Eiichi Tokuda

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Stagnation of glymphatic interstitial fluid flow and delay in waste clearance in the SOD1-G93A mouse model of ALS. Neuroscience Research, 2021, 171, 74-82.	1.9	15
2	A Metal-Free, Disulfide Oxidized Form of Superoxide Dismutase 1 as a Primary Misfolded Species with Prion-Like Properties in the Extracellular Environments Surrounding Motor Neuron-Like Cells. International Journal of Molecular Sciences, 2021, 22, 4155.	4.1	3
3	Oxidative misfolding of Cu/Zn-superoxide dismutase triggered by non-canonical intramolecular disulfide formation. Free Radical Biology and Medicine, 2020, 147, 187-199.	2.9	13
4	Does wild-type Cu/Zn-superoxide dismutase have pathogenic roles in amyotrophic lateral sclerosis?. Translational Neurodegeneration, 2020, 9, 33.	8.0	26
5	Wild-type Cu/Zn-superoxide dismutase is misfolded in cerebrospinal fluid of sporadic amyotrophic lateral sclerosis. Molecular Neurodegeneration, 2019, 14, 42.	10.8	50
6	A copper-deficient form of mutant Cu/Zn-superoxide dismutase as an early pathological species in amyotrophic lateral sclerosis. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2018, 1864, 2119-2130.	3.8	22
7	Cu/Zn-superoxide dismutase forms fibrillar hydrogels in a pH-dependent manner via a water-rich extended intermediate state. PLoS ONE, 2018, 13, e0205090.	2.5	3
8	Immunochemical characterization on pathological oligomers of mutant Cu/Zn-superoxide dismutase in amyotrophic lateral sclerosis. Molecular Neurodegeneration, 2017, 12, 2.	10.8	16
9	Regulatory Role of RNA Chaperone TDP-43 for RNA Misfolding and Repeat-Associated Translation in SCA31. Neuron, 2017, 94, 108-124.e7.	8.1	114
10	A misfolded dimer of Cu/Znâ€superoxide dismutase leading to pathological oligomerization in amyotrophic lateral sclerosis. Protein Science, 2017, 26, 484-496.	7.6	28
11	An essential role of N-terminal domain of copper chaperone in the enzymatic activation of Cu/Zn-superoxide dismutase. Journal of Inorganic Biochemistry, 2017, 175, 208-216.	3.5	20
12	Abnormal protein oligomers for neurodegeneration. Oncotarget, 2017, 8, 39943-39944.	1.8	9
13	Screening of Drugs Inhibiting In vitro Oligomerization of Cu/Zn-Superoxide Dismutase with a Mutation Causing Amyotrophic Lateral Sclerosis. Frontiers in Molecular Biosciences, 2016, 3, 40.	3.5	19
14	Copper Homeostasis as a Therapeutic Target in Amyotrophic Lateral Sclerosis with SOD1 Mutations. International Journal of Molecular Sciences, 2016, 17, 636.	4.1	49
15	Aggregation of FET Proteins as a Pathological Change in Amyotrophic Lateral Sclerosis. Advances in Experimental Medicine and Biology, 2016, 925, 1-12.	1.6	3
16	Regulation of Intracellular Copper by Induction of Endogenous Metallothioneins Improves the Disease Course in a Mouse Model of Amyotrophic Lateral Sclerosis. Neurotherapeutics, 2015, 12, 461-476.	4.4	19
17	Overexpression of metallothionein-I, a copper-regulating protein, attenuates intracellular copper dyshomeostasis and extends lifespan in a mouse model of amyotrophic lateral sclerosis caused by mutant superoxide dismutase-1. Human Molecular Genetics, 2014, 23, 1271-1285.	2.9	37
18	Dysregulation of intracellular copper trafficking pathway in a mouse model of mutant copper/zinc superoxide dismutaseâ€linked familial amyotrophic lateral sclerosis. Journal of Neurochemistry, 2009, 111, 181-191.	3.9	50

Епсні Токида

#	Article	IF	CITATIONS
19	Ammonium tetrathiomolybdate delays onset, prolongs survival, and slows progression of disease in a mouse model for amyotrophic lateral sclerosis. Experimental Neurology, 2008, 213, 122-128.	4.1	51
20	Dysequilibrium between caspases and their inhibitors in a mouse model for amyotrophic lateral sclerosis. Brain Research, 2007, 1148, 234-242.	2.2	14
21	Metallothionein proteins expression, copper and zinc concentrations, and lipid peroxidation level in a rodent model for amyotrophic lateral sclerosis. Toxicology, 2007, 229, 33-41.	4.2	72