: Insights into the mechanisms of mGluR mediated long-term depression from a rat model of early life seizures. Neuropharmacology, 2014, 84, 1-12.	4.1	20
39	Differential regulation by ATP versus ADP further links CaMKII aggregation to ischemic conditions. FEBS Letters, 2009, 583, 3577-3581.	2.8	17
40	Conserved and divergent features of neuronal CaMKII holoenzyme structure, function, and high-order assembly. Cell Reports, 2021, 37, 110168.	6.4	17
41	CaMKII isoforms differ in their specific requirements for regulation by nitric oxide. FEBS Letters, 2014, 588, 4672-4676.	2.8	15
42	Live imaging of endogenous Ca ²⁺ /calmodulinâ€dependent protein kinase <scp>II</scp> in neurons reveals that ischemiaâ€related aggregation does not require kinase activity. Journal of Neurochemistry, 2015, 135, 666-673.	3.9	15
43	CaMKII Binding to GluN2B Is Differentially Affected by Macromolecular Crowding Reagents. PLoS ONE, 2014, 9, e96522.	2.5	13
44	Calcium/Calmodulin-Dependent Kinase (CaMKII) Inhibition Protects Against Purkinje Cell Damage Following CA/CPR in Mice. Molecular Neurobiology, 2020, 57, 150-158.	4.0	12
45	The CaMKII K42M and K42R mutations are equivalent in suppressing kinase activity and targeting. PLoS ONE, 2020, 15, e0236478.	2.5	11
46	GluN2B S1303 phosphorylation by CaMKII or DAPK1: No indication for involvement in ischemia or LTP. IScience, 2021, 24, 103214.	4.1	11
47	Ca <scp>MKII</scp> â€mediated displacement of <scp>AIDA</scp> â€1 out of the postsynaptic density core. FEBS Letters, 2016, 590, 2934-2939.	2.8	10
48	Characterization of six CaMKIIÎ \pm variants found in patients with schizophrenia. IScience, 2021, 24, 103184.	4.1	10
49	Developmental restoration of LTP deficits in heterozygous CaMKIIα KO mice. Journal of Neurophysiology, 2016, 116, 2140-2151.	1.8	9
50	NMDA-induced accumulation of Shank at the postsynaptic density is mediated by CaMKII. Biochemical and Biophysical Research Communications, 2014, 450, 808-811.	2.1	7
51	CaMKIIα knockout protects from ischemic neuronal cell death after resuscitation from cardiac arrest. Brain Research, 2021, 1773, 147699.	2.2	5
52	Young DAPK1 knockout mice have altered presynaptic function. Journal of Neurophysiology, 2021, 125, 1973-1981.	1.8	4
53	Aβ-induced synaptic impairments require CaMKII activity that is stimulated by indirect signaling events. IScience, 2022, 25, 104368.	4.1	0