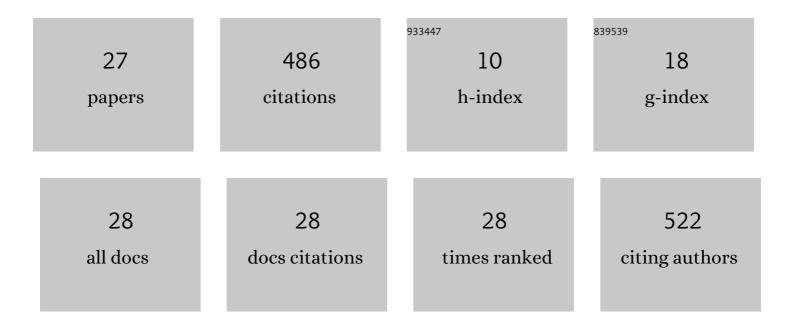
Isabel Martins

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Pain Modulation from the Locus Coeruleus in a Model of Hydrocephalus: Searching for Oxidative Stress-Induced Noradrenergic Neuroprotection. International Journal of Molecular Sciences, 2022, 23, 3970.	4.1	1
2	Monoaminergic and Opioidergic Modulation of Brainstem Circuits: New Insights Into the Clinical Challenges of Pain Treatment?. Frontiers in Pain Research, 2021, 2, 696515.	2.0	10
3	Serotoninergic pain modulation from the rostral ventromedial medulla (RVM) in chemotherapyâ€induced neuropathy: The role of spinal 5â€HT3 receptors. European Journal of Neuroscience, 2020, 51, 1756-1769.	2.6	25
4	Attenuation of the Diffuse Noxious Inhibitory Controls in Chronic Joint Inflammatory Pain Is Accompanied by Anxiodepressive-Like Behaviors and Impairment of the Descending Noradrenergic Modulation. International Journal of Molecular Sciences, 2020, 21, 2973.	4.1	10
5	Shift of µ-opioid Receptor Signaling in the Dorsal Reticular Nucleus Is Implicated in Morphine-induced Hyperalgesia in Male Rats. Anesthesiology, 2020, 133, 628-644.	2.5	9
6	Neuropathic Pain Induced Alterations in the Opioidergic Modulation of a Descending Pain Facilitatory Area of the Brain. Frontiers in Cellular Neuroscience, 2019, 13, 287.	3.7	9
7	Chronic opioid administration results in μ-opioid receptor excitatory signaling at a descending pain facilitatory area of the brain. IBRO Reports, 2019, 6, S292-S293.	0.3	0
8	Role of Spinal Cord α2-Adrenoreceptors in Noradrenergic Inhibition of Nociceptive Transmission During Chemotherapy-Induced Peripheral Neuropathy. Frontiers in Neuroscience, 2019, 13, 1413.	2.8	17
9	Paclitaxel-induced neuropathic pain: Unravelling the underlying mechanisms at the central nervous system. Porto Biomedical Journal, 2017, 2, 192.	1.0	0
10	Reticular Formation and Pain: The Past and the Future. Frontiers in Neuroanatomy, 2017, 11, 51.	1.7	71
11	Increased Noradrenergic Neurotransmission to a Pain Facilitatory Area of the Brain Is Implicated in Facilitation of Chronic Pain. Anesthesiology, 2015, 123, 642-653.	2.5	32
12	GABA acting on GABAB receptors located in a medullary pain facilitatory area enhances nociceptive behaviors evoked by intraplantar formalin injection. Pain, 2015, 156, 1555-1565.	4.2	12
13	Gene Therapy for Chronic Pain: How to Manipulate and Unravel Pain Control Circuits from the Brain?. Neuromethods, 2015, , 321-339.	0.3	0
14	Noradrenaline increases pain facilitation from the brain during inflammatory pain. Neuropharmacology, 2013, 71, 299-307.	4.1	28
15	Decrease in the expression of Nâ€methylâ€Dâ€aspartate receptors in the nucleus tractus solitarii induces antinociception and increases blood pressure. Journal of Neuroscience Research, 2012, 90, 356-366.	2.9	6
16	Reversal of inflammatory pain by HSVâ€1â€mediated overexpression of enkephalin in the caudal ventrolateral medulla. European Journal of Pain, 2011, 15, 1008-1014.	2.8	11
17	Inhibition of nociceptive responses after systemic administration of amidated kyotorphin. British Journal of Pharmacology, 2011, 163, 964-973.	5.4	25
18	Reversal of neuropathic pain by HSV-1-mediated decrease of noradrenaline in a pain facilitatory area of the brain. Pain, 2010, 151, 137-145.	4.2	40

#	Article	IF	CITATIONS
19	203 ANTINOCICEPTION PRODUCED BY VIRALâ€DRIVEN OVEREXPRESSION OF PREPROENKEPHALIN IN THE CAUDAL VENTROLATERAL MEDULLA. European Journal of Pain, 2009, 13, S67.	2.8	0
20	Dynamic of migration of HSVâ€1 from a medullary pronociceptive centre: antinociception by overexpression of the preproenkephalin transgene. European Journal of Neuroscience, 2008, 28, 2075-2083.	2.6	22
21	283 NEUROPATHIC PAIN IS ATTENUATED BY A VIRAL VECTOR TARGETING NORADRENERGIC INPUT TO THE DORSAL RETICULAR NUCLEUS. European Journal of Pain, 2007, 11, S125-S126.	2.8	2
22	Cardiovascular abnormalities with normal blood pressure in tissue kallikrein-deficient mice. Proceedings of the National Academy of Sciences of the United States of America, 2001, 98, 2634-2639.	7.1	155
23	Gene Therapy for Chronic Pain Management. , 0, , .		0
24	Descending serotonergic pain modulation during chemotherapy-induced neuropathy: the role of spinal 5-HT3 receptor. Frontiers in Cellular Neuroscience, 0, 13, .	3.7	0
25	"The descending noradrenergic spinal modulation is impaired during prolonged joint inflammatory pain conditions.― Frontiers in Cellular Neuroscience, 0, 13, .	3.7	0
26	Chronic morphine enhances descending pain facilitation from the brain through a switch of µ-opioid receptor signaling. Frontiers in Cellular Neuroscience, 0, 13, .	3.7	0
27	Mu-opioid receptor signalling switch to excitatory following chronic morphine persists upon treatment cessation. Frontiers in Cellular Neuroscience, 0, 13, .	3.7	1