

Henrik Viberg

List of Publications by Year in descending order

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40
papers

2,749
citations

186209

28
h-index

315616

38
g-index

40
all docs

40
docs citations

40
times ranked

2172
citing authors

#	ARTICLE	IF	CITATIONS
1	Evaluation of the dentate gyrus in adult mice exposed to acetaminophen (paracetamol) on postnatal day 10. <i>International Journal of Developmental Neuroscience</i> , 2021, 81, 91-97.	0.7	4
2	A Single δ^9 -Tetrahydrocannabinol (THC) Dose During Brain Development Affects Markers of Neurotrophin, Oxidative Stress, and Apoptosis. <i>Frontiers in Pharmacology</i> , 2019, 10, 1156.	1.6	8
3	A Cannabinoid Receptor Type 1 (CB1R) Agonist Enhances the Developmental Neurotoxicity of Acetaminophen (Paracetamol). <i>Toxicological Sciences</i> , 2018, 166, 203-212.	1.4	14
4	Adult neurobehavioral alterations in male and female mice following developmental exposure to paracetamol (acetaminophen): characterization of a critical period. <i>Journal of Applied Toxicology</i> , 2017, 37, 1174-1181.	1.4	42
5	Perfluorooctane Sulfonate and Perfluorooctanoic Acid. , 2017, , 811-827.		3
6	Short-term exposure and long-term consequences of neonatal exposure to δ^9 -tetrahydrocannabinol (THC) and ibuprofen in mice. <i>Behavioural Brain Research</i> , 2016, 307, 137-144.	1.2	24
7	Postnatal exposure to PFOS, but not PBDE 99, disturb dopaminergic gene transcription in the mouse CNS. <i>Environmental Toxicology and Pharmacology</i> , 2016, 41, 121-126.	2.0	27
8	Effects of neonatal exposure to the flame retardant tetrabromobisphenol-A, aluminum diethylphosphinate or zinc stannate on long-term potentiation and synaptic protein levels in mice. <i>Archives of Toxicology</i> , 2015, 89, 2345-2354.	1.9	10
9	Developmental neurotoxic effects of two pesticides: Behavior and neuroprotein studies on endosulfan and cypermethrin. <i>Toxicology</i> , 2015, 335, 1-10.	2.0	58
10	Developmental neurotoxic effects of two pesticides: Behavior and biomolecular studies on chlorpyrifos and carbaryl. <i>Toxicology and Applied Pharmacology</i> , 2015, 288, 429-438.	1.3	52
11	More signs of neurotoxicity of surfactants and flame retardants – Neonatal PFOS and PBDE 99 cause transcriptional alterations in cholinergic genes in the mouse CNS. <i>Environmental Toxicology and Pharmacology</i> , 2015, 40, 409-416.	2.0	32
12	Neurotoxicity. <i>Molecular and Integrative Toxicology</i> , 2015, , 219-238.	0.5	2
13	Paracetamol (Acetaminophen) Administration During Neonatal Brain Development Affects Cognitive Function and Alters Its Analgesic and Anxiolytic Response in Adult Male Mice. <i>Toxicological Sciences</i> , 2014, 138, 139-147.	1.4	114
14	Neonatal exposure to a moderate dose of ionizing radiation causes behavioural defects and altered levels of tau protein in mice. <i>NeuroToxicology</i> , 2014, 45, 48-55.	1.4	27
15	Developmental exposure to the polybrominated diphenyl ether PBDE 209: Neurobehavioural and neuroprotein analysis in adult male and female mice. <i>Environmental Toxicology and Pharmacology</i> , 2014, 38, 570-585.	2.0	34
16	Adult dose-dependent behavioral and cognitive disturbances after a single neonatal PFHxS dose. <i>Toxicology</i> , 2013, 304, 185-191.	2.0	91
17	A single neonatal exposure to perfluorohexane sulfonate (PFHxS) affects the levels of important neuroproteins in the developing mouse brain. <i>NeuroToxicology</i> , 2013, 37, 190-196.	1.4	62
18	A single exposure to bisphenol A alters the levels of important neuroproteins in adult male and female mice. <i>NeuroToxicology</i> , 2012, 33, 1390-1395.	1.4	36

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19	Neonatal exposure to propofol affects BDNF but not CaMKII, GAP-43, synaptophysin and tau in the neonatal brain and causes an altered behavioural response to diazepam in the adult mouse brain. <i>Behavioural Brain Research</i> , 2011, 223, 75-80.	1.2	32
20	Differences in neonatal neurotoxicity of brominated flame retardants, PBDE 99 and TBBPA, in mice. <i>Toxicology</i> , 2011, 289, 59-65.	2.0	70
21	Dose-dependent behavioral disturbances after a single neonatal Bisphenol A dose. <i>Toxicology</i> , 2011, 290, 187-194.	2.0	44
22	Neonatal exposure to sucralose does not alter biochemical markers of neuronal development or adult behavior. <i>Nutrition</i> , 2011, 27, 81-85.	1.1	17
23	Perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA). , 2011, , 623-635.		7
24	Exposure to Polybrominated Diphenyl Ethers 203 and 206 during the Neonatal Brain Growth Spurt Affects Proteins Important for Normal Neurodevelopment in Mice. <i>Toxicological Sciences</i> , 2009, 109, 306-311.	1.4	40
25	Neonatal Exposure to PFOS and PFOA in Mice Results in Changes in Proteins which are Important for Neuronal Growth and Synaptogenesis in the Developing Brain. <i>Toxicological Sciences</i> , 2009, 108, 412-418.	1.4	219
26	Neonatal ontogeny and neurotoxic effect of decabrominated diphenyl ether (PBDE 209) on levels of synaptophysin and tau. <i>International Journal of Developmental Neuroscience</i> , 2009, 27, 423-429.	0.7	40
27	Neonatal ketamine exposure results in changes in biochemical substrates of neuronal growth and synaptogenesis, and alters adult behavior irreversibly. <i>Toxicology</i> , 2008, 249, 153-159.	2.0	83
28	Response to the comment on Viberg et al. (2008) "Neonatal ketamine exposure results in changes in biochemical substrates of neuronal growth and synaptogenesis, and alters adult behavior irreversibly" by Ching-Hung Hsu. <i>Toxicology</i> , 2008, 253, 154.	2.0	1
29	Neonatal exposure to decabrominated diphenyl ether (PBDE 209) results in changes in BDNF, CaMKII and GAP-43, biochemical substrates of neuronal survival, growth, and synaptogenesis. <i>NeuroToxicology</i> , 2008, 29, 152-159.	1.4	120
30	Changes in spontaneous behaviour and altered response to nicotine in the adult rat, after neonatal exposure to the brominated flame retardant, decabrominated diphenyl ether (PBDE 209). <i>NeuroToxicology</i> , 2007, 28, 136-142.	1.4	134
31	Neonatal Exposure to Higher Brominated Diphenyl Ethers, Hepta-, Octa-, or Nonabromodiphenyl Ether, Impairs Spontaneous Behavior and Learning and Memory Functions of Adult Mice. <i>Toxicological Sciences</i> , 2006, 92, 211-218.	1.4	157
32	Proteomic Evaluation of Neonatal Exposure to 2,2,4,4,5-Pentabromodiphenyl Ether. <i>Environmental Health Perspectives</i> , 2006, 114, 254-259.	2.8	60
33	Deranged spontaneous behaviour and decrease in cholinergic muscarinic receptors in hippocampus in the adult rat, after neonatal exposure to the brominated flame-retardant, 2,2,4,4,5-pentabromodiphenyl ether (PBDE 99). <i>Environmental Toxicology and Pharmacology</i> , 2005, 20, 283-288.	2.0	52
34	Dose-Response Modeling and Benchmark Calculations from Spontaneous Behavior Data on Mice Neonatally Exposed to 2,2',4,4',5-Pentabromodiphenyl Ether. <i>Toxicological Sciences</i> , 2004, 81, 491-501.	1.4	35
35	Investigations of Strain and/or Gender Differences in Developmental Neurotoxic Effects of Polybrominated Diphenyl Ethers in Mice. <i>Toxicological Sciences</i> , 2004, 81, 344-353.	1.4	113
36	Neonatal exposure to the brominated flame-retardant, 2,2,4,4,5-pentabromodiphenyl ether, decreases cholinergic nicotinic receptors in hippocampus and affects spontaneous behaviour in the adult mouse. <i>Environmental Toxicology and Pharmacology</i> , 2004, 17, 61-65.	2.0	84

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37	Neonatal exposure to polybrominated diphenyl ether (PBDE 153) disrupts spontaneous behaviour, impairs learning and memory, and decreases hippocampal cholinergic receptors in adult mice. <i>Toxicology and Applied Pharmacology</i> , 2003, 192, 95-106.	1.3	298
38	Neurobehavioral Derangements in Adult Mice Receiving Decabrominated Diphenyl Ether (PBDE 209) during a Defined Period of Neonatal Brain Development. <i>Toxicological Sciences</i> , 2003, 76, 112-120.	1.4	282
39	Neonatal Exposure to the Brominated Flame Retardant 2,2',4,4',5-Pentabromodiphenyl Ether Causes Altered Susceptibility in the Cholinergic Transmitter System in the Adult Mouse. <i>Toxicological Sciences</i> , 2002, 67, 104-107.	1.4	172
40	The developing cholinergic system as target for environmental toxicants, nicotine and polychlorinated biphenyls (PCBs): Implications for neurotoxicological processes in mice. <i>Neurotoxicity Research</i> , 2001, 3, 37-51.	1.3	49