

Arnaud Duchon

List of Publications by Year in descending order

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Version: 2024-02-01

28
papers

1,684
citations

394421

19
h-index

477307

29
g-index

32
all docs

32
docs citations

32
times ranked

2214
citing authors

#	ARTICLE	IF	CITATIONS
1	Epigallocatechinâ€gallate, a DYRK1A inhibitor, rescues cognitive deficits in Down syndrome mouse models and in humans. <i>Molecular Nutrition and Food Research</i> , 2014, 58, 278-288.	3.3	234
2	Identification of the translocation breakpoints in the Ts65Dn and Ts1Cje mouse lines: relevance for modeling down syndrome. <i>Mammalian Genome</i> , 2011, 22, 674-684.	2.2	186
3	Specific targeting of the GABA-A receptor $\alpha 5$ subtype by a selective inverse agonist restores cognitive deficits in Down syndrome mice. <i>Journal of Psychopharmacology</i> , 2011, 25, 1030-1042.	4.0	153
4	DYRK1A, a Dosage-Sensitive Gene Involved in Neurodevelopmental Disorders, Is a Target for Drug Development in Down Syndrome. <i>Frontiers in Behavioral Neuroscience</i> , 2016, 10, 104.	2.0	142
5	Analysis of mammalian gene function through broad-based phenotypic screens across a consortium of mouse clinics. <i>Nature Genetics</i> , 2015, 47, 969-978.	21.4	137
6	Excitation/inhibition balance and learning are modified by Dyrk1a gene dosage. <i>Neurobiology of Disease</i> , 2014, 69, 65-75.	4.4	104
7	A new mouse model for the trisomy of the Abcg1â€U2af1 region reveals the complexity of the combinatorial genetic code of down syndrome. <i>Human Molecular Genetics</i> , 2009, 18, 4756-4769.	2.9	101
8	Pharmacological correction of excitation/inhibition imbalance in Down syndrome mouse models. <i>Frontiers in Behavioral Neuroscience</i> , 2015, 9, 267.	2.0	57
9	Correction of cognitive deficits in mouse models of Down syndrome by a pharmacological inhibitor of DYRK1A. <i>DMM Disease Models and Mechanisms</i> , 2018, 11, .	2.4	55
10	Chronic Treatment with a Promnesiant GABA-A -Selective Inverse Agonist Increases Immediate Early Genes Expression during Memory Processing in Mice and Rectifies Their Expression Levels in a Down Syndrome Mouse Model. <i>Advances in Pharmacological Sciences</i> , 2011, 2011, 1-11.	3.7	51
11	DYRK1A, a Novel Determinant of the Methionine-Homocysteine Cycle in Different Mouse Models Overexpressing this Down-Syndrome-Associated Kinase. <i>PLoS ONE</i> , 2009, 4, e7540.	2.5	50
12	Opposite Phenotypes of Muscle Strength and Locomotor Function in Mouse Models of Partial Trisomy and Monosomy 21 for the Proximal Hspa13-App Region. <i>PLoS Genetics</i> , 2015, 11, e1005062.	3.5	39
13	Modeling Chromosomes in Mouse to Explore the Function of Genes, Genomic Disorders, and Chromosomal Organization. <i>PLoS Genetics</i> , 2006, 2, e86.	3.5	38
14	Prenatal treatment with EGCG enriched green tea extract rescues GAD67 related developmental and cognitive defects in Down syndrome mouse models. <i>Scientific Reports</i> , 2019, 9, 3914.	3.3	35
15	The telomeric part of the human chromosome 21 from Cstb to Prmt2 is not necessary for the locomotor and short-term memory deficits observed in the Tc1 mouse model of Down syndrome. <i>Behavioural Brain Research</i> , 2011, 217, 271-281.	2.2	34
16	The in vivo Down syndrome genomic library in mouse. <i>Progress in Brain Research</i> , 2012, 197, 169-197.	1.4	33
17	Modeling the monosomy for the telomeric part of human chromosome 21 reveals haploinsufficient genes modulating the inflammatory and airway responses. <i>Human Molecular Genetics</i> , 2007, 16, 2040-2052.	2.9	30
18	Dyrk1a from Gene Function in Development and Physiology to Dosage Correction across Life Span in Down Syndrome. <i>Genes</i> , 2021, 12, 1833.	2.4	28

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19	Long-lasting correction of in vivo LTP and cognitive deficits of mice modelling Down syndrome with an α -selective GABA _A inverse agonist. <i>British Journal of Pharmacology</i> , 2020, 177, 1106-1118.	5.4	27
20	The App-Runx1 Region Is Critical for Birth Defects and Electrocardiographic Dysfunctions Observed in a Down Syndrome Mouse Model. <i>PLoS Genetics</i> , 2012, 8, e1002724.	3.5	25
21	Multi-influential genetic interactions alter behaviour and cognition through six main biological cascades in Down syndrome mouse models. <i>Human Molecular Genetics</i> , 2021, 30, 771-788.	2.9	24
22	A Small Compound Targeting Prohibitin with Potential Interest for Cognitive Deficit Rescue in Aging mice and Tau Pathology Treatment. <i>Scientific Reports</i> , 2020, 10, 1143.	3.3	21
23	Inducing Segmental Aneuploid Mosaicism in the Mouse Through Targeted Asymmetric Sister Chromatid Event of Recombination. <i>Genetics</i> , 2008, 180, 51-59.	2.9	17
24	TUBG1 missense variants underlying cortical malformations disrupt neuronal locomotion and microtubule dynamics but not neurogenesis. <i>Nature Communications</i> , 2019, 10, 2129.	12.8	17
25	Dosage of the Abcg1-U2af1 Region Modifies Locomotor and Cognitive Deficits Observed in the Tc1 Mouse Model of Down Syndrome. <i>PLoS ONE</i> , 2015, 10, e0115302.	2.5	16
26	Deletion of the <i>App-Runx1</i> region in mice models human partial monosomy 21. <i>DMM Disease Models and Mechanisms</i> , 2015, 8, 623-634.	2.4	12
27	DYRK1A overexpression decreases plasma lecithin:cholesterol acyltransferase activity and apolipoprotein A-I levels. <i>Molecular Genetics and Metabolism</i> , 2013, 110, 371-377.	1.1	5
28	Controlled Somatic and Germline Copy Number Variation in the Mouse Model. <i>Current Genomics</i> , 2010, 11, 470-480.	1.6	3