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List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Cobalt oxide nanoparticles induce oxidative stress and alter electromechanical function in rat ventricular myocytes. Particle and Fibre Toxicology, 2021, 18, 1.	6.2	21
2	Elevated miR-34a expression and altered transcriptional profile are associated with adverse electromechanical remodeling in the heart of male rats exposed to social stress. Stress, 2021, 24, 621-634.	1.8	6
3	Effects of Standardized Green Tea Extract and Its Main Component, EGCG, on Mitochondrial Function and Contractile Performance of Healthy Rat Cardiomyocytes. Nutrients, 2020, 12, 2949.	4.1	6
4	Blockade of Oncogenic NOTCH1 with the SERCA Inhibitor CAD204520 in T Cell Acute Lymphoblastic Leukemia. Cell Chemical Biology, 2020, 27, 678-697.e13.	5.2	27
5	Subchronic exposure to titanium dioxide nanoparticles modifies cardiac structure and performance in spontaneously hypertensive rats. Particle and Fibre Toxicology, 2019, 16, 25.	6.2	32
6	5-(Hydroxyphenyl)-Î ³ -Valerolactone-Sulfate, a Key Microbial Metabolite of Flavan-3-ols, Is Able to Reach the Brain: Evidence from Different in Silico, In Vitro and In Vivo Experimental Models. Nutrients, 2019, 11, 2678.	4.1	55
7	The Histone Deacetylase Inhibitor Suberoylanilide Hydroxamic Acid (SAHA) Restores Cardiomyocyte Contractility in a Rat Model of Early Diabetes. International Journal of Molecular Sciences, 2019, 20, 1873.	4.1	15
8	Targeting the Activating Mutations of NOTCH1 in T-Cell Lymphoblastic Leukemia with a New SERCA Inhibitor CAD204520. Blood, 2019, 134, 407-407.	1.4	0
9	HDAC Inhibition Improves the Sarcoendoplasmic Reticulum Ca2+-ATPase Activity in Cardiac Myocytes. International Journal of Molecular Sciences, 2018, 19, 419.	4.1	21
10	Trimethylamine-N-Oxide (TMAO)-Induced Impairment of Cardiomyocyte Function and the Protective Role of Urolithin B-Glucuronide. Molecules, 2018, 23, 549.	3.8	71
11	Long-Term Oral Administration of Theaphenon-E Improves Cardiomyocyte Mechanics and Calcium Dynamics by Affecting Phospholamban Phosphorylation and ATP Production. Cellular Physiology and Biochemistry, 2018, 47, 1230-1243.	1.6	12
12	In vivo administration of urolithin A and B prevents the occurrence of cardiac dysfunction in streptozotocin-induced diabetic rats. Cardiovascular Diabetology, 2017, 16, 80.	6.8	99
13	Parenchymal and Stromal Cells Contribute to Pro-Inflammatory Myocardial Environment at Early Stages of Diabetes: Protective Role of Resveratrol. Nutrients, 2016, 8, 729.	4.1	14
14	Antiarrhythmic effect of growth factor-supplemented cardiac progenitor cells in chronic infarcted heart. American Journal of Physiology - Heart and Circulatory Physiology, 2016, 310, H1622-H1648.	3.2	23
15	Enhanced engraftment and repairing ability of human adiposeâ€derived stem cells, conveyed by pharmacologically active microcarriers continuously releasing <scp>HGF</scp> and <scp>IGF</scp> â€1, in healing myocardial infarction in rats. Journal of Biomedical Materials Research - Part A, 2015, 103, 3012-3025.	4.0	37
16	Urolithins at physiological concentrations affect the levels of pro-inflammatory cytokines and growth factor in cultured cardiac cells in hyperglucidic conditions. Journal of Functional Foods, 2015, 15, 97-105.	3.4	49
17	Titanium dioxide nanoparticles promote arrhythmias via a direct interaction with rat cardiac tissue. Particle and Fibre Toxicology, 2014, 11, 63.	6.2	76
18	Resveratrol Treatment Reduces Cardiac Progenitor Cell Dysfunction and Prevents Morpho-Functional Ventricular Remodeling in Type-1 Diabetic Rats. PLoS ONE, 2012, 7, e39836.	2.5	63

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19	Growth Factor-Induced Mobilization of Cardiac Progenitor Cells Reduces the Risk of Arrhythmias, in a Rat Model of Chronic Myocardial Infarction. PLoS ONE, 2011, 6, e17750.	2.5	31
20	N ^ε -lysine acetylation determines dissociation from GAP junctions and lateralization of connexin 43 in normal and dystrophic heart. Proceedings of the National Academy of Sciences of the United States of America, 2011, 108, 2795-2800.	7.1	93
21	The histone deacetylase inhibitor suberoylanilide hydroxamic acid reduces cardiac arrhythmias in dystrophic mice. Cardiovascular Research, 2010, 87, 73-82.	3.8	43
22	Modulation of actin isoform expression before the transition from experimental compensated pressure-overload cardiac hypertrophy to decompensation. American Journal of Physiology - Heart and Circulatory Physiology, 2009, 296, H1625-H1632.	3.2	24
23	Preservation of ventricular performance at early stages of diabetic cardiomyopathy involves changes in myocyte size, number and intercellular coupling. Basic Research in Cardiology, 2007, 102, 488-499.	5.9	30
24	Effects of the ??2-Adrenergic/DA2-Dopaminergic Agonist CHF-1024 in Preventing Ventricular Arrhythmogenesis and Myocyte Electrical Remodeling, in a Rat Model of Pressure-Overload Cardiac Hypertrophy. Journal of Cardiovascular Pharmacology, 2006, 47, 295-302.	1.9	6
25	Correlation of α-skeletal actin expression, ventricular fibrosis and heart function with the degree of pressure overload cardiac hypertrophy in rats. Experimental Physiology, 2006, 91, 571-580.	2.0	36
26	Behavioural, neural and cardiovascular adaptations in mice lacking the NPY Y1 receptor. Neuroscience and Biobehavioral Reviews, 2005, 29, 113-123.	6.1	24
27	Vulnerability to ventricular arrhthmias and heterogeneity of action potential duration in normal rats. Experimental Physiology, 2004, 89, 387-396.	2.0	6
28	Effects of chronic psychosocial stress on cardiac autonomic responsiveness and myocardial structure in mice. American Journal of Physiology - Heart and Circulatory Physiology, 2004, 286, H2133-H2140.	3.2	55
29	Intermittent Exposure to Social Defeat and Open-field Test in Rats: Acute and Long-term Effects on ECG, Body Temperature and Physical Activity. Stress, 2002, 5, 23-35.	1.8	58
30	Social stress, myocardial damage and arrhythmias in rats with cardiac hypertrophy. Physiology and Behavior, 2001, 73, 351-358.	2.1	9
31	Cardiac autonomic responses to intermittent social conflict in rats. Physiology and Behavior, 2001, 73, 343-349.	2.1	43
32	Myocardial remodeling and arrhythmogenesis in moderate cardiac hypertrophy in rats. American Journal of Physiology - Heart and Circulatory Physiology, 2001, 280, H142-H150.	3.2	44
33	Acute social stress and cardiac electrical activity in rats. Aggressive Behavior, 1998, 24, 287-296.	2.4	30
34	High-density epicardial mapping during current injection and ventricular activation in rat hearts. American Journal of Physiology - Heart and Circulatory Physiology, 1998, 275, H1886-H1897.	3.2	17
35	Electrode Positioning for Reliable Telemetry ECG Recordings During Social Stress in Unrestrained Rats. Physiology and Behavior, 1996, 60, 1397-1401.	2.1	125
36	Maternal aggression as a model for acute social stress in the rat: A behavioral-electrocardiographic study. Aggressive Behavior, 1995, 21, 79-89.	2.4	11

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37	Offensive and defensive biteâ€ŧarget topographies in attacks by lactating rats. Aggressive Behavior, 1992, 18, 47-52.	2.4	23
38	Diagnostic features of body surface potential maps in patients with myocardial ischemia and normal resting 12-lead electrocardiograms. American Journal of Cardiology, 1990, 65, 973-979.	1.6	3
39	Newer data on the configuration and variability ranges of body surface maps in a sample of normal subjects. Journal of Electrocardiology, 1988, 21, 1-14.	0.9	25
40	Body surface maps in left bundle branch block uncomplicated or complicated by myocardial infarction, left ventricular hypertrophy or myocardial ischemia. Journal of Electrocardiology, 1987, 20, 1-20.	0.9	36