

Adrian Israelson

List of Publications by Year in descending order

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36
papers

3,334
citations

279487

23
h-index

360668

35
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all docs

36
docs citations

36
times ranked

4358
citing authors

#	ARTICLE	IF	CITATIONS
1	Potential roles of gut microbiome and metabolites in modulating ALS in mice. <i>Nature</i> , 2019, 572, 474-480.	13.7	454
2	In self-defence: hexokinase promotes voltage-dependent anion channel closure and prevents mitochondria-mediated apoptotic cell death. <i>Biochemical Journal</i> , 2004, 377, 347-355.	1.7	363
3	The Voltage-Dependent Anion Channel (VDAC): Function in Intracellular Signalling, Cell Life and Cell Death. <i>Current Pharmaceutical Design</i> , 2006, 12, 2249-2270.	0.9	283
4	The voltage-dependent anion channel-1 modulates apoptotic cell death. <i>Cell Death and Differentiation</i> , 2005, 12, 751-760.	5.0	268
5	Misfolded Mutant SOD1 Directly Inhibits VDAC1 Conductance in a Mouse Model of Inherited ALS. <i>Neuron</i> , 2010, 67, 575-587.	3.8	256
6	Hexokinase-I Protection against Apoptotic Cell Death Is Mediated via Interaction with the Voltage-dependent Anion Channel-1. <i>Journal of Biological Chemistry</i> , 2008, 283, 13482-13490.	1.6	226
7	The VDAC1 N-terminus is essential both for apoptosis and the protective effect of anti-apoptotic proteins. <i>Journal of Cell Science</i> , 2009, 122, 1906-1916.	1.2	201
8	Oligomeric states of the voltage-dependent anion channel and cytochrome c release from mitochondria. <i>Biochemical Journal</i> , 2005, 386, 73-83.	1.7	194
9	Methyl jasmonate binds to and detaches mitochondria-bound hexokinase. <i>Oncogene</i> , 2008, 27, 4636-4643.	2.6	175
10	ALS-linked mutant superoxide dismutase 1 (SOD1) alters mitochondrial protein composition and decreases protein import. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2010, 107, 21146-21151.	3.3	155
11	Macrophage Migration Inhibitory Factor as a Chaperone Inhibiting Accumulation of Misfolded SOD1. <i>Neuron</i> , 2015, 86, 218-232.	3.8	98
12	Fluoxetine (Prozac) interaction with the mitochondrial voltage-dependent anion channel and protection against apoptotic cell death. <i>FEBS Letters</i> , 2005, 579, 5105-5110.	1.3	85
13	The Voltage-dependent Anion Channel in Endoplasmic/Sarcoplasmic Reticulum: Characterization, Modulation and Possible Function. <i>Journal of Membrane Biology</i> , 2005, 204, 57-66.	1.0	76
14	Localization of the voltage-dependent anion channel-1 Ca ²⁺ -binding sites. <i>Cell Calcium</i> , 2007, 41, 235-244.	1.1	66
15	Macrophage migration inhibitory factor: A multifaceted cytokine implicated in multiple neurological diseases. <i>Experimental Neurology</i> , 2018, 301, 83-91.	2.0	59
16	MIF inhibits the formation and toxicity of misfolded SOD1 amyloid aggregates: implications for familial ALS. <i>Cell Death and Disease</i> , 2018, 9, 107.	2.7	50
17	Mapping the ruthenium red-binding site of the voltage-dependent anion channel-1. <i>Cell Calcium</i> , 2008, 43, 196-204.	1.1	43
18	Endogenous macrophage migration inhibitory factor reduces the accumulation and toxicity of misfolded SOD1 in a mouse model of ALS. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2016, 113, 10198-10203.	3.3	36

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19	AAV2/9-mediated overexpression of MIF inhibits SOD1 misfolding, delays disease onset, and extends survival in mouse models of ALS. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 14755-14760.	3.3	33
20	Superoxide Dismutase 1 (SOD1)-Derived Peptide Inhibits Amyloid Aggregation of Familial Amyotrophic Lateral Sclerosis SOD1 Mutants. ACS Chemical Neuroscience, 2016, 7, 1595-1606.	1.7	32
21	Cu/Zn-superoxide dismutase and wild-type like fALS SOD1 mutants produce cytotoxic quantities of H2O2 via cysteine-dependent redox short-circuit. Scientific Reports, 2019, 9, 10826.	1.6	27
22	Misfolded SOD1 Accumulation and Mitochondrial Association Contribute to the Selective Vulnerability of Motor Neurons in Familial ALS: Correlation to Human Disease. ACS Chemical Neuroscience, 2017, 8, 2225-2234.	1.7	26
23	A Photoactivable Probe for Calcium Binding Proteins. Chemistry and Biology, 2005, 12, 1169-1178.	6.2	25
24	A VDAC1-Derived N-Terminal Peptide Inhibits Mutant SOD1-VDAC1 Interactions and Toxicity in the SOD1 Model of ALS. Frontiers in Cellular Neuroscience, 2019, 13, 346.	1.8	23
25	All Roads Lead to Rome: Different Molecular Players Converge to Common Toxic Pathways in Neurodegeneration. Cells, 2021, 10, 2438.	1.8	22
26	A Chemical Chaperone-Based Drug Candidate is Effective in a Mouse Model of Amyotrophic Lateral Sclerosis (ALS). ChemMedChem, 2015, 10, 850-861.	1.6	20
27	Empty mesoporous silica particles significantly delay disease progression and extend survival in a mouse model of ALS. Scientific Reports, 2020, 10, 20675.	1.6	7
28	Azido ruthenium: a new photoreactive probe for calcium-binding proteins. Nature Protocols, 2006, 1, 111-117.	5.5	6
29	Assay to Measure Nucleocytoplasmic Transport in Real Time within Motor Neuron-like NSC-34 Cells. Journal of Visualized Experiments, 2017, , .	0.2	6
30	Early upregulation of cytosolic phospholipase A2 in motor neurons is induced by misfolded SOD1 in a mouse model of amyotrophic lateral sclerosis. Journal of Neuroinflammation, 2021, 18, 274.	3.1	5
31	Why lithium studies for ALS treatment should not be halted prematurely. Frontiers in Neuroscience, 2014, 8, 267.	1.4	4
32	Exposure of β -Loop in Zn/Cu Superoxide Dismutase (SOD1) Is Coupled to Metal Loss and Is Transiently Reversible During Misfolding. ACS Chemical Neuroscience, 2021, 12, 49-62.	1.7	3
33	MIF homolog d-dopachrome tautomerase (D-DT/MIF-2) does not inhibit accumulation and toxicity of misfolded SOD1. Scientific Reports, 2022, 12, .	1.6	3
34	Macrophage migration inhibitory factor as a component of selective vulnerability of motor neurons in ALS. Rare Diseases (Austin, Tex), 2015, 3, e1061164.	1.8	2
35	New fluorescent reagents specific for Ca ²⁺ -binding proteins. Biochemical and Biophysical Research Communications, 2012, 426, 158-164.	1.0	1
36	Methyl jasmonate binds to and detaches mitochondria-bound hexokinase. , 0, .		1