Stanley H Appel

List of Publications by Year in descending order

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STANLEY Η ΔΟDE

#	Article	IF	CITATIONS
1	Tregs Attenuate Peripheral Oxidative Stress and Acute Phase Proteins in <scp>ALS</scp> . Annals of Neurology, 2022, 92, 195-200.	5.3	14
2	Serum programmed cell death proteins in amyotrophic lateral sclerosis. Brain, Behavior, & Immunity - Health, 2021, 12, 100209.	2.5	6
3	Neuroinflammation is highest in areas of disease progression in semantic dementia. Brain, 2021, 144, 1565-1575.	7.6	23
4	Ex vivo expansion of dysfunctional regulatory T lymphocytes restores suppressive function in Parkinson's disease. Npj Parkinson's Disease, 2021, 7, 41.	5.3	32
5	Fast Progression in Amyotrophic Lateral Sclerosis Is Associated With Greater TDP-43 Burden in Spinal Cord. Journal of Neuropathology and Experimental Neurology, 2021, 80, 754-763.	1.7	7
6	Amyotrophic lateral sclerosis is a systemic disease: peripheral contributions to inflammation-mediated neurodegeneration. Current Opinion in Neurology, 2021, 34, 765-772.	3.6	35
7	Radicava/Edaravone Findings in Biomarkers From Amyotrophic Lateral Sclerosis (REFINE-ALS): Protocol and Study Design. Neurology: Clinical Practice, 2021, 11, e472-e479.	1.6	0
8	Multimodal ¹⁸ F-AV-1451 and MRI Findings in Nonfluent Variant of Primary Progressive Aphasia: Possible Insights on Nodal Propagation of Tau Protein Across the Syntactic Network. Journal of Nuclear Medicine, 2020, 61, 263-269.	5.0	7
9	Restoring regulatory T-cell dysfunction in Alzheimer's disease through ex vivo expansion. Brain Communications, 2020, 2, fcaa112.	3.3	48
10	Elevated acute phase proteins reflect peripheral inflammation and disease severity in patients with amyotrophic lateral sclerosis. Scientific Reports, 2020, 10, 15295.	3.3	34
11	Immunosuppressive Functions of M2 Macrophages Derived from iPSCs of Patients with ALS and Healthy Controls. IScience, 2020, 23, 101192.	4.1	27
12	Increased activation ability of monocytes from ALS patients. Experimental Neurology, 2020, 328, 113259.	4.1	30
13	Dipeptide repeat (DPR) pathology in the skeletal muscle of ALS patients with C9ORF72 repeat expansion. Acta Neuropathologica, 2019, 138, 667-670.	7.7	32
14	Immune dysregulation in amyotrophic lateral sclerosis: mechanisms and emerging therapies. Lancet Neurology, The, 2019, 18, 211-220.	10.2	206
15	The Role of Regulatory T Lymphocytes in Amyotrophic Lateral Sclerosis. JAMA Neurology, 2018, 75, 656.	9.0	24
16	Head-to-Head Comparison of ¹¹ C-PBR28 and ¹⁸ F-GE180 for Quantification of the Translocator Protein in the Human Brain. Journal of Nuclear Medicine, 2018, 59, 1260-1266.	5.0	48
17	An open label study of a novel immunosuppression intervention for the treatment of amyotrophic lateral sclerosis. Amyotrophic Lateral Sclerosis and Frontotemporal Degeneration, 2018, 19, 242-249.	1.7	35
18	Phosphorylated TDP-43 (pTDP-43) aggregates in the axial skeletal muscle of patients with sporadic and familial amyotrophic lateral sclerosis. Acta Neuropathologica Communications, 2018, 6, 28.	5.2	59

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19	Functional alterations of myeloid cells during the course of Alzheimer's disease. Molecular Neurodegeneration, 2018, 13, 61.	10.8	44
20	Expanded autologous regulatory T-lymphocyte infusions in ALS. Neurology: Neuroimmunology and NeuroInflammation, 2018, 5, e465.	6.0	116
21	Defining SOD1 ALS natural history to guide therapeutic clinical trial design. Journal of Neurology, Neurosurgery and Psychiatry, 2017, 88, 99-105.	1.9	68
22	Characterization of Gene Expression Phenotype in Amyotrophic Lateral Sclerosis Monocytes. JAMA Neurology, 2017, 74, 677.	9.0	130
23	Pyrimethamine significantly lowers cerebrospinal fluid Cu/Zn superoxide dismutase in amyotrophic lateral sclerosis patients with <i>SOD1</i> mutations. Annals of Neurology, 2017, 81, 837-848.	5.3	32
24	Serum C-Reactive Protein as a Prognostic Biomarker in Amyotrophic Lateral Sclerosis. JAMA Neurology, 2017, 74, 660.	9.0	96
25	TDP-43 Depletion in Microglia Promotes Amyloid Clearance but Also Induces Synapse Loss. Neuron, 2017, 95, 297-308.e6.	8.1	171
26	Clinical Significance of TDP-43 Neuropathology in Amyotrophic Lateral Sclerosis. Journal of Neuropathology and Experimental Neurology, 2017, 76, 402-413.	1.7	53
27	ALS patients' regulatory T lymphocytes are dysfunctional, and correlate with disease progression rate and severity. JCI Insight, 2017, 2, e89530.	5.0	141
28	The immune system continues to knock at the ALS door. Neuromuscular Disorders, 2016, 26, 335-336.	0.6	3
29	A robust, good manufacturing practice–compliant, clinical-scale procedure to generate regulatory T cells from patients with amyotrophic lateral sclerosis for adoptive cell therapy. Cytotherapy, 2016, 18, 1312-1324.	0.7	39
30	Stem cells in amyotrophic lateral sclerosis. Neurology, 2016, 87, 348-349.	1.1	6
31	Exome sequencing in amyotrophic lateral sclerosis identifies risk genes and pathways. Science, 2015, 347, 1436-1441.	12.6	823
32	Protective and Toxic Neuroinflammation in Amyotrophic Lateral Sclerosis. Neurotherapeutics, 2015, 12, 364-375.	4.4	236
33	TDP-43 activates microglia through NF-κB and NLRP3 inflammasome. Experimental Neurology, 2015, 273, 24-35.	4.1	174
34	<i>TREM2</i> Variant p.R47H as a Risk Factor for Sporadic Amyotrophic Lateral Sclerosis. JAMA Neurology, 2014, 71, 449.	9.0	221
35	Immune-mediated Mechanisms in the Pathoprogression of Amyotrophic Lateral Sclerosis. Journal of NeuroImmune Pharmacology, 2013, 8, 888-899.	4.1	253
36	Blood–spinal cord barrier breakdown and pericyte reductions in amyotrophic lateral sclerosis. Acta Neuropathologica, 2013, 125, 111-120.	7.7	263

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37	Regulatory T″ymphocytes mediate amyotrophic lateral sclerosis progression and survival. EMBO Molecular Medicine, 2013, 5, 64-79.	6.9	289
38	Deciphering amyotrophic lateral sclerosis: What phenotype, neuropathology and genetics are telling us about pathogenesis. Amyotrophic Lateral Sclerosis and Frontotemporal Degeneration, 2013, 14, 5-18.	1.7	142
39	Peripheral nerve inflammation in ALS mice: cause or consequence. Neurology, 2012, 78, 833-835.	1.1	39
40	Transformation from a neuroprotective to a neurotoxic microglial phenotype in a mouse model of ALS. Experimental Neurology, 2012, 237, 147-152.	4.1	346
41	Regulatory T lymphocytes from ALS mice suppress microglia and effector T lymphocytes through different cytokine-mediated mechanisms. Neurobiology of Disease, 2012, 48, 418-428.	4.4	109
42	Inflammation in Parkinson's disease: Cause or consequence?. Movement Disorders, 2012, 27, 1075-1077.	3.9	38
43	Neuroinflammation modulates distinct regional and temporal clinical responses in ALS mice. Brain, Behavior, and Immunity, 2011, 25, 1025-1035.	4.1	170
44	Endogenous regulatory T lymphocytes ameliorate amyotrophic lateral sclerosis in mice and correlate with disease progression in patients with amyotrophic lateral sclerosis. Brain, 2011, 134, 1293-1314.	7.6	323
45	Extracellular mutant SOD1 induces microglialâ€mediated motoneuron injury. Glia, 2010, 58, 231-243.	4.9	232
46	T cell-microglial dialogue in Parkinson's disease and amyotrophic lateral sclerosis: are we listening?. Trends in Immunology, 2010, 31, 7-17.	6.8	218
47	ALSFRS and appel ALS scores: Discordance with disease progression. Muscle and Nerve, 2008, 37, 668-672.	2.2	32
48	CD4+ T cells support glial neuroprotection, slow disease progression, and modify glial morphology in an animal model of inherited ALS. Proceedings of the National Academy of Sciences of the United States of America, 2008, 105, 15558-15563.	7.1	401
49	Mutant SOD1G93Amicroglia are more neurotoxic relative to wild-type microglia. Journal of Neurochemistry, 2007, 102, 2008-2019.	3.9	139
50	Protective effects of an antiâ€inflammatory cytokine, interleukinâ€4, on motoneuron toxicity induced by activated microglia. Journal of Neurochemistry, 2006, 99, 1176-1187.	3.9	138
51	Wild-type microglia extend survival in PU.1 knockout mice with familial amyotrophic lateral sclerosis. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103, 16021-16026.	7.1	647
52	Calcium binding proteins in selective vulnerability of motor neurons. , 2005, , 65-79.		2
53	AALSS. Amyotrophic Lateral Sclerosis and Other Motor Neuron Disorders: Official Publication of the World Federation of Neurology, Research Group on Motor Neuron Diseases, 2004, 5, 94-98.	1.2	1
54	Activated Microglia Initiate Motor Neuron Injury by a Nitric Oxide and Glutamate-Mediated Mechanism. Journal of Neuropathology and Experimental Neurology, 2004, 63, 964-977.	1.7	147

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55	β-Amyloid1-40 Increases Expression of β-Amyloid Precursor Protein in Neuronal Hybrid Cells. Journal of Neurochemistry, 2002, 65, 2373-2376.	3.9	18
56	Activated microglia: The silent executioner in neurodegenerative disease?. Current Neurology and Neuroscience Reports, 2001, 1, 303-305.	4.2	17
57	Parvalbumin overexpression alters immuneâ€mediated increases in intracellular calcium, and delays disease onset in a transgenic model of familial amyotrophic lateral sclerosis. Journal of Neurochemistry, 2001, 79, 499-509.	3.9	129
58	Protective role of heme oxygenase-1 in oxidative stress-induced neuronal injury. , 1999, 56, 652-658.		141
59	Protective role of heme oxygenaseâ€1 in oxidative stressâ€induced neuronal injury. Journal of Neuroscience Research, 1999, 56, 652-658.	2.9	9
60	Presence of 4â€hydroxynonenal in cerebrospinal fluid of patients with sporadic amyotrophic lateral sclerosis. Annals of Neurology, 1998, 44, 696-699.	5.3	219
61	Protein modification by the lipid peroxidation product 4â€hydroxynonenal in the spinal cords of amyotrophic lateral sclerosis patients. Annals of Neurology, 1998, 44, 819-824.	5.3	355
62	1Alpha, 25 dihydroxyvitamin D3-dependent up-regulation of calcium-binding proteins in motoneuron cells. , 1998, 51, 58.		64
63	Antibodies from patients with Parkinson's disease react with protein modified by dopamine oxidation. Journal of Neuroscience Research, 1998, 53, 551-558.	2.9	59
64	Role of Potassium Channels in Amyloidâ€Induced Cell Death. Journal of Neurochemistry, 1998, 70, 1925-1934.	3.9	138
65	Antibodies from patients with Parkinson's disease react with protein modified by dopamine oxidation. Journal of Neuroscience Research, 1998, 53, 551-558.	2.9	3
66	The differential in vitro stimulation of 3′,5′ yclic nucleotide phosphodiesterase by calcium binding proteins. IUBMB Life, 1997, 43, 1195-1205.	3.4	0
67	βâ€Amyloidâ€Induced Neurotoxicity of a Hybrid Septal Cell Line Associated with Increased Tau Phosphorylation and Expression of βâ€Amyloid Precursor Protein. Journal of Neurochemistry, 1997, 69, 978-985.	3.9	47
68	Ultrastructural evidence for altered calcium in motor nerve terminals in amyotrophc lateral sclerosis. Annals of Neurology, 1996, 39, 203-216.	5.3	274
69	Amyotrophic lateral sclerosis immunoglobulins increase Ca2+ currents in a motoneuron cell line. Annals of Neurology, 1995, 37, 102-109.	5.3	62
70	Antibodies to calcium channels from ALS patients passively transferred to mice selectively increase intracellular calcium and induce ultrastructural changes in motoneurons. Synapse, 1995, 20, 185-199.	1.2	81
71	MOLECULAR APPROACHES TO AMYOTROPHIC LATERAL SCLEROSIS. Annual Review of Medicine, 1995, 46, 133-145.	12.2	34
72	Natural history of amyotrophic lateral sclerosis in a database population Validation of a scoring system and a model for survival prediction. Brain, 1995, 118, 707-719.	7.6	627

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73	Decremental motor responses to repetitive nerve stimulation in ALS. Muscle and Nerve, 1994, 17, 747-754.	2.2	85
74	Amyotrophic lateral sclerosis patient antibodies label Ca2+ channel ?1 subunit. Annals of Neurology, 1994, 35, 164-171.	5.3	91
75	Trial of immunosuppression in amyotrophic lateral sclerosis using total lymphoid irradiation. Annals of Neurology, 1994, 36, 253-254.	5.3	10
76	The role of calciumâ€binding proteins in selective motoneuron vulnerability in amyotrophic lateral sclerosis. Annals of Neurology, 1994, 36, 846-858.	5.3	328
77	Apoptotic Cell Death of a Hybrid Motoneuron Cell Line Induced by Immunoglobulins from Patients with Amyotrophic Lateral Sclerosis. Journal of Neurochemistry, 1994, 63, 2365-2368.	3.9	55
78	The Lambert-Eaton Syndrome. Hospital Practice (1995), 1992, 27, 101-116.	1.0	4
79	Phosphoethanolamine Enhances High-Affinity Choline Uptake and Acetylcholine Synthesis in Dissociated Cell Cultures of the Rat Septal Nucleus. Journal of Neurochemistry, 1992, 59, 236-244.	3.9	11
80	Nigral damage and dopaminergic hypofunction in mesencephalon-immunized guinea pigs. Annals of Neurology, 1992, 32, 494-501.	5.3	31
81	Increased MEPP frequency as an early sign of experimental immune-mediated motoneuron disease. Annals of Neurology, 1990, 28, 329-334.	5.3	17
82	Experimental autoimmune motoneuron disease. Annals of Neurology, 1989, 26, 368-376.	5.3	77
83	Purification and Characterization of a Central Cholinergic Enhancing Factor from Rat Brain: Its Identity as Phosphoethanolamine. Journal of Neurochemistry, 1989, 53, 448-458.	3.9	14
84	Trophic Effects of Skeletal Muscle Extracts on Spinal Cord Cultures. International Journal of Neuroscience, 1989, 47, 203-208.	1.6	4
85	Choice Reaction Time Modifiability in Dementia and depression. International Journal of Neuroscience, 1985, 26, 1-7.	1.6	16
86	Endogenous Inhibitor of Ligand Binding to the Muscarinic Acetylcholine Receptor. Journal of Neurochemistry, 1985, 44, 622-628.	3.9	24
87	Interaction of myasthenic immunoglobulins and cholinergic agonists on acetylcholine receptors of rat myotubes. Annals of Neurology, 1982, 11, 22-27.	5.3	10
88	A unifying hypothesis for the cause of amyotrophic lateral sclerosis, parkinsonism, and alzheimer disease. Annals of Neurology, 1981, 10, 499-505.	5.3	755
89	Red blood cell alterations in muscular dystrophy: The role of lipids. Muscle and Nerve, 1980, 3, 70-81.	2.2	50
90	Neonatal myasthenia gravis in the infant of a myasthenic mother in remission. Annals of Neurology, 1979, 6, 72-75.	5.3	37

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91	Acetylcholine receptor in myasthenia gravis: Increased affinity for ?-bungarotoxin. Annals of Neurology, 1978, 4, 250-252.	5.3	7
92	Extracellular Vesicles Derived From Ex Vivo Expanded Regulatory T Cells Modulate In Vitro and In Vivo Inflammation. Frontiers in Immunology, 0, 13, .	4.8	14