FranÃ§oise Muscatelli

List of Publications by Year
in descending order

36	3,623	257450 24	330143 37
papers	citations	h-index	g-index

44	44	44	2935
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Early life oxytocin treatment improves thermo-sensory reactivity and maternal behavior in neonates lacking the autism-associated gene Magel2. Neuropsychopharmacology, 2022, 47, 1901-1912.	5.4	9
2	Correction of vasopressin deficit in the lateral septum ameliorates social deficits of mouse autism model. Journal of Clinical Investigation, 2021, 131, .	8.2	23
3	The impact of oxytocin on neurite outgrowth and synaptic proteins in Magel2â€deficient mice. Developmental Neurobiology, 2021, 81, 366-388.	3.0	16
4	Oxytocin administration in neonates shapes hippocampal circuitry and restores social behavior in a mouse model of autism. Molecular Psychiatry, 2021, 26, 7582-7595.	7.9	45
5	Colocalization of Oxtr with Prader-Willi syndrome transcripts in the trigeminal ganglion of neonatal mice. Human Molecular Genetics, 2020, 29, 2065-2075.	2.9	4
6	Wired for eating: how is an active feeding circuitry established in the postnatal brain?. Current Opinion in Neurobiology, 2018, 52, 165-171.	4.2	15
7	The Use of Oxytocin to Improve Feeding and Social Skills in Infants With Praderâ€“Willi Syndrome. Pediatrics, 2017, 139, .	2.1	117
8	Oxytocin Signaling in the Early Life of Mammals: Link to Neurodevelopmental Disorders Associated with ASD. Current Topics in Behavioral Neurosciences, 2017, 35, 239-268.	1.7	30
9	Necdin shapes serotonergic development and SERT activity modulating breathing in a mouse model for Prader-Willi syndrome. ELife, 2017, 6, .	6.0	27
10	An Early Postnatal Oxytocin Treatment Prevents Social and Learning Deficits in Adult Mice Deficient for Magel2, a Gene Involved in Prader-Willi Syndrome and Autism. Biological Psychiatry, 2015, 78, 85-94.	1.3	140
11	Ontogenesis of oxytocin pathways in the mammalian brain: late maturation and psychosocial disorders. Frontiers in Neuroanatomy, 2014, 8, 164.	1.7	81
12	Tubacin prevents neuronal migration defects and epileptic activity caused by rat Srpx2 silencing in utero. Brain, 2013, 136, 2457-2473.	7.6	52
13	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.	3.5	30
14	Natural breaking of the maternal silence at the mouse and human imprinted Prader-Willi locus. Rare Diseases (Austin, Tex), 2013, 1, e27228.	1.8	21
15	Inactivation ofSocs3in the Hypothalamus Enhances the Hindbrain Response to Endogenous Satiety Signals via Oxytocin Signaling. Journal of Neuroscience, 2012, 32, 17097-17107.	3.6	42
16	Functional Consequences of Necdin Nucleocytoplasmic Localization. PLoS ONE, 2012, 7, e33786.	2.5	10
17	Necdin Protects Embryonic Motoneurons from Programmed Cell Death. PLoS ONE, 2011, 6, e23764.	2.5	22
18	A single postnatal injection of oxytocin rescues the lethal feeding behaviour in mouse newborns deficient for the imprinted Magel2 gene. Human Molecular Genetics, 2010, 19, 4895-4905.	2.9	198

#	Article	IF	Citations
19	Breathing deficits of the Prader-Willi syndrome. Respiratory Physiology and Neurobiology, 2009, 168, 119-124.	1.6	27
20	Necdin Gene, Respiratory Disturbances and Prader-Willi Syndrome. Advances in Experimental Medicine and Biology, 2008, 605, 159-164.	1.6	23
21	Necdin Plays a Role in the Serotonergic Modulation of the Mouse Respiratory Network: Implication for Prader-Willi Syndrome. Journal of Neuroscience, 2008, 28, 1745-1755.	3.6	73
22	Necdin mediates skeletal muscle regeneration by promoting myoblast survival and differentiation. Journal of Cell Biology, 2007, 179, 305-319.	5.2	46
23	Sensory defects in Necdin deficient mice result from a loss of sensory neurons correlated within an increase of developmental programmed cell death. BMC Developmental Biology, 2006, 6, 56.	2.1	46
24	The Prader-Willi syndrome murine imprinting center is not involved in the spatio-temporal transcriptional regulation of the Necdin gene. BMC Genetics, 2005, 6, 1.	2.7	56
25	ADAR2-mediated editing of RNA substrates in the nucleolus is inhibited by C/D small nucleolar RNAs. Journal of Cell Biology, 2005, 169, 745-753.	5.2	223
26	Dynamic developmental regulation of the large non-coding RNA associated with the mouse 7C imprinted chromosomal region. Developmental Biology, 2005, 286, 587-600.	2.0	67
27	Regulation of the large (Â1000 kb) imprinted murine Ube3a antisense transcript by alternative exons upstream of Snurf/Snrpn. Nucleic Acids Research, 2004, 32, 3480-3492.	14.5	139
28	Expression of the Prader-Willi gene Necdin during mouse nervous system development correlates with neuronal differentiation and p75NTR expression. Gene Expression Patterns, 2003, 3, 761-765.	0.8	48
29	Comprehensive methylation analysis in typical and atypical PWS and AS patients with normal biparental chromosomes 15. European Journal of Human Genetics, 2001, 9, 519-526.	2.8	13
30	Two Members of the HumanMAGEBGene Family Located in Xp21.3 Are Expressed in Tumors of Various Histological Origins. Genomics, 1997, 46, 397-408.	2.9	119
31	The human necdin gene, NDN, is maternally imprinted and located in the Prader-Willi syndrome chromosomal region. Nature Genetics, 1997, 17, 357-361.	21.4	241
32	The Mouse Necdin Gene Is Expressed from the Paternal Allele Only and Lies in the 7C Region of the Mouse Chromosome 7, a Region of Conserved Synteny to the Human Prader-Willi Syndrome Region. European Journal of Human Genetics, 1997, 5, 324-332.	2.8	30
33	An unusual member of the nuclear hormone receptor superfamily responsible for X-linked adrenal hypoplasia congenita. Nature, 1994, 372, 635-641.	27.8	796
34	Mutations in the DAX-1 gene give rise to both X-linked adrenal hypoplasia congenita and hypogonadotropic hypogonadism. Nature, 1994, 372, 672-676.	27.8	722
35	The High-Affinity Interleukin 8 Receptor Gene (IL8RA) Maps to the 2q33-q36 Region of the Human Genome: Cloning of a Pseudogene (IL8RBP) for the Low-Affinity Receptor. Genomics, 1993, 16, 248-251.	2.9	23
36	Isolation of the human Xp21 glycerol kinase gene by positional cloning. Human Molecular Genetics, 1993, 2, 107-114.	2.9	42