Cristina NÃºÃ±ez

List of Publications by Year
in descending order

31 papers	724 citations	430874 18 h-index	552781 26 g-index
35 all docs	35 docs citations	35 times ranked	777 citing authors

#	Article	IF	Citations
1	Molecular Mechanisms Underlying the Retrieval and Extinction of Morphine Withdrawal-Associated Memories in the Basolateral Amygdala and Dentate Gyrus. Biomedicines, 2022, 10, 588.	3.2	5
2	Unraveling the molecular mechanisms involved in alcohol intake and withdrawal in adolescent mice exposed to alcohol during early life stages. Progress in Neuro-Psychopharmacology and Biological Psychiatry, 2021, 104, 110025.	4.8	3
3	Distinct Regulation of Dopamine D3 Receptor in the Basolateral Amygdala and Dentate Gyrus during the Reinstatement of Cocaine CPP Induced by Drug Priming and Social Stress. International Journal of Molecular Sciences, 2021, 22, 3100.	4.1	8
4	Blockade of D3 receptor prevents changes in DAT and D3R expression in the mesolimbic dopaminergic circuit produced by social stress- and cocaine prime-induced reinstatement of cocaine-CPP. Journal of Psychopharmacology, 2020, 34, 1300-1315.	4.0	5
5	Modulation of stress- and cocaine prime-induced reinstatement of conditioned place preference after memory extinction through dopamine D3 receptor. Progress in Neuro-Psychopharmacology and Biological Psychiatry, 2019, 92, 308-320.	4.8	15
6	Pharmacological modulation of the behavioral effects of social defeat in memory and learning in male mice. Psychopharmacology, 2019, 236, 2797-2810.	3.1	10
7	The involvement of CRF1 receptor within the basolateral amygdala and dentate gyrus in the naloxone-induced conditioned place aversion in morphine-dependent mice. Progress in Neuro-Psychopharmacology and Biological Psychiatry, 2018, 84, 102-114.	4.8	21
8	Repeated social defeat and the rewarding effects of cocaine in adult and adolescent mice: dopamine transcription factors, proBDNF signaling pathways, and the TrkB receptor in the mesolimbic system. Psychopharmacology, 2017, 234, 2063-2075.	3.1	37
9	Glucocorticoid Homeostasis in the Dentate Gyrus Is Essential for Opiate Withdrawal-Associated Memories. Molecular Neurobiology, 2017, 54, 6523-6541.	4.0	21
10	Different contribution of glucocorticoids in the basolateral amygdala to the formation and expression of opiate withdrawal-associated memories. Psychoneuroendocrinology, 2016, 74, 350-362.	2.7	18
11	Regulation of dopaminergic markers expression in response to acute and chronic morphine and to morphine withdrawal. Addiction Biology, 2016, 21, 374-386.	2.6	18
12	Corticotropin-releasing factor 1 receptor mediates the activity of the reward system evoked by morphine-induced conditioned place preference. Neuropharmacology, 2015, 95, 168-180.	4.1	26
13	Morphine regulates Argonaute 2 and TH expression and activity but not miR-133b in midbrain dopaminergic neurons. Addiction Biology, 2015, 20, 104-119.	2.6	25
14	Dysregulation of dopaminergic regulatory mechanisms in the mesolimbic pathway induced by morphine and morphine withdrawal. Brain Structure and Function, 2015, 220, 1901-1919.	2.3	18
15	Glial activation and midkine and pleiotrophin transcription in the ventral tegmental area are modulated by morphine administration. Journal of Neuroimmunology, 2014, 274, 244-248.	2.3	14
16	Morphine administration modulates expression of <scp>A</scp>rgonaute 2 and dopamineâ€related transcription factors involved in midbrain dopaminergic neurons function. British Journal of Pharmacology, 2013, 168, 1889-1901.	5.4	20
17	Differential Changes in Expression of Stress- and Metabolic-Related Neuropeptides in the Rat Hypothalamus during Morphine Dependence and Withdrawal. PLoS ONE, 2013, 8, e67027.	2.5	18
18	Involvement of Noradrenergic Transmission in the PVN on CREB Activation, TORC1 Levels, and Pituitary-Adrenal Axis Activity during Morphine Withdrawal. PLoS ONE, 2012, 7, e31119.	2.5	10

#	Article	IF	Citations
19	Hypothalamic Orexin-A Neurons Are Involved in the Response of the Brain Stress System to Morphine Withdrawal. PLoS ONE, 2012, 7, e36871.	2.5	47
20	Glucocorticoids Regulation of FosB/Î”FosB Expression Induced by Chronic Opiate Exposure in the Brain Stress System. PLoS ONE, 2012, 7, e50264.	2.5	31
21	ÎFosB expression in the brain stress system from adrenalectomized rats during morphine dependence. Pharmacological Reports, 2011, 63, 255-256.	3.3	0
22	CRF₂ mediates the increased noradrenergic activity in the hypothalamic paraventricular nucleus and the negative state of morphine withdrawal in rats. British Journal of Pharmacology, 2011, 162, 851-862.	5.4	24
23	Induction of FosB/Î”FosB in the brain stress systemâ€related structures during morphine dependence and withdrawal. Journal of Neurochemistry, 2010, 114, 475-487.	3.9	36
24	Effects of Corticotropin-Releasing Factor Receptor-1 Antagonists on the Brain Stress System Responses to Morphine Withdrawal. Molecular Pharmacology, 2010, 77, 864-873.	2.3	50
25	Changes in metabolic-related variables during chronic morphine treatment. Neurochemistry International, 2010, 57, 323-330.	3.8	29
26	Elevated Glucocorticoid Levels Are Responsible for Induction of Tyrosine Hydroxylase mRNA Expression, Phosphorylation, and Enzyme Activity in the Nucleus of the Solitary Tract during Morphine Withdrawal. Endocrinology, 2009, 150, 3118-3127.	2.8	41
27	Effects of rolipram and diazepam on the adaptive changes induced by morphine withdrawal in the hypothalamic paraventricular nucleus. European Journal of Pharmacology, 2009, 620, 1-8.	3.5	10
28	Regulation of extracellular signal-regulated kinases (ERKs) by naloxone-induced morphine withdrawal in the brain stress system. Naunyn-Schmiedeberg's Archives of Pharmacology, 2008, 378, 407-420.	3.0	19
29	Regulation of Serine (Ser)-31 and Ser40 Tyrosine Hydroxylase Phosphorylation during Morphine Withdrawal in the Hypothalamic Paraventricular Nucleus and Nucleus Tractus Solitarius-A2 Cell Group: Role of ERK1/2. Endocrinology, 2007, 148, 5780-5793.	2.8	37
30	Activation of stressâ€related hypothalamic neuropeptide gene expression during morphine withdrawal. Journal of Neurochemistry, 2007, 101, 1060-1071.	3.9	54
31	Morphine withdrawal-induced c-fos expression in the hypothalamic paraventricular nucleus is dependent on the activation of catecholaminergic neurones. Journal of Neurochemistry, 2002, 83, 132-140.	3.9	50