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List of Publications by Year
in descending order

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47 papers	4,283 citations	186265 28 h-index	214800 47 g-index
50 all docs	50 docs citations	50 times ranked	5863 citing authors

#	Article	IF	Citations
1	Kidney inflammaging is promoted by CCR2+â€‰macrophages and tissue-derived micro-environmental factors. Cellular and Molecular Life Sciences, 2021, 78, 3485-3501.	5.4	13
2	Selective Cardiomyocyte Oxidative Stress Leads to Bystander Senescence of Cardiac Stromal Cells. International Journal of Molecular Sciences, 2021, 22, 2245.	4.1	7
3	Monoamine oxidases in age-associated diseases: New perspectives for old enzymes. Ageing Research Reviews, 2021, 66, 101256.	10.9	44
4	Cyclic AMP-binding protein Epac1 acts as a metabolic sensor to promote cardiomyocyte lipotoxicity. Cell Death and Disease, 2021, 12, 824.	6.3	12
5	Guidelines for the use and interpretation of assays for monitoring autophagy (4th) Tj ETQq1 1 0.784314 rgBT /Overlock 10 Tf 50 582	9.1	1,430
6	Mitochondrial 4-HNE derived from MAO-A promotes mitoCa2+ overload in chronic postischemic cardiac remodeling. Cell Death and Differentiation, 2020, 27, 1907-1923.	11.2	51
7	Role of EPAC1 Signalosomes in Cell Fate: Friends or Foes?. Cells, 2020, 9, 1954.	4.1	7
8	Cellular Senescence in Renal and Urinary Tract Disorders. Cells, 2020, 9, 2420.	4.1	7
9	Rational Redesign of Monoamine Oxidase A into a Dehydrogenase to Probe ROS in Cardiac Aging. ACS Chemical Biology, 2020, 15, 1795-1800.	3.4	12
10	Cardiac monoamine oxidases: at the heart of mitochondrial dysfunction. Cell Death and Disease, 2020, 11, 54.	6.3	10
11	Clearance of senescent cells during cardiac ischemiaâ€“reperfusion injury improves recovery. Aging Cell, 2020, 19, e13249.	6.7	79
12	Aging induces cardiac mesenchymal stromal cell senescence and promotes endothelial cell fate of the CD90Â+Âsubset. Aging Cell, 2019, 18, e13015.	6.7	31
13	Identification of a pharmacological inhibitor of Epac1 that protects the heart against acute and chronic models of cardiac stress. Cardiovascular Research, 2019, 115, 1766-1777.	3.8	25
14	Lengthâ€independent telomere damage drives postâ€mitotic cardiomyocyte senescence. EMBO Journal, 2019, 38, .	7.8	307
15	Body fat reduction without cardiovascular changes in mice after oral treatment with the <scp>MAO</scp> inhibitor phenelzine. British Journal of Pharmacology, 2018, 175, 2428-2440.	5.4	18
16	Tight-Binding Inhibition of Human Monoamine Oxidase B by Chromone Analogs: A Kinetic, Crystallographic, and Biological Analysis. Journal of Medicinal Chemistry, 2018, 61, 4203-4212.	6.4	58
17	Oleuropein Aglycone Protects against MAO-A-Induced Autophagy Impairment and Cardiomyocyte Death through Activation of TFEB. Oxidative Medicine and Cellular Longevity, 2018, 2018, 1-13.	4.0	35
18	Monoamine oxidase-A, serotonin and norepinephrine: synergistic players in cardiac physiology and pathology. Journal of Neural Transmission, 2018, 125, 1627-1634.	2.8	32

#	Article	IF	Citations
19	Monoamine oxidaseâ€A is a novel driver of stressâ€induced premature senescence through inhibition of parkinâ€mediated mitophagy. Aging Cell, 2018, 17, e12811.	6.7	78
20	Multifunctional Mitochondrial Epac1 Controls Myocardial Cell Death. Circulation Research, 2017, 120, 645-657.	4.5	81
21	La Â«ÂdissectionÂÂ» molÃ©culaire du remodelage cardiaqueÂ: perspectives thÃ©rapeutiques. Archives Des Maladies Du Coeur Et Des Vaisseaux - Pratique, 2017, 2017, 18-21.	0.0	0
22	Major depression and heart failure: Interest of monoamine oxidase inhibitors. International Journal of Cardiology, 2017, 247, 1-6.	1.7	26
23	Autophagy in health and disease: focus on the cardiovascular system. Essays in Biochemistry, 2017, 61, 721-732.	4.7	123
24	Monoamine Oxidases, Oxidative Stress, and Altered Mitochondrial Dynamics in Cardiac Ageing. Oxidative Medicine and Cellular Longevity, 2017, 2017, 1-8.	4.0	76
25	High intake of dietary tyramine does not deteriorate glucose handling and does not cause adverse cardiovascular effects in mice. Journal of Physiology and Biochemistry, 2016, 72, 539-553.	3.0	6
26	Oxidative Stress by Monoamine Oxidase-A Impairs Transcription Factor EB Activation and Autophagosome Clearance, Leading to Cardiomyocyte Necrosis and Heart Failure. Antioxidants and Redox Signaling, 2016, 25, 10-27.	5.4	76
27	Platelet activation and arterial peripheral serotonin turnover in cardiac remodeling associated to aortic stenosis. American Journal of Hematology, 2015, 90, 15-19.	4.1	26
28	Gadd45<i>Î³</i>regulates cardiomyocyte death and post-myocardial infarction left ventricular remodelling. Cardiovascular Research, 2015, 108, 254-267.	3.8	39
29	Monoamine oxidases as sources of oxidants in the heart. Journal of Molecular and Cellular Cardiology, 2014, 73, 34-42.	1.9	197
30	Role of serotonin 5-HT2A receptors in the development of cardiac hypertrophy in response to aortic constriction in mice. Journal of Neural Transmission, 2013, 120, 927-935.	2.8	31
31	Anesthetic regimen for cardiac function evaluation by echocardiography in mice: comparison between ketamine, etomidate and isoflurane versus conscious state. Laboratory Animals, 2013, 47, 284-290.	1.0	29
32	First Evidence of Increased Plasma Serotonin Levels in Tako-Tsubo Cardiomyopathy. BioMed Research International, 2013, 2013, 1-5.	1.9	9
33	p53-PGC-1Î± Pathway Mediates Oxidative Mitochondrial Damage and Cardiomyocyte Necrosis Induced by Monoamine Oxidase-A Upregulation: Role in Chronic Left Ventricular Dysfunction in Mice. Antioxidants and Redox Signaling, 2013, 18, 5-18.	5.4	117
34	Serotonin 5-HT2A receptor-mediated hypertrophy is negatively regulated by caveolin-3 in cardiomyoblasts and neonatal cardiomyocytes. Journal of Molecular and Cellular Cardiology, 2012, 52, 502-510.	1.9	21
35	Essential role of TRPC1 channels in cardiomyoblasts hypertrophy mediated by 5-HT2A serotonin receptors. Biochemical and Biophysical Research Communications, 2010, 391, 979-983.	2.1	39
36	Dose-dependent activation of distinct hypertrophic pathways by serotonin in cardiac cells. American Journal of Physiology - Heart and Circulatory Physiology, 2009, 297, H821-H828.	3.2	24

#	ARTICLE	IF	CITATIONS
37	Genetic deletion of MAO-A promotes serotonin-dependent ventricular hypertrophy by pressure overload. Journal of Molecular and Cellular Cardiology, 2009, 46, 587-595.	1.9	41
38	Platelet derived serotonin drives the activation of rat cardiac fibroblasts by 5-HT2A receptors. Journal of Molecular and Cellular Cardiology, 2009, 46, 518-525.	1.9	76
39	Genetic Variation Within the Î²1-Adrenergic Receptor Gene Results in Haplotype-Specific Expression Phenotypes. Journal of Cardiovascular Pharmacology, 2008, 51, 106-110.	1.9	17
40	Genetic Variation of Human Adrenergic Receptors: From Molecular and Functional Properties to Clinical and Pharmacogenetic Implications. Current Topics in Medicinal Chemistry, 2007, 7, 217-231.	2.1	21
41	New insights on receptor-dependent and monoamine oxidase-dependent effects of serotonin in the heart. Journal of Neural Transmission, 2007, 114, 823-827.	2.8	33
42	Myocardial Î²1-adrenergic receptor polymorphisms affect functional recovery after ischemic injury. American Journal of Physiology - Heart and Circulatory Physiology, 2006, 290, H1427-H1432.	3.2	34
43	A polymorphism within a conserved beta1-adrenergic receptor motif alters cardiac function and beta-blocker response in human heart failure. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103, 11288-11293.	7.1	435
44	Differential functional effects of two 5-HT receptor isoforms in adult cardiomyocytes. Journal of Molecular and Cellular Cardiology, 2005, 39, 335-344.	1.9	24
45	Polymorphisms of cardiac presynaptic Â2C adrenergic receptors: Diverse intragenic variability with haplotype-specific functional effects. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 13020-13025.	7.1	51
46	A Primate-dominant Third Glycosylation Site of the Î²2-Adrenergic Receptor Routes Receptors to Degradation during Agonist Regulation. Journal of Biological Chemistry, 2004, 279, 38603-38607.	3.4	42
47	Î²1-adrenergic receptor polymorphisms confer differential function and predisposition to heart failure. Nature Medicine, 2003, 9, 1300-1305.	30.7	328