Rachel Wevrick

List of Publications by Year
in descending order

73	4,182	117453	114278
papers	citations	34 h-index	63 g-index

74	74	74	3369
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	The N-terminal domain of the Schaafâ€“Yang syndrome protein MAGEL2 likely has a role in RNA metabolism. Journal of Biological Chemistry, 2021, 297, 100959.	1.6	5
2	Regulation of autism-relevant behaviors by cerebellarâ€“prefrontal cortical circuits. Nature Neuroscience, 2020, 23, 1102-1110.	7.1	149
3	The necdin interactome: evaluating the effects of amino acid substitutions and cell stress using proximity-dependent biotinylation (BioID) and mass spectrometry. Human Genetics, 2020, 139, 1513-1529.	1.8	4
4	Disentangling ingestive behavior-related phenotypes in Praderâ€“Willi syndrome: Integrating information from nonclinical studies and clinical trials to better understand the pathophysiology of hyperphagia and obesity. Physiology and Behavior, 2020, 219, 112864.	1.0	4
5	A MAGEL2-deubiquitinase complex modulates the ubiquitination of circadian rhythm protein CRY1. PLoS ONE, 2020, 15, e0230874.	1.1	14
6	Preclinical Testing in Translational Animal Models of Prader-Willi Syndrome: Overview and Gap Analysis. Molecular Therapy - Methods and Clinical Development, 2019, 13, 344-358.	1.8	22
7	Clinical and genetic analysis of children with a dual diagnosis of Tourette syndrome and autism spectrum disorder. Journal of Psychiatric Research, 2019, 111, 145-153.	1.5	10
8	Chronic diazoxide treatment decreases fat mass and improves endurance capacity in an obese mouse model of Prader-Willi syndrome. Molecular Genetics and Metabolism, 2018, 123, 511-517.	0.5	15
9	Genetic analysis of very obese children with autism spectrum disorder. Molecular Genetics and Genomics, 2018, 293, 725-736.	1.0	7
10	ROHHAD and Prader-Willi syndrome (PWS): clinical and genetic comparison. Orphanet Journal of Rare Diseases, 2018, 13, 124.	1.2	27
11	The Prader-Willi syndrome proteins MAGEL2 and necdin regulate leptin receptor cell surface abundance through ubiquitination pathways. Human Molecular Genetics, 2017, 26, 4215-4230.	1.4	41
12	Magel2â€•null mice are hyperâ€•responsive to setmelanotide, a melanocortin 4 receptor agonist. British Journal of Pharmacology, 2016, 173, 2614-2621.	2.7	26
13	Targeting the endocannabinoid/CB1 receptor system for treating obesity in Praderâ€“Willi syndrome. Molecular Metabolism, 2016, 5, 1187-1199.	3.0	64
14	Muscle dysfunction caused by loss ofMagel2in a mouse model of Prader-Willi and Schaaf-Yang syndromes. Human Molecular Genetics, 2016, 25, 3798-3809.	1.4	38
15	Sleeve gastrectomy leads to weight loss in the Magel2 knockout mouse. Surgery for Obesity and Related Diseases, 2016, 12, 1795-1802.	1.0	16
16	Dopamine pathway imbalance in mice lacking Magel2, a Prader-Willi syndrome candidate gene.. Behavioral Neuroscience, 2016, 130, 448-459.	0.6	20
17	Evaluation of melanoma antigen (MAGE) gene expression in human cancers using The Cancer Genome Atlas. Cancer Genetics, 2015, 208, 25-34.	0.2	15
18	Progressive postnatal decline in leptin sensitivity of arcuate hypothalamic neurons in theMagel2-null mouse model of Praderâ€“Willi syndrome. Human Molecular Genetics, 2015, 24, 4276-4283.	1.4	37

#	Article	IF	Citations
19	Magel2, a Prader-Willi syndrome candidate gene, modulates the activities of circadian rhythm proteins in cultured cells. Journal of Circadian Rhythms, 2014, 9, 12.	2.9	20
20	Recommendations for the investigation of animal models of Praderâ€“Willi syndrome. Mammalian Genome, 2013, 24, 165-178.	1.0	37
21	Magel2 Is Required for Leptin-Mediated Depolarization of POMC Neurons in the Hypothalamic Arcuate Nucleus in Mice. PLoS Genetics, 2013, 9, e1003207.	1.5	60
22	Stochastic Loss of Silencing of the Imprinted Ndn/NDN Allele, in a Mouse Model and Humans with Prader-Willi Syndrome, Has Functional Consequences. PLoS Genetics, 2013, 9, e1003752.	1.5	30
23	Leptin signaling defects in a mouse model of Prader-Willi syndrome. Rare Diseases (Austin, Tex), 2013, 1, e24421.	1.8	8
24	The Smc5/Smc6/MAGE Complex Confers Resistance to Caffeine and Genotoxic Stress in Drosophila melanogaster. PLoS ONE, 2013, 8, e59866.	1.1	19
25	Claudin-4 forms a paracellular barrier, revealing the interdependence of claudin expression in the loose epithelial cell culture model opossum kidney cells. American Journal of Physiology - Cell Physiology, 2012, 303, C1278-C1291.	2.1	30
26	Necdin, a p53 target gene, regulates the quiescence and response to genotoxic stress of hematopoietic stem/progenitor cells. Blood, 2012, 120, 1601-1612.	0.6	69
27	Coâ€morbidity of complex genetic disorders and hypersomnias of central origin: lessons from the underlying neurobiology of wake and sleep. Clinical Genetics, 2012, 82, 379-387.	1.0	1
28	Loss of the Praderâ€“Willi obesity syndrome protein necdin promotes adipogenesis. Gene, 2012, 497, 45-51.	1.0	26
29	Energy homeostasis in Praderâ€“Willi Syndrome: How clinical research informs studies of animal models of genetic obesity. American Journal of Medical Genetics, Part A, 2012, 158A, 966-968.	0.7	6
30	Impaired Hypothalamic Regulation of Endocrine Function and Delayed Counterregulatory Response to Hypoglycemia in Magel2-Null Mice. Endocrinology, 2011, 152, 967-978.	1.4	58
31	Loss of Necdin impairs myosin activation and delays cell polarization. Genesis, 2010, 48, 540-553.	0.8	12
32	Regionally reduced brain volume, altered serotonin neurochemistry, and abnormal behavior in mice null for the circadian rhythm output gene Magel2. American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 2009, 150B, 1085-1099.	1.1	90
33	Loss of Magel2, a Candidate Gene for Features of Prader-Willi Syndrome, Impairs Reproductive Function in Mice. PLoS ONE, 2009, 4, e4291.	1.1	83
34	Prader-Willi Syndrome. , 2009, , 223-250.		0
35	Necdin Regulates Hematopoietic Stem Cell Quiescence and Sensitivity to Genotoxic Stress.. Blood, 2009, 114, 379-379.	0.6	1
36	Loss of the Praderâ€Willi syndrome protein necdin causes defective migration, axonal outgrowth, and survival of embryonic sympathetic neurons. Developmental Dynamics, 2008, 237, 1935-1943.	0.8	42

#	Article	IF	Citations
37	The Praderâ€“Willi syndrome protein necdin interacts with the E1A-like inhibitor of differentiation EID-1 and promotes myoblast differentiation. Differentiation, 2008, 76, 994-1005.	1.0	26
38	Neurodevelopmental Abnormalities in the Brainstem of Prenatal Mice Lacking the Prader-Willi Syndrome Gene Necdin. Advances in Experimental Medicine and Biology, 2008, 605, 139-143.	0.8	7
39	Necdin, a Prader-Willi syndrome candidate gene, regulates gonadotropin-releasing hormone neurons during development. Human Molecular Genetics, 2008, 18, 248-260.	1.4	90
40	Respiratory control abnormalities in necdinnull mice: implications for the pathogenesis of Prader-Willi syndrome. , 2008, , 259-269.		0
41	Inactivation of the mouse Magel2 gene results in growth abnormalities similar to Prader-Willi syndrome. Human Molecular Genetics, 2007, 16, 2713-2719.	1.4	170
42	The imprinted gene Magel2 regulates normal circadian output. Nature Genetics, 2007, 39, 1266-1272.	9.4	196
43	inv dup(15) and inv dup(22). , 2006, , 315-325.		0
44	Evaluation of Praderâ€Willi Syndrome Gene MAGEL2 in Severe Childhoodâ€Onset Obesity. Obesity, 2005, 13, 1841-1842.	4.0	5
45	Chromatin modification of the human imprintedNDN (necdin) gene detected by in vivo footprinting. Journal of Cellular Biochemistry, 2005, 94, 1046-1057.	1.2	4
46	Genome-wide analysis of gene transcription in the hypothalamus. Physiological Genomics, 2005, 22, 191-196.	1.0	13
47	Essential role for the Praderâ€“Willi syndrome protein necdin in axonal outgrowth. Human Molecular Genetics, 2005, 14, 627-637.	1.4	141
48	Developmental Abnormalities of Neuronal Structure and Function in Prenatal Mice Lacking the Prader-Willi Syndrome Gene Necdin. American Journal of Pathology, 2005, 167, 175-191.	1.9	86
49	Tissue-specific and imprinted epigenetic modifications of the human NDN gene. Nucleic Acids Research, 2004, 32, 3376-3382.	6.5	53
50	Praderâ€“Willi syndrome transcripts are expressed in phenotypically significant regions of the developing mouse brain. Gene Expression Patterns, 2003, 3, 599-609.	0.3	70
51	A MAGE/NDN-like gene in zebrafish. Developmental Dynamics, 2003, 228, 475-479.	0.8	15
52	Absence ofNdn, Encoding the Prader-Willi Syndrome-Deleted Genenecdin, Results in Congenital Deficiency of Central Respiratory Drive in Neonatal Mice. Journal of Neuroscience, 2003, 23, 1569-1573.	1.7	121
53	The role of genomic imprinting in human developmental disorders: lessons from Prader-Willi syndrome. Clinical Genetics, 2001, 59, 156-164.	1.0	49
54	A necdin/MAGE-like gene in the chromosome 15 autism susceptibility region: expression, imprinting, and mapping of the human and mouse orthologues. BMC Genetics, 2001, 2, 22.	2.7	33

#	Article	IF	Citations
55	Establishment and Maintenance of DNA Methylation Patterns in Mouse Ndn : Implications for Maintenance of Imprinting in Target Genes of the Imprinting Center. Molecular and Cellular Biology, 2001, 21, 2384-2392.	1.1	48
56	Identification of Novel Imprinted Transcripts in the Prader-Willi Syndrome and Angelman Syndrome Deletion Region: Further Evidence for Regional Imprinting Control. American Journal of Human Genetics, 2000, 66, 848-858.	2.6	56
57	Disruption of the mouse necdin gene results in early post-natal lethality. Nature Genetics, 1999, 23, 199-202.	9.4	191
58	A mouse model for Prader-Willi syndrome imprinting-centre mutations. Nature Genetics, 1998, 19, 25-31.	9.4	285
59	An Imprinted Mouse Transcript Homologous to the Human Imprinted in Prader-Willi Syndrome (IPW) Gene. Human Molecular Genetics, 1997, 6, 325-332.	1.4	105
60	The Necdin Gene is Deleted in Prader-Willi Syndrome and is Imprinted in Human and Mouse. Human Molecular Genetics, 1997, 6, 1873-1878.	1.4	226
61	'Glow in the Dark' crayons as inexpensive autoradiography markers. Technical Tips Online, 1997, 2, 87-88.	0.2	1
62	Human centromeric DNAs. Human Genetics, 1997, 100, 291-304.	1.8	217
63	Diagnostic test for the Prader-Willi syndrome by SNRPN expression in blood. Lancet, The, 1996, 348, 1068-1069.	6.3	46
64	Expression of the Fanconi Anemia Gene FAC in Human Cell Lines: Lack of Effect of Oxygen Tension. Blood Cells, Molecules, and Diseases, 1995, 21, 182-191.	0.6	18
65	Identification of a novel paternally expressed gene in the Prader - Willi syndrome region. Human Molecular Genetics, 1994, 3, 1877-1883.	1.4	243
66	Mapping of the murine and rat Facc genes and assessment of flexed-tail as a candidate mouse homolog of Fanconi anemia group C. Mammalian Genome, 1993, 4, 440-444.	1.0	9
67	Mammalian DNA-repair genes. Current Opinion in Genetics and Development, 1993, 3, 470-474.	1.5	6
68	Cloning and analysis of the murine Fanconi anemia group C cDNA. Human Molecular Genetics, 1993, 2, 655-662.	1.4	68
69	Pulsed-Field and Two-Dimensional Gel Electrophoresis of Long Arrays of Tandemly Repeated DNA: Analysis of Human Centromeric Alpha Satellite. , 1992, 12, 299-318.		10
70	Structure of DNA near long tandem arrays of alpha satellite DNA at the centromere of human chromosome 7. Genomics, 1992, 14, 912-923.	1.3	74
71	Physical map of the centromeric region of human chromosome 7: relationship between two distinct alpha satellite arrays. Nucleic Acids Research, 1991, 19, 2295-2301.	6.5	82
72	Long-range organization of tandem arrays of alpha satellite DNA at the centromeres of human chromosomes: high-frequency array-length polymorphism and meiotic stability.. Proceedings of the National Academy of Sciences of the United States of America, 1989, 86, 9394-9398.	3.3	220

#	Article	IF	Citations
73	Evolution of bubbles in vacuum. Canadian Journal of Physics, 1986, 64, 165-173.	0.4	18