M Victoria MilanÃ©s

List of Publications by Year
in descending order

132 papers	2,211 citations	236833 25 h-index	360920 35 g-index
137 all docs	137 docs citations	137 times ranked	1533 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.	1.4	5
2	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.	1.8	8
3	Role of glucocorticoids on noradrenergic and dopaminergic neurotransmission within the basolateral amygdala and dentate gyrus during morphine withdrawal place aversion. Addiction Biology, 2020, 25, e12728.	1.4	4
4	Conditioned aversive memory associated with morphine withdrawal increases brainâ€derived neurotrophic factor in dentate gyrus and basolateral amygdala. Addiction Biology, 2020, 25, e12792.	1.4	4
5	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.	2.0	5
6	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.	2.5	15
7	Binge ethanol and MDMA combination exacerbates HSP27 and Trx-1 (biomarkers of toxic cardiac) Tj ETQq1 1 0.784314 rgBT /Overlock	2.0	8
8	Pharmacological modulation of the behavioral effects of social defeat in memory and learning in male mice. Psychopharmacology, 2019, 236, 2797-2810.	1.5	10
9	Distinct regulation pattern of Egr-1, BDNF and Arc during morphine-withdrawal conditioned place aversion paradigm: Role of glucocorticoids. Behavioural Brain Research, 2019, 360, 244-254.	1.2	5
10	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.	2.5	21
11	Acute Morphine, Chronic Morphine, and Morphine Withdrawal Differently Affect Pleiotrophin, Midkine, and Receptor Protein Tyrosine Phosphatase Î²/Î¶ Regulation in the Ventral Tegmental Area. Molecular Neurobiology, 2017, 54, 495-510.	1.9	9
12	Glucocorticoid receptor but not mineralocorticoid receptor mediates the activation of ERK pathway and CREB during morphine withdrawal. Addiction Biology, 2017, 22, 342-353.	1.4	14
13	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.	1.5	37
14	Glucocorticoid Homeostasis in the Dentate Gyrus Is Essential for Opiate Withdrawal-Associated Memories. Molecular Neurobiology, 2017, 54, 6523-6541.	1.9	21
15	Different contribution of glucocorticoids in the basolateral amygdala to the formation and expression of opiate withdrawal-associated memories. Psychoneuroendocrinology, 2016, 74, 350-362.	1.3	18
16	Regulation of dopaminergic markers expression in response to acute and chronic morphine and to morphine withdrawal. Addiction Biology, 2016, 21, 374-386.	1.4	18
17	Regulation of Pleiotrophin, Midkine, Receptor Protein Tyrosine Phosphatase Î²/Î¶, and Their Intracellular Signaling Cascades in the Nucleus Accumbens During Opiate Administration. International Journal of Neuropsychopharmacology, 2016, 19, pyv077.	1.0	6
18	Maternal Separation Impairs Cocaine-Induced Behavioural Sensitization in Adolescent Mice. PLoS ONE, 2016, 11, e0167483.	1.1	36

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19	Binge Ethanol and MDMA Combination Exacerbates Toxic Cardiac Effects by Inducing Cellular Stress. PLoS ONE, 2015, 10, e0141502.	1.1	15
20	Sympathetic activity induced by naloxone-precipitated morphine withdrawal is blocked in genetically engineered mice lacking functional CRF1 receptor. Toxicology and Applied Pharmacology, 2015, 283, 42-49.	1.3	3
21	Corticotropin-releasing factor 1 receptor mediates the activity of the reward system evoked by morphine-induced conditioned place preference. Neuropharmacology, 2015, 95, 168-180.	2.0	26
22	Expression of heat shock protein 27 and troponin T and troponin I after naloxone-precipitated morphine withdrawal. European Journal of Pharmacology, 2015, 766, 142-150.	1.7	8
23	Morphine regulates Argonaute 2 and TH expression and activity but not miR-133b in midbrain dopaminergic neurons. Addiction Biology, 2015, 20, 104-119.	1.4	25
24	Dysregulation of dopaminergic regulatory mechanisms in the mesolimbic pathway induced by morphine and morphine withdrawal. Brain Structure and Function, 2015, 220, 1901-1919.	1.2	18
25	Sex Differences between CRF1 Receptor Deficient Mice following Naloxone-Precipitated Morphine Withdrawal in a Conditioned Place Aversion Paradigm: Implication of HPA Axis. PLoS ONE, 2015, 10, e0121125.	1.1	24
26	CP-154,526 Modifies CREB Phosphorylation and Thioredoxin-1 Expression in the Dentate Gyrus following Morphine-Induced Conditioned Place Preference. PLoS ONE, 2015, 10, e0136164.	1.1	14
27	Corticotropinâ€releasing factor (CRF) receptorâ€1 is involved in cardiac noradrenergic activity observed during naloxoneâ€precipitated morphine withdrawal. British Journal of Pharmacology, 2014, 171, 688-700.	2.7	13
28	Cardiac adverse effects of naloxone-precipitated morphine withdrawal on right ventricle: Role of corticotropin-releasing factor (CRF) 1 receptor. Toxicology and Applied Pharmacology, 2014, 275, 28-35.	1.3	6
29	Spironolactone decreases the somatic signs of opiate withdrawal by blocking the mineralocorticoid receptors (MR). Toxicology, 2014, 326, 36-43.	2.0	10
30	Glial activation and midkine and pleiotrophin transcription in the ventral tegmental area are modulated by morphine administration. Journal of Neuroimmunology, 2014, 274, 244-248.	1.1	14
31	Brain stress system response after morphine-conditioned place preference. International Journal of Neuropsychopharmacology, 2013, 16, 1999-2011.	1.0	13
32	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.	2.7	20
33	Differential Changes in Expression of Stress- and Metabolic-Related Neuropeptides in the Rat Hypothalamus during Morphine Dependence and Withdrawal. PLoS ONE, 2013, 8, e67027.	1.1	18
34	Crosstalk between G protein-coupled receptors (GPCRs) and tyrosine kinase receptor (TXR) in the heart after morphine withdrawal. Frontiers in Pharmacology, 2013, 4, 164.	1.6	1
35	Morphine Withdrawal Activates Hypothalamic-Pituitary-Adrenal Axis and Heat Shock Protein 27 in the Left Ventricle: The Role of Extracellular Signal-Regulated Kinase. Journal of Pharmacology and Experimental Therapeutics, 2012, 342, 665-675.	1.3	14
36	Glucocorticoid receptors participate in the opiate withdrawalâ€induced stimulation of rats NTS noradrenergic activity and in the somatic signs of morphine withdrawal. British Journal of Pharmacology, 2012, 166, 2136-2147.	2.7	19

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37	Accumbal dopamine, noradrenaline and serotonin activity after naloxone-conditioned place aversion in morphine-dependent mice. Neurochemistry International, 2012, 61, 433-440.	1.9	19
38	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.	1.1	10
39	Hypothalamic Orexin-A Neurons Are Involved in the Response of the Brain Stress System to Morphine Withdrawal. PLoS ONE, 2012, 7, e36871.	1.1	47
40	Role of Corticotropin-Releasing Factor (CRF) Receptor-1 on the Catecholaminergic Response to Morphine Withdrawal in the Nucleus Accumbens (NAc). PLoS ONE, 2012, 7, e47089.	1.1	18
41	Glucocorticoids Regulation of FosB/Î”FosB Expression Induced by Chronic Opiate Exposure in the Brain Stress System. PLoS ONE, 2012, 7, e50264.	1.1	31
42	Restricted role of CRF1 receptor for the activity of brainstem catecholaminergic neurons in the negative state of morphine withdrawal. Psychopharmacology, 2012, 220, 379-393.	1.5	17
43	Naloxone-precipitated morphine withdrawal evokes phosphorylation of heat shock protein 27 in rat heart through extracellular signal-regulated kinase. Journal of Molecular and Cellular Cardiology, 2011, 51, 129-139.	0.9	16
44	Haloperidol treatment increases the biosynthesis and release of endorphins in guinea-pig ileum. Journal of Pharmacy and Pharmacology, 2011, 36, 446-449.	1.2	13
45	Influence of Dopaminergic and Noradrenergic Systems on the Release of Opioid Peptides in Guinea-pig Ileum. Journal of Pharmacy and Pharmacology, 2011, 41, 607-611.	1.2	6
46	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.	2.7	24
47	Protein kinase C phosphorylates the cAMP response element binding protein in the hypothalamic paraventricular nucleus during morphine withdrawal. British Journal of Pharmacology, 2011, 163, 857-875.	2.7	24
48	Influence of maternal redox status on birth weight. Reproductive Toxicology, 2011, 31, 35-40.	1.3	10
49	Enhanced tyrosine hydroxylase phosphorylation in the nucleus accumbens and nucleus tractus solitarius-A2 cell group after morphine-conditioned place preference. Naunyn-Schmiedeberg's Archives of Pharmacology, 2010, 382, 525-534.	1.4	10
50	Induction of FosB/Î”FosB in the brain stress systemâ€related structures during morphine dependence and withdrawal. Journal of Neurochemistry, 2010, 114, 475-487.	2.1	36
51	Increased Spinal Dynorphin Levels and Phospho-Extracellular Signal-Regulated Kinases 1 and 2 and c-Fos Immunoreactivity after Surgery under Remifentanil Anesthesia in Mice. Molecular Pharmacology, 2010, 77, 185-194.	1.0	31
52	Effects of Corticotropin-Releasing Factor Receptor-1 Antagonists on the Brain Stress System Responses to Morphine Withdrawal. Molecular Pharmacology, 2010, 77, 864-873.	1.0	50
53	Changes in metabolic-related variables during chronic morphine treatment. Neurochemistry International, 2010, 57, 323-330.	1.9	29
54	Cross-Talk between Protein Kinase A and Mitogen-Activated Protein Kinases Signalling in the Adaptive Changes Observed during Morphine Withdrawal in the Heart. Journal of Pharmacology and Experimental Therapeutics, 2009, 330, 771-782.	1.3	4

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55	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.	1.4	41
56	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.	1.7	10
57	Tyrosine hydroxylase phosphorylation after naloxone-induced morphine withdrawal in the left ventricle. Basic Research in Cardiology, 2009, 104, 366-376.	2.5	7
58	Effects of chronic inflammation and morphine tolerance on the expression of phospho-ERK 1/2 and phospho-P38 in the injured tissue. Naunyn-Schmiedeberg's Archives of Pharmacology, 2009, 379, 315-323.	1.4	9
59	Morphine withdrawal regulates phosphorylation of cAMP response element binding protein (CREB) through PKC in the nucleus tractus solitariusâ€A₂ catecholaminergic neurons. Journal of Neurochemistry, 2009, 110, 1422-1432.	2.1	20
60	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.	1.4	19
61	The PKs PKA and ERK 1/2 are involved in phosphorylation of TH at Serine 40 and 31 during morphine withdrawal in rat hearts. British Journal of Pharmacology, 2008, 155, 73-83.	2.7	14
62	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.	1.4	37
63	Activation of the ERK signalling pathway contributes to the adaptive changes in rat hearts during naloxone-induced morphine withdrawal. British Journal of Pharmacology, 2007, 151, 787-797.	2.7	20
64	Activation of stressâ€related hypothalamic neuropeptide gene expression during morphine withdrawal. Journal of Neurochemistry, 2007, 101, 1060-1071.	2.1	54
65	Phosphodiesterase 4 inhibitors, rolipram and diazepam block the adaptive changes observed during morphine withdrawal in the heart. European Journal of Pharmacology, 2007, 570, 1-9.	1.7	11
66	Differential involvement of 3â€², 5â€²-cyclic adenosine monophosphate-dependent protein kinase in regulation of Fos and tyrosine hydroxylase expression in the heart after naloxone induced morphine withdrawal. Naunyn-Schmiedeberg's Archives of Pharmacology, 2007, 374, 293-303.	1.4	4
67	Role of PKC in regulation of Fos and TH expression after naloxone induced morphine withdrawal in the heart. Naunyn-Schmiedeberg's Archives of Pharmacology, 2006, 372, 374-382.	1.4	8
68	Involvement of 3',5'-cyclic adenosine monophosphate-dependent protein kinase in regulation of Fos expression and tyrosine hydroxylase levels during morphine withdrawal in the hypothalamic paraventricular nucleus and medulla oblongata catecholaminergic cell groups. Journal of Neurochemistry, 2005, 92, 246-254.	2.1	10
69	Role of PKC-Î±,Î³ isoforms in regulation of c-Fos and TH expression after naloxone-induced morphine withdrawal in the hypothalamic PVN and medulla oblongata catecholaminergic cell groups. Journal of Neurochemistry, 2005, 95, 1249-1258.	2.1	14
70	Alterations in protein kinase A and different protein kinase C isoforms in the heart during morphine withdrawal. European Journal of Pharmacology, 2005, 522, 9-19.	1.7	11
71	Evidence of involvement of the nNOS and the Îº-opioid receptor in the same intracellular network of the rat periaqueductal gray that controls morphine tolerance and dependence. Molecular Brain Research, 2005, 137, 166-173.	2.5	7
72	Morphine withdrawal-induced c-fos expression in the heart: a peripheral mechanism. European Journal of Pharmacology, 2004, 487, 117-124.	1.7	4

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73	Increase of tyrosine hydroxylase levels and activity during morphine withdrawal in the heart. European Journal of Pharmacology, 2004, 506, 119-128.	1.7	25
74	Changes in c-fos expression in the rat heart during morphine withdrawal. Involvement of ?2-adrenoceptors. Naunyn-Schmiedeberg's Archives of Pharmacology, 2004, 370, 17-25.	1.4	7
75	Implication of the signal transduction pathways in the enhancement of noradrenaline turnover induced by morphine withdrawal in the heart. European Journal of Pharmacology, 2003, 471, 113-119.	1.7	11
76	Quantitative Analysis of Corticotropinâ€Releasing Factor and Arginine Vasopressin mRNA in the Hypothalamus During Chronic Morphine Treatment in Rats: An <i>In Situ</i> Hybridization Study. Journal of Neuroendocrinology, 2003, 15, 586-591.	1.2	7
77	Regulation of tyrosine hydroxylase levels and activity and Fos expression during opioid withdrawal in the hypothalamic PVN and medulla oblongata catecholaminergic cell groups innervating the PVN. European Journal of Neuroscience, 2003, 17, 103-112.	1.2	33
78	Activation of c-fos expression in the heart after morphine but not U-50,488H withdrawal. British Journal of Pharmacology, 2003, 138, 626-633.	2.7	14
79	Effects of U-50,488H and U-50,488H withdrawal on c-fos expression in the rat paraventricular nucleus. Correlation with c-fos in brainstem catecholaminergic neurons. British Journal of Pharmacology, 2003, 138, 1544-1552.	2.7	15
80	Effect of naloxone-precipitated morphine withdrawal on CRH and vasopressin mRNA expression in the rat hypothalamic paraventricular nucleus. Neuroscience Letters, 2002, 334, 58-62.	1.0	14
81	Inhibition of protein kinase C but not protein kinase A attenuates morphine withdrawal excitation of rat hypothalamusâ€“pituitaryâ€“adrenal axis. European Journal of Pharmacology, 2002, 452, 57-66.	1.7	21
82	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.	2.1	50
83	Effects of morphine and morphine withdrawal on brainstem neurons innervating hypothalamic nuclei that control thepituitary-adrenocortical axis in rats. British Journal of Pharmacology, 2002, 136, 67-75.	2.7	51
84	Effects of morphine withdrawal on catecholaminergic neurons on heart right ventricle; implication of dopamine receptors. Canadian Journal of Physiology and Pharmacology, 2001, 79, 885-891.	0.7	1
85	Evidence for a peripheral mechanism in cardiac opioid withdrawal. Naunyn-Schmiedeberg's Archives of Pharmacology, 2001, 364, 193-198.	1.4	18
86	Characterization of the signal transduction pathways mediating morphine withdrawal-stimulated c-fos expression in hypothalamic nuclei. European Journal of Pharmacology, 2001, 430, 59-68.	1.7	6
87	Autoradiographic Evidence of Delta-Opioid Receptor Downregulation after Prenatal Stress in Offspring Rat Brain. Pharmacology, 2000, 60, 13-18.	0.9	16
88	Changes in hypothalamic paraventricular nucleus catecholaminergic activity after acute and chronic morphine administration. European Journal of Pharmacology, 2000, 388, 49-56.	1.7	26
89	Changes in catecholaminergic pathways innervating the rat heart ventricle during morphine dependence. Involvement of Î±1- and Î±2-adrenoceptors. European Journal of Pharmacology, 2000, 397, 311-318.	1.7	21
90	Catecholaminergic activity and 3',5'-cyclic adenosine monophosphate levels in heart right ventricle after naloxone induced withdrawal. Naunyn-Schmiedeberg's Archives of Pharmacology, 2000, 361, 61-66.	1.4	23

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91	Noradrenergic and Dopaminergic Activity in the Hypothalamic Paraventricular Nucleus after Naloxone-Induced Morphine Withdrawal. Neuroendocrinology, 2000, 71, 60-67.	1.2	48
92	Activation of c-fosExpression in Hypothalamic Nuclei by Î¼- and Îº-Receptor Agonists: Correlation with Catecholaminergic Activity in the Hypothalamic Paraventricular Nucleus*. Endocrinology, 2000, 141, 1366-1376.	1.4	46
93	Effects of U-50,488H and U-50,488H withdrawal on catecholaminergic neurons of the rat hypothalamus. Life Sciences, 2000, 66, 803-815.	2.0	14
94	Catecholaminergic activity and 3',5'-cyclic adenosine monophosphate concentrations in the right ventricle after acute and chronic morphine administration in the rat. British Journal of Anaesthesia, 1999, 83, 784-788.	1.5	8
95	Effects of morphine and U-50,488H on neurochemical activity of the hypothalamic noradrenergic neurons and pituitary-adrenal response. Neuropeptides, 1999, 33, 131-135.	0.9	2
96	Effects of U-50,488H withdrawal on catecholaminergic neurones of the rat ventricle. British Journal of Pharmacology, 1998, 124, 1060-1064.	2.7	8
97	Changes in oxytocin content in rat brain during morphine withdrawal. Neuropeptides, 1998, 32, 67-71.	0.9	11
98	Changes in right atrial catecholamine content in naÃ¯ve rats and after naloxone-induced withdrawal. British Journal of Anaesthesia, 1998, 80, 354-359.	1.5	21
99	Effects of acute administration of morphine on right atrial catecholamine content and heart rate in chronically morphine-treated rats. British Journal of Anaesthesia, 1997, 78, 439-441.	1.5	8
100	Serotonergic mechanisms involved in calcitonin potentiation of Îº-opioid receptor-mediated effects on adrenal secretion. European Journal of Pharmacology, 1997, 340, 81-87.	1.7	2
101	Effects of chronic U-50,488H treatment on the isolated right atrium of the rat. Neuropeptides, 1997, 31, 511-515.	0.9	3
102	Changes in hypothalamic oxytocin levels during morphine tolerance. Neuropeptides, 1997, 31, 143-146.	0.9	12
103	Neurochemical activity of noradrenergic neurons and pituitary-adrenal response after naloxone-induced withdrawal: the role of calcium channels. Naunyn-Schmiedeberg's Archives of Pharmacology, 1997, 355, 501-506.	1.4	32
104	Differential regulation of corticotropin-releasing factor and vasopressin in discrete brain regions after morphine administration: correlations with hypothalamic noradrenergic activity and pituitary-adrenal response. Naunyn-Schmiedeberg's Archives of Pharmacology, 1997, 356, 603-610.	1.4	27
105	Effect of chronic administration of dihydropyridine Ca2+ channel ligands on sufentanil-induced tolerance to Î¼- and Îº-opioid agonists in the guinea pig ileum myenteric plexus. Regulatory Peptides, 1996, 63, 1-8.	1.9	8
106	Autoradiographic evidence of Î¼-opioid receptors down-regulation after prenatal stress in offspring rat brain. Developmental Brain Research, 1996, 94, 14-21.	2.1	22
107	Chronic naloxone-induced supersensitivity affects neither tolerance to nor physical dependence on morphine at hypothalamus-pituitary-adrenocortical axis. Neuropeptides, 1996, 30, 29-36.	0.9	7
108	Modulation by catecholamine of hypothalamus-pituitary-adrenocortical (HPA) axis activity in morphine-tolerance and withdrawal. General Pharmacology, 1994, 25, 187-192.	0.7	12

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109	Lack of involvement of Î´-opioid receptor in mediating physical dependence at the hypothalamus-pituitary-adrenocortical (HPA) axis in the rat. General Pharmacology, 1994, 25, 719-723.	0.7	2
110	Catecholaminergic mediation of morphine-induced activation of pituitary-adrenocortical axis in the rat: implication of Î±- and Î²-adrenoceptors. Brain Research, 1994, 668, 122-128.	1.1	35
111	Effects of intracerebroventricular clonidine on the hypothalamic noradrenaline and plasma corticosterone levels of opiate naive rats and after naloxone-induced withdrawal. Brain Research, 1994, 647, 199-203.	1.1	35
112	Involvement of Îº-opioid receptor mechanisms in the calcitonin-induced potentiation of opioid effects at the hypothalamus-pituitary-adrenocortical axis. European Journal of Pharmacology, 1994, 271, 103-109.	1.7	7
113	Simultaneous changes in hypothalamic catecholamine levels and plasma corticosterone concentration in the rat after acute morphine and during tolerance. Neuropeptides, 1993, 24, 279-284.	0.9	31
114	L-type Ca2+ channel ligands modulate morphine effects on the hypothalamus-pituitary-adrenocortical axis in rats. European Journal of Pharmacology, 1993, 232, 191-198.	1.7	26
115	The Î²-endorphin response to prenatal stress during postnatal development in the rat. Developmental Brain Research, 1993, 74, 142-145.	2.1	19
116	Effects of Clonidine on Pituitary- Adrenocortical Axis in Morphine-Tolerant Rats and after Naloxone-Induced Withdrawal. Pharmacology, 1992, 44, 158-164.	0.9	10
117	Influence of temperature on the effects of Î¼-, Î´- and Îº-opioid receptor agonists in the guinea-pig ileum myenteric plexus. European Journal of Pharmacology, 1992, 223, 19-23.	1.7	5
118	Prenatal stress alters the hypothalamic levels of methionine-enkephalin in pup rats. Neuropeptides, 1992, 23, 131-135.	0.9	14
119	Influence of stress in the effects of morphine on pituitary-adrenocortical activity in the guinea-pig. General Pharmacology, 1991, 22, 223-226.	0.7	3
120	Effects of acute and chronic administration of Î¼- and Î´-opeoid agonists on the hypothalamic-pituitary-adrenocortical (HPA) axis in the rat. European Journal of Pharmacology, 1991, 200, 155-158.	1.7	39
121	Pituitary-adrenocortical response to acute and chronic administration of U-50 488H in the rat. Neuropeptides, 1991, 20, 95-102.	0.9	30
122	Effects of morphine on plasma B-endorphin and cortisol levels and on body temperature in guinea-pigs pretreated with 6-hydroxydopamine. General Pharmacology, 1990, 21, 799-803.	0.7	2
123	Stress and morphine mediated changes in pituitary-adrenal axis in guinea-pigs. General Pharmacology, 1990, 21, 569-571.	0.7	6
124	Effects of U-50,488H on plasma levels of B-endorphin and cortisol in the rat. European Journal of Pharmacology, 1990, 183, 2332-2333.	1.7	1
125	Plasma Î²-endorphin and cortisol levels in morphine-tolerant rats and in naloxone-induced withdrawal. European Journal of Pharmacology, 1990, 182, 117-123.	1.7	48
126	Effect of selective monoamine oxidase inhibitors on the morphine-induced hypothermia in restrained rats. General Pharmacology, 1987, 18, 185-188.	0.7	6

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127	Effects of droperidol on the biosynthesis and release of endogenous opioid peptides in guinea-pig ileum. General Pharmacology, 1987, 18, 283-286.	0.7	5
128	Pharmacological evidence for the existence of interactions between dopaminergic and opioid peptidergic systems in guinea-pig ileum myenteric plexus. European Journal of Pharmacology, 1986, 128, 259-263.	1.7	6
129	Effect of ACTH-like peptides on morphine-induced hypothermia in unrestrained guinea pigs. Brain Research, 1986, 375, 13-19.	1.1	10
130	Effects of morphine and their antagonism by dexamethasone on body temperature in restrained and unrestrained guinea-pigs. Pharmacological Research Communications, 1985, 17, 385-394.	0.2	6
131	Effects of chronic treatment with atypical neuroleptics on the biosynthesis and release of opioid peptides in guinea-pig ileum. Regulatory Peptides, 1985, 10, 319-327.	1.9	5
132	Possible mechanisms implicated on the hypothermic effect induced by morphine in guinea-pig. General Pharmacology, 1984, 15, 357-360.	0.7	6