Noboru Hiroi

List of Publications by Year
in descending order

59 papers	4,660 citations	159585 30 h-index	149698 56 g-index
62 all docs	62 docs citations	62 times ranked	4212 citing authors

#	Article	IF	Citations
1	Tbx1, a gene encoded in 22q11.2 copy number variant, is a link between alterations in fimbria myelination and cognitive speed in mice. Molecular Psychiatry, 2022, 27, 929-938.	7.9	13
2	Maternal approach behaviors toward neonatal calls are impaired by mother's experiences of raising pups with a risk gene variant for autism. Developmental Psychobiology, 2021, 63, 108-113.	1.6	9
3	Heterozygosity of murine Crkl does not recapitulate behavioral dimensions of human 22q11.2 hemizygosity. Genes, Brain and Behavior, 2021, 20, e12719.	2.2	4
4	Computational identification of variables in neonatal vocalizations predictive for postpubertal social behaviors in a mouse model of 16p11.2 deletion. Molecular Psychiatry, 2021, 26, 6578-6588.	7.9	7
5	Presynaptic Vesicle Protein SEPTIN5 Regulates the Degradation of APP C-Terminal Fragments and the Levels of Aî². Cells, 2020, 9, 2482.	4.1	8
6	Neurobiological perspective of 22q11.2 deletion syndrome. Lancet Psychiatry,the, 2019, 6, 951-960.	7.4	70
7	Modeling and Predicting Developmental Trajectories of Neuropsychiatric Dimensions Associated With Copy Number Variations. International Journal of Neuropsychopharmacology, 2019, 22, 488-500.	2.1	19
8	Critical reappraisal of mechanistic links of copy number variants to dimensional constructs of neuropsychiatric disorders in mouse models. Psychiatry and Clinical Neurosciences, 2018, 72, 301-321.	1.8	29
9	Computational Analysis of Neonatal Mouse Ultrasonic Vocalization. Current Protocols in Mouse Biology, 2018, 8, e46.	1.2	7
10	A Self-Generated Environmental Factor as a Potential Contributor to Atypical Early Social Communication in Autism. Neuropsychopharmacology, 2017, 42, 378-378.	5.4	15
11	Human COMT over-expression confers a heightened susceptibility to dyskinesia in mice. Neurobiology of Disease, 2017, 102, 133-139.	4.4	21
12	Cry, Baby, Cry: Expression of Distress As a Biomarker and Modulator in Autism Spectrum Disorder. International Journal of Neuropsychopharmacology, 2017, 20, 498-503.	2.1	75
13	Dimensional Deconstruction and Reconstruction of CNV-Associated Neuropsychiatric Disorders. Handbook of Behavioral Neuroscience, 2016, , 285-302.	0.7	10
14	Molecular Histochemistry Identifies Peptidomic Organization and Reorganization Along Striatal Projection Units. Biological Psychiatry, 2016, 79, 415-420.	1.3	5
15	Constance E. Lieber, Theodore R. Stanley, and the Enduring Impact of Philanthropy on Psychiatry Research. Biological Psychiatry, 2016, 80, 84-86.	1.3	2
16	Neonatal Maternal Separation Alters the Capacity of Adult Neural Precursor Cells to Differentiate into Neurons Via Methylation of Retinoic Acid Receptor Gene Promoter. Biological Psychiatry, 2015, 77, 335-344.	1.3	47
17	Small Cracks in the Dam: Rare Genetic Variants Provide Opportunities to Delve into Mechanisms of Neuropsychiatric Disorders. Biological Psychiatry, 2014, 76, 91-92.	1.3	4
18	Transgenic expression of ZBP1 in neurons suppresses cocaine-associated conditioning. Learning and Memory, 2012, 19, 35-42.	1.3	4

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19	Alterations of social interaction through genetic and environmental manipulation of the 22q11.2 gene Sept5 in the mouse brain. Human Molecular Genetics, 2012, 21, 3489-3499.	2.9	53
20	Localization of septin proteins in the mouse cochlea. Hearing Research, 2012, 289, 40-51.	2.0	10
21	Mouse Models of 22q11.2-Associated Autism Spectrum Disorder. Autism-open Access, 2012, 01, 001.	0.2	15
22	Deconstructing Craving: Dissociable Cortical Control of Cue Reactivity in Nicotine Addiction. Biological Psychiatry, 2011, 69, 1052-1059.	1.3	60
23	Tbx1: identification of a 22q11.2 gene as a risk factor for autism spectrum disorder in a mouse model. Human Molecular Genetics, 2011, 20, 4775-4785.	2.9	86
24	N-Methyl-D-Aspartic Acid Receptors on Striatal Neurons Are Essential for Cocaine Cue Reactivity in Mice. Biological Psychiatry, 2010, 67, 778-780.	1.3	14
25	Emergence of Dormant Conditioned Incentive Approach by Conditioned Withdrawal in Nicotine Addiction. Biological Psychiatry, 2010, 68, 726-732.	1.3	10
26	Over-expression of a human chromosome 22q11.2 segment including TXNRD2, COMT and ARVCF developmentally affects incentive learning and working memory in mice. Human Molecular Genetics, 2009, 18, 3914-3925.	2.9	53
27	Sept5 deficiency exerts pleiotropic influence on affective behaviors and cognitive functions in mice. Human Molecular Genetics, 2009, 18, 1652-1660.	2.9	78
28	Constitutional mechanisms of vulnerability and resilience to nicotine dependence. Molecular Psychiatry, 2009, 14, 653-667.	7.9	21
29	Pleiotropic impact of constitutive fosB inactivation on nicotine-induced behavioral alterations and stress-related traits in mice. Human Molecular Genetics, 2007, 16, 820-836.	2.9	34
30	Monoamine oxidase A knockout mice exhibit impaired nicotine preference but normal responses to novel stimuli. Human Molecular Genetics, 2006, 15, 2721-2731.	2.9	44
31	Genetic susceptibility to substance dependence. Molecular Psychiatry, 2005, 10, 336-344.	7.9	107
32	A 200-kb region of human chromosome 22q11.2 confers antipsychotic-responsive behavioral abnormalities in mice. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 19132-19137.	7.1	44
33	DARPP-32 Phosphorylation Opposes the Behavioral Effects of Nicotine. Biological Psychiatry, 2005, 58, 981-989.	1.3	21
34	MAO-B knockout mice exhibit deficient habituation of locomotor activity but normal nicotine intake. Genes, Brain and Behavior, 2004, 3, 216-227.	2.2	31
35	Chronic treatment with atypical neuroleptics induces striosomal FosB/Î”FosB expression in rats. Biological Psychiatry, 2004, 55, 457-463.	1.3	44
36	Molecular dissection of dopamine receptor signaling. Journal of Chemical Neuroanatomy, 2002, 23, 237-242.	2.1	24

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37	Differential behavioral responses to cocaine are associated with dynamics of mesolimbic dopamine proteins in Lewis and Fischer 344 rats. Synapse, 2001, 41, 179-190.	1.2	80
38	Neuronal and behavioural abnormalities in striatal function in DARPP-32-mutant mice. European Journal of Neuroscience, 1999, 11, 1114-1118.	2.6	73
39	Region-specific induction of ?FosB by repeated administration of typical versus atypical antipsychotic drugs. Synapse, 1999, 33, 118-128.	1.2	89
40	Regionâ€specific induction of Î”FosB by repeated administration of typical versus atypical antipsychotic drugs. Synapse, 1999, 33, 118-128.	1.2	1
41	Dependence, Tolerance, and Alteration in Gene Expression. , 1999, , 207-211.		1
42	Increased vulnerability to cocaine in mice lacking the serotonin-1B receptor. Nature, 1998, 393, 175-178.	27.8	309
43	DARPP-32: Regulator of the Efficacy of Dopaminergic Neurotransmission. , 1998, 281, 838-842.		428
44	Regulation of Cocaine Reward by CREB. Science, 1998, 282, 2272-2275.	12.6	689
45	Essential Role of thefosB Gene in Molecular, Cellular, and Behavioral Actions of Chronic Electroconvulsive Seizures. Journal of Neuroscience, 1998, 18, 6952-6962.	3.6	115
46	Preferential localization of self-stimulation sites in striosomes/patches in the rat striatum. Proceedings of the National Academy of Sciences of the United States of America, 1998, 95, 6486-6491.	7.1	144
47	Influence of Cocaine on the JAKâ€“STAT Pathway in the Mesolimbic Dopamine System. Journal of Neuroscience, 1996, 16, 8019-8026.	3.6	50
48	Regulation of ERK (Extracellular Signal Regulated Kinase), Part of the Neurotrophin Signal Transduction Cascade, in the Rat Mesolimbic Dopamine System by Chronic Exposure to Morphine or Cocaine. Journal of Neuroscience, 1996, 16, 4707-4715.	3.6	296
49	Atypical and typical neuroleptic treatments induce distinct programs of transcription factor expression in the striatum. , 1996, 374, 70-83.		95
50	Compartmental organization of calretinin in the rat striatum. Neuroscience Letters, 1995, 197, 223-226.	2.1	23
51	Dopamine D1 receptor mutant mice are deficient in striatal expression of dynorphin and in dopamine-mediated behavioral responses. Cell, 1994, 79, 729-742.	28.9	474
52	Amphetamine conditioned cue preference and the neurobiology of drug-seeking. Seminars in Neuroscience, 1993, 5, 329-336.	2.2	28
53	The ventral pallidum area is involved in the acquisition but not expression of the amphetamine conditioned place preference. Neuroscience Letters, 1993, 156, 9-12.	2.1	58
54	Pipradrol conditioned place preference is blocked by SCH23390. Pharmacology Biochemistry and Behavior, 1992, 43, 377-380.	2.9	14

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55	The amphetamine conditioned place preference: differential involvement of dopamine receptor subtypes and two dopaminergic terminal areas. Brain Research, 1991, 552, 141-152.	2.2	149
56	The lateral nucleus of the amygdala mediates expression of the amphetamine-produced conditioned place preference. Journal of Neuroscience, 1991, 11, 2107-2116.	3.6	209
57	Place conditioning with dopamine D1 and D2 agonists injected peripherally or into nucleus accumbens. Psychopharmacology, 1991, 103, 271-276.	3.1	140
58	The reserpine-sensitive dopamine pool mediates (+)-amphetamine-conditioned reward in the place preference paradigm. Brain Research, 1990, 510, 33-42.	2.2	63
59	Conditioned stereotypy: Behavioral specification of the UCS and pharmacological investigation of the neural change. Pharmacology Biochemistry and Behavior, 1989, 32, 249-258.	2.9	30