

# Nasser H Zawia

## List of Publications by Year in descending order

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Version: 2024-02-01

51  
papers

3,229  
citations

201575

27  
h-index

214721

47  
g-index

53  
all docs

53  
docs citations

53  
times ranked

2896  
citing authors

#	ARTICLE	IF	CITATIONS
1	Fenamates as Potential Therapeutics for Neurodegenerative Disorders. <i>Cells</i> , 2021, 10, 702.	1.8	14
2	Developmental Perfluorooctanesulfonic acid (PFOS) exposure as a potential risk factor for late-onset Alzheimer's disease in CD-1 mice and SH-SY5Y cells. <i>NeuroToxicology</i> , 2021, 86, 26-36.	1.4	14
3	Pomegranate ellagitannin-gut microbial-derived metabolites, urolithins, inhibit neuroinflammation <i>in vitro</i> . <i>Nutritional Neuroscience</i> , 2019, 22, 185-195.	1.5	65
4	Altered microRNA, mRNA, and Protein Expression of Neurodegeneration-Related Biomarkers and Their Transcriptional and Epigenetic Modifiers in a Human Tau Transgenic Mouse Model in Response to Developmental Lead Exposure. <i>Journal of Alzheimer's Disease</i> , 2018, 63, 273-282.	1.2	12
5	Histone acetylation maps in aged mice developmentally exposed to lead: epigenetic drift and Alzheimer-related genes. <i>Epigenomics</i> , 2018, 10, 573-583.	1.0	15
6	Influence of Early Life Lead (Pb) Exposure on $\beta$ -Synuclein, GSK-3 $\beta$ and Caspase-3 Mediated Tauopathy: Implications on Alzheimer's Disease. <i>Current Alzheimer Research</i> , 2018, 15, 1114-1122.	0.7	19
7	Latent consequences of early-life lead (Pb) exposure and the future: Addressing the Pb crisis. <i>NeuroToxicology</i> , 2018, 68, 126-132.	1.4	24
8	Tolfenamic Acid: A Modifier of the Tau Protein and its Role in Cognition and Tauopathy. <i>Current Alzheimer Research</i> , 2018, 15, 655-663.	0.7	14
9	Lead exposure and tau hyperphosphorylation: An <i>in vitro</i> study. <i>NeuroToxicology</i> , 2017, 62, 218-223.	1.4	16
10	Unique aspects of the epigenetic code in the brain. <i>Epigenomics</i> , 2017, 9, 1157-1159.	1.0	2
11	Early-Life Exposure to Lead (Pb) Alters the Expression of microRNA that Target Proteins Associated with Alzheimer's Disease. <i>Journal of Alzheimer's Disease</i> , 2016, 51, 1257-1264.	1.2	60
12	Developmental lead exposure and lifespan alterations in epigenetic regulators and their correspondence to biomarkers of Alzheimer's disease. <i>Alzheimer's and Dementia: Diagnosis, Assessment and Disease Monitoring</i> , 2016, 2, 123-131.	1.2	65
13	Tolfenamic acid reduces tau and CDK5 levels: implications for dementia and tauopathies. <i>Journal of Neurochemistry</i> , 2015, 133, 266-272.	2.1	23
14	Infantile exposure to lead and late-life cognitive decline: Relevance to AD. <i>Alzheimer's and Dementia</i> , 2014, 10, 187-195.	0.4	79
15	Tolfenamic acid downregulates BACE1 and protects against lead-induced upregulation of Alzheimer's disease related biomarkers. <i>Neuropharmacology</i> , 2014, 79, 596-602.	2.0	34
16	Infantile postnatal exposure to lead (Pb) enhances tau expression in the cerebral cortex of aged mice: Relevance to AD. <i>NeuroToxicology</i> , 2014, 44, 114-120.	1.4	65
17	Reduction of Amyloid- $\beta$ Deposition and Attenuation of Memory Deficits by Tolfenamic Acid. <i>Journal of Alzheimer's Disease</i> , 2014, 43, 425-433.	1.2	16
18	Cellular and Organismal Toxicity of the Anti-Cancer Small Molecule, Tolfenamic Acid: a Pre-Clinical Evaluation. <i>Cellular Physiology and Biochemistry</i> , 2013, 32, 675-686.	1.1	21

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19	Short-term treatment with tolfenamic acid improves cognitive functions in Alzheimer's disease mice. <i>Neurobiology of Aging</i> , 2013, 34, 2421-2430.	1.5	40
20	Enhanced taupathy and AD-like pathology in aged primate brains decades after infantile exposure to lead (Pb). <i>NeuroToxicology</i> , 2013, 39, 95-101.	1.4	89
21	Epigenetics: A novel therapeutic approach for the treatment of Alzheimer's disease. , 2013, 139, 41-50.		95
22	Alzheimer's Disease Biomarkers and Epigenetic Intermediates Following Exposure to Pb In Vitro. <i>Current Alzheimer Research</i> , 2012, 9, 555-562.	0.7	54
23	Integration of genome-wide expression and methylation data: Relevance to aging and Alzheimer's disease. <i>NeuroToxicology</i> , 2012, 33, 1450-1453.	1.4	26
24	Do Epigenetic Pathways Initiate Late Onset Alzheimer Disease (LOAD): Towards a New Paradigm. <i>Current Alzheimer Research</i> , 2012, 9, 574-588.	0.7	46
25	Genome-wide expression and methylation profiling in the aged rodent brain due to early-life Pb exposure and its relevance to aging. <i>Mechanisms of Ageing and Development</i> , 2012, 133, 435-443.	2.2	80
26	In vitro Pb exposure disturbs the balance between A $\beta$ production and elimination: The role of A $\beta$ PP and neprilysin. <i>NeuroToxicology</i> , 2011, 32, 300-306.	1.4	54
27	The Ability of Tolfenamic Acid to Penetrate the Brain: A Model for Testing the Brain Disposition of Candidate Alzheimers Drugs Using Multiple Platforms. <i>Current Alzheimer Research</i> , 2011, 8, 860-867.	0.7	24
28	Infant Exposure to Lead (Pb) and Epigenetic Modifications in the Aging Primate Brain: Implications for Alzheimer's Disease. <i>Journal of Alzheimer's Disease</i> , 2011, 27, 819-833.	1.2	140
29	Epigenetics and Late-Onset Alzheimer's Disease. , 2011, , 175-186.		1
30	An Epigenetic Model for Susceptibility to Oxidative DNA Damage in the Aging Brain and Alzheimer's Disease. , 2010, , 439-453.		1
31	Lifespan Profiles of Alzheimer's Disease-Associated Genes and Products in Monkeys and Mice. <i>Journal of Alzheimer's Disease</i> , 2009, 18, 211-230.	1.2	28
32	Epigenetics, oxidative stress, and Alzheimer disease. <i>Free Radical Biology and Medicine</i> , 2009, 46, 1241-1249.	1.3	311
33	How Biochemical Pathways for Disease May be Triggered by Early-Life Events. , 2009, , 205-214.		1
34	The Environment, Epigenetics and Amyloidogenesis. <i>Journal of Molecular Neuroscience</i> , 2008, 34, 1-7.	1.1	90
35	Early-life events may trigger biochemical pathways for Alzheimer's disease: the "LEAR" model. <i>Bogerontology</i> , 2008, 9, 375-379.	2.0	58
36	An initial animal proof-of-concept study for central administration of clozapine to schizophrenia patients. <i>Schizophrenia Research</i> , 2008, 100, 86-96.	1.1	11

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37	Alzheimer's Disease (AD)-Like Pathology in Aged Monkeys after Infantile Exposure to Environmental Metal Lead (Pb): Evidence for a Developmental Origin and Environmental Link for AD. <i>Journal of Neuroscience</i> , 2008, 28, 3-9.	1.7	438
38	Co-localization and Distribution of Cerebral APP and SP1 and its Relationship to Amyloidogenesis. <i>Journal of Alzheimer's Disease</i> , 2008, 13, 71-80.	1.2	25
39	How and When Environmental Agents and Dietary Factors Affect the Course of Alzheimers Disease: The &#x201C;LEARN&#x201D; Model (Latent Early-Life Associated Regulation) May Explain the Triggering of AD. <i>Current Alzheimer Research</i> , 2007, 4, 219-228.	0.7	140
40	Environmental and dietary risk factors in Alzheimerâ€™s disease. <i>Expert Review of Neurotherapeutics</i> , 2007, 7, 887-900.	1.4	93
41	Ontogenetic alterations in prototypical transcription factors in the rat cerebellum and hippocampus following perinatal exposure to a commercial PCB mixture. <i>NeuroToxicology</i> , 2006, 27, 118-124.	1.4	28
42	Characterization of nigerlysin Â©, hemolysin produced by <i>Aspergillus niger</i> , and effect on mouse neuronal cells in vitro. <i>Toxicology</i> , 2006, 219, 150-155.	2.0	14
43	Lead Exposure: Expression and Activity Levels of Oct-2 in the Developing Rat Brain. <i>Toxicological Sciences</i> , 2006, 95, 436-442.	1.4	10
44	Exposure to lead (Pb) and the developmental origin of oxidative DNA damage in the aging brain. <i>FASEB Journal</i> , 2006, 20, 788-790.	0.2	156
45	Lead (Pb) exposure and its effect on APP proteolysis and AÎ² aggregation. <i>FASEB Journal</i> , 2005, 19, 2083-2084.	0.2	87
46	The Fetal Basis of Amyloidogenesis: Exposure to Lead and Latent Overexpression of Amyloid Precursor Protein and A-Amyloid in the Aging Brain. <i>Journal of Neuroscience</i> , 2005, 25, 823-829.	1.7	338
47	Environmental Risk Factors and the Developmental Basis for Alzheimer's Disease. <i>Reviews in the Neurosciences</i> , 2005, 16, 325-37.	1.4	66
48	Transcriptional involvement in neurotoxicity. <i>Toxicology and Applied Pharmacology</i> , 2003, 190, 177-188.	1.3	22
49	Utilization of a Synthetic Peptide as a Tool to Study the Interaction of Heavy Metals with the Zinc Finger Domain of Proteins Critical for Gene Expression in the Developing Brain. <i>Toxicology and Applied Pharmacology</i> , 2000, 166, 1-12.	1.3	43
50	Intranigral iron infusion in the rat. <i>Biological Trace Element Research</i> , 1997, 58, 177-195.	1.9	18
51	c-fos and ornithine decarboxylase gene expression in brain as early markers of neurotoxicity. <i>Brain Research</i> , 1991, 544, 291-296.	1.1	44