Cibele M. Prado

List of Publications by Year
in descending order

57	1,400	279487	329751
papers	citations	23 h-index	37 g-index

57	57	57	2051
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Prospective analysis of myocardial strain through the evolution of Chagas disease in the hamster animal model. International Journal of Cardiovascular Imaging, 2022, 38, 117-129.	0.7	2
2	Changes in Oviductal Cells and Small Extracellular Vesicles miRNAs in Pregnant Cows. Frontiers in Veterinary Science, 2021, 8, 639752.	0.9	19
3	Rubia Gallega x Nelore crossbred cattle improve beef tenderness through changes in protein abundance and gene expression. Livestock Science, 2021, 251, 104634.	0.6	0
4	Reversion of cardiovascular remodelling in renovascular hypertensive 2Kâ€1C rats by reninâ€“angiotensin system inhibitors. Clinical and Experimental Pharmacology and Physiology, 2020, 47, 1965-1977.	0.9	5
5	The comparative efficacy of renin-angiotensin system blockers in schistosomal hepatic fibrosis. Experimental Parasitology, 2018, 191, 9-18.	0.5	3
6	Elastaseâ€2, an angiotensin Iâ€generating enzyme, contributes to increased angiotensin II in resistance arteries of mice with myocardial infarction. British Journal of Pharmacology, 2017, 174, 1104-1115.	2.7	12
7	Dantrolene improves in vitro structural changes induced by serum from Trypanosoma cruzi-infected mice. Parasitology Research, 2017, 116, 429-433.	0.6	3
8	Elastase-2, a Tissue Alternative Pathway for Angiotensin II Generation, Plays a Role in Circulatory Sympathovagal Balance in Mice. Frontiers in Physiology, 2017, 8, 170.	1.3	7
9	Early dystrophin loss is coincident with the transition of compensated cardiac hypertrophy to heart failure. PLoS ONE, 2017, 12, e0189469.	1.1	11
10	Reduced expression of adherens and gap junction proteins can have a fundamental role in the development of heart failure following cardiac hypertrophy in rats. Experimental and Molecular Pathology, 2016, 100, 167-176.	0.9	16
11	Erectile dysfunction in heart failure rats is associated with increased neurogenic contractions in cavernous tissue and internal pudendal artery. Life Sciences, 2016, 145, 9-18.	2.0	14
12	Early neonatal echocardiographic findings in an experimental rabbit model of congenital diaphragmatic hernia. Brazilian Journal of Medical and Biological Research, 2015, 48, 234-239.	0.7	5
13	Pyridostigmine prevents haemodynamic alterations but does not affect their nycthemeral oscillations in infarcted mice. Autonomic Neuroscience: Basic and Clinical, 2015, 187, 50-55.	1.4	8
14	Acetylcholinesterase Inhibition Attenuates the Development of Hypertension and Inflammation in Spontaneously Hypertensive Rats. American Journal of Hypertension, 2015, 28, 1201-1208.	1.0	52
15	Pyridostigmine Restores Cardiac Autonomic Balance after Small Myocardial Infarction in Mice. PLoS ONE, 2014, 9, e104476.	1.1	29
16	Immobilization and therapeutic passive stretching generate thickening and increase the expression of laminin and dystrophin in skeletal muscle. Brazilian Journal of Medical and Biological Research, 2014, 47, 483-491.	0.7	3
17	Role of dystrophin in acute Trypanosoma cruzi infection. Microbes and Infection, 2014, 16, 768-777.	1.0	4
18	Î²1-Adrenergic blockers exert antioxidant effects, reduce matrix metalloproteinase activity, and improve renovascular hypertension-induced cardiac hypertrophy. Free Radical Biology and Medicine, 2014, 73, 308-317.	1.3	37

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19	Abstract 421: Alterations in Adherens Junction and Gap Junction Precede Desmosomes Remodeling During the Transition from Experimental Compensated Cardiac Hypertrophy to Decompensation. Hypertension, 2014, 64, .	1.3	0
20	Tempol inhibits TGF-Î² and MMPs upregulation and prevents cardiac hypertensive changes. International Journal of Cardiology, 2013, 165, 165-173.	0.8	45
21	Remobilization through stretching improves gait recovery in the rat. Acta Histochemica, 2013, 115, 460-469.	0.9	15
22	Temporal changes in cardiac matrix metalloproteinase activity, oxidative stress, and TGF-Î² in renovascular hypertension-induced cardiac hypertrophy. Experimental and Molecular Pathology, 2013, 94, 1-9.	0.9	51
23	Increase in parasympathetic tone by pyridostigmine prevents ventricular dysfunction during the onset of heart failure. American Journal of Physiology - Regulatory Integrative and Comparative Physiology, 2013, 305, R908-R916.	0.9	62
24	Sepsis: Going to the Heart of the Matter. Pathobiology, 2013, 80, 70-86.	1.9	78
25	Disruption of Calcium Homeostasis in Cardiomyocytes Underlies Cardiac Structural and Functional Changes in Severe Sepsis. PLoS ONE, 2013, 8, e68809.	1.1	47
26	Are adherens junctions implicated in the transition from physiological to pathological cardiac hypertrophy?. FASEB Journal, 2013, 27, 745.6.	0.2	0
27	Chagas Heart Disease. Cardiology in Review, 2012, 20, 53-65.	0.6	90
28	Early dystrophin disruption in the pathogenesis of experimental chronic Chagas cardiomyopathy. Microbes and Infection, 2012, 14, 59-68.	1.0	9
29	Proinflamatory cytokines affect dystrophin expression in cultured newborn cardiomyocytes under different stimuli. FASEB Journal, 2012, 26, 1036.2.	0.2	0
30	Mice and rats have different responses to abdominal aorta constriction. FASEB Journal, 2012, 26, 726.4.	0.2	0
31	Calpainâ€mediated dystrophin disruption may be a potential structural culprit behind chronic doxorubicinâ€induced cardiomyopathy. FASEB Journal, 2012, 26, 1036.1.	0.2	0
32	The Vasculature in Chagas Disease. Advances in Parasitology, 2011, 76, 83-99.	1.4	27
33	Combining two potential causes of metalloproteinase secretion causes abdominal aortic aneurysms in rats: a new experimental model. International Journal of Experimental Pathology, 2011, 92, 26-39.	0.6	25
34	Calpain-mediated dystrophin disruption may be a potential structural culprit behind chronic doxorubicin-induced cardiomyopathy. European Journal of Pharmacology, 2011, 670, 541-553.	1.7	32
35	Aspirin Treatment of Mice Infected with Trypanosoma cruzi and Implications for the Pathogenesis of Chagas Disease. PLoS ONE, 2011, 6, e16959.	1.1	59
36	Cardiac mast cell proteases do not contribute to the regulation of the rat coronary vascular responsiveness to arterial delivered angiotensin I and II. Vascular Pharmacology, 2010, 53, 22-27.	1.0	7

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37	5-Lipoxygenase is a key determinant of acute myocardial inflammation and mortality during Trypanosoma cruzi infection. Microbes and Infection, 2010, 12, 587-597.	1.0	38
38	Disruption of sarcolemmal dystrophin and Î²-dystroglycan may be a potential mechanism for myocardial dysfunction in severe sepsis. Laboratory Investigation, 2010, 90, 531-542.	1.7	26
39	Coronary Microvascular Disease in Chronic Chagas Cardiomyopathy Including an Overview on History, Pathology, and Other Proposed Pathogenic Mechanisms. PLoS Neglected Tropical Diseases, 2010, 4, e674.	1.3	89
40	INCREASED SARCOLEMMAL PERMEABILITY AS AN EARLY EVENT IN EXPERIMENTAL SEPTIC CARDIOMYOPATHY. Shock, 2010, 33, 322-331.	1.0	33
41	Imbalance between matrix metalloproteinases and tissue inhibitor of metalloproteinases in hypertensive vascular remodeling. Matrix Biology, 2010, 29, 194-201.	1.5	100
42	Matrix Metalloproteinase Inhibition Improves Cardiac Dysfunction and Remodeling in 2-Kidney, 1-Clip Hypertension. Journal of Cardiac Failure, 2010, 16, 599-608.	0.7	67
43	Micro-Positron Emission Tomography in the Evaluation of Trypanosoma cruzi-Induced Heart Disease: Comparison with Other Modalities. American Journal of Tropical Medicine and Hygiene, 2009, 81, 900-905.	0.6	21
44	Turbulent blood flow plays an essential localizing role in the development of atherosclerotic lesions in experimentally induced hypercholesterolaemia in rats. International Journal of Experimental Pathology, 2008, 89, 72-80.	0.6	22
45	Isoproterenol induces primary loss of dystrophin in rat hearts: correlation with myocardial injury. International Journal of Experimental Pathology, 2008, 89, 367-381.	0.6	22
46	Myocardial structural changes in long-term human severe sepsis/septic shock may be responsible for cardiac dysfunction. Heart Lung and Circulation, 2008, 17, S37.	0.2	0
47	Changes in hemodynamic and neurohumoral control cause cardiac damage in one-kidney, one-clip hypertensive mice. American Journal of Physiology - Regulatory Integrative and Comparative Physiology, 2008, 295, R1904-R1913.	0.9	10
48	Glycol methacrylate-embedding medium to study morphological alterations of saphenous vein under brief and crescent pressurizations. Acta Cirurgica Brasileira, 2008, 23, 77-82.	0.3	2
49	Arterial pressure variability, baroreflex sensitivity and organ damage in one clip, one kidney (1K1C) hypertensive conscious mice. FASEB Journal, 2008, 22, 968.17.	0.2	0
50	Aorta Remodeling Responses to Distinct Atherogenic Stimuli: Hyperten-sion, Hypercholesterolemia and Turbulent Flow/Low Wall Shear Stress. Open Cardiovascular Medicine Journal, 2008, 2, 41-48.	0.6	4
51	Cardiovascular risk factors: can long-term alcohol withdrawal benefit heavy drinkers?. Journal of Hypertension, 2007, 25, 285-288.	0.3	0
52	MYOCARDIAL STRUCTURAL CHANGES IN LONG-TERM HUMAN SEVERE SEPSIS/SEPTIC SHOCK MAY BE RESPONSIBLE FOR CARDIAC DYSFUNCTION. Shock, 2007, 27, 10-18.	1.0	92
53	L 003 TURBULENT BLOOD FLOW PLAYS AN ESSENTIAL LOCALIZING ROLE IN THE DEVELOPMENT OF ATHEROSCLEROTIC PLAQUES IN EXPERIMENTALLY-INDUCED HYPERCHOLESTEROLEMIA IN RATS. Atherosclerosis Supplements, 2007, 8, 17.	1.2	0
54	Turbulent flow/low wall shear stress and stretch differentially affect aorta remodeling in rats. Journal of Hypertension, 2006, 24, 503-515.	0.3	38

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55	Circumferential wall tension due to hypertension plays a pivotal role in aorta remodelling. International Journal of Experimental Pathology, 2006, 87, 425-436.	0.6	36
56	Aorta Remodeling Due to Stretch or Turbulent Flow/Low Wall Shear Stress: Distinctly Different Biological Phenomena?. Current Cardiology Reviews, 2006, 2, 109-117.	0.6	0
57	Chronic inhibition of nitric oxide synthase induces hypertension and cardiomyocyte mitochondrial and myocardial collagen remodelling in the absence of hypertrophy. Journal of Hypertension, 2003, 21, 993-1001.	0.3	23