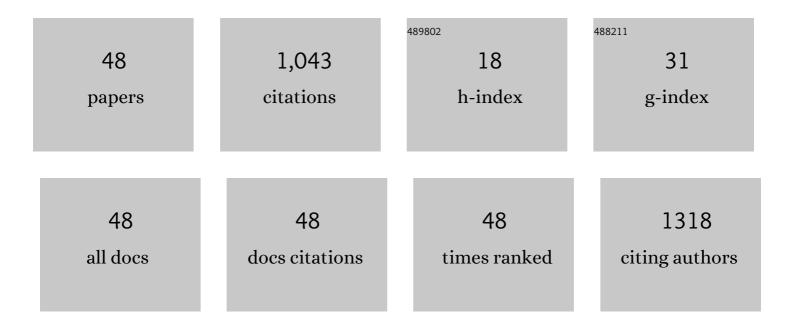
## Oleg E Osadchii

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Electrocardiographic marker of the cardiac action potential triangulation induced by antiarrhythmic drugs in perfused guineaâ€pig heart. Experimental Physiology, 2022, 107, 864-878.	0.9	1
2	Antiarrhythmic drug effects on premature beats are partly determined by prior cardiac activation frequency in perfused guineaâ€pig heart. Experimental Physiology, 2020, 105, 819-830.	0.9	1
3	Integrated curriculum: a contemporary innovation strategy in medical education. Kuban Scientific Medical Bulletin, 2020, 27, 51-61.	0.1	1
4	What is the Difference between Online Learning and Traditional Teaching in Medical Education?. Kuban Scientific Medical Bulletin, 2020, 27, 175-183.	0.1	2
5	Effects of antiarrhythmics on the electrical restitution in perfused guineaâ€pig heart are critically determined by the applied cardiac pacing protocol. Experimental Physiology, 2019, 104, 490-504.	0.9	9
6	Effects of antiarrhythmics and hypokalemia on the rate adaptation of cardiac repolarization. Scandinavian Cardiovascular Journal, 2018, 52, 218-226.	0.4	4
7	Determinants of slowed conduction in premature ventricular beats induced during programmed stimulations in perfused guineaâ€pig heart. Experimental Physiology, 2018, 103, 1230-1242.	0.9	2
8	Arrhythmogenic drugs can amplify spatial heterogeneities in the electrical restitution in perfused guinea-pig heart: An evidence from assessments of monophasic action potential durations and JT intervals. PLoS ONE, 2018, 13, e0191514.	1.1	5
9	Assessments of the QT/QRS restitution in perfused guinea-pig heart can discriminate safe and arrhythmogenic drugs. Journal of Pharmacological and Toxicological Methods, 2017, 87, 27-37.	0.3	1
10	Effects of Na+ channel blockers on the restitution of refractory period, conduction time, and excitation wavelength in perfused guinea-pig heart. PLoS ONE, 2017, 12, e0172683.	1.1	8
11	Flecainide attenuates rate adaptation of ventricular repolarization in guinea-pig heart. Scandinavian Cardiovascular Journal, 2016, 50, 28-35.	0.4	8
12	Flecainide-induced prolongation of ventricular repolarization contributes to the proarrhythmic profile of action. International Journal of Cardiology, 2015, 197, 81-82.	0.8	3
13	Emerging role of neurotensin in regulation of the cardiovascular system. European Journal of Pharmacology, 2015, 762, 184-192.	1.7	45
14	Impact of Hypokalemia on Electromechanical Window, Excitation Wavelength and Repolarization Gradients in Guinea-Pig and Rabbit Hearts. PLoS ONE, 2014, 9, e105599.	1.1	21
15	Reduced intrinsic heart rate is associated with reduced arrhythmic susceptibility in guinea-pig heart. Scandinavian Cardiovascular Journal, 2014, 48, 357-367.	0.4	4
16	Impaired epicardial activation–repolarization coupling contributes to the proarrhythmic effects of hypokalaemia and dofetilide in guinea pig ventricles. Acta Physiologica, 2014, 211, 48-60.	1.8	11
17	Effects of Na+ Channel Blockers on Extrasystolic Stimulation-evoked Changes in Ventricular Conduction and Repolarization. Journal of Cardiovascular Pharmacology, 2014, 63, 240-251.	0.8	12
18	Procainamide and lidocaine produce dissimilar changes in ventricular repolarization and arrhythmogenicity in guineaâ€pig. Fundamental and Clinical Pharmacology, 2014, 28, 382-393.	1.0	16

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19	Quinidine elicits proarrhythmic changes in ventricular repolarization and refractoriness in guinea-pig. Canadian Journal of Physiology and Pharmacology, 2013, 91, 306-315.	0.7	18
20	Flecainide-Induced Proarrhythmia Is Attributed to Abnormal Changes in Repolarization and Refractoriness in Perfused Guinea-Pig Heart. Journal of Cardiovascular Pharmacology, 2012, 60, 456-466.	0.8	18
21	Dofetilide Promotes Repolarization Abnormalities in Perfused Guinea-pig Heart. Cardiovascular Drugs and Therapy, 2012, 26, 489-500.	1.3	20
22	Effects of ventricular pacing protocol on electrical restitution assessments in guineaâ€pig heart. Experimental Physiology, 2012, 97, 807-821.	0.9	29
23	Impact of Na+ channel blockers on transmural dispersion of refractoriness and arrhythmic susceptibility in guinea-pig left ventricle. European Journal of Pharmacology, 2012, 691, 173-181.	1.7	7
24	Electrophysiological determinants of arrhythmic susceptibility upon endocardial and epicardial pacing in guineaâ€pig heart. Acta Physiologica, 2012, 205, 494-506.	1.8	6
25	Myocardial structural, contractile and electrophysiological changes in the guinea-pig heart failure model induced by chronic sympathetic activation. Experimental Physiology, 2011, 96, 647-663.	0.9	35
26	Chronic sympathetic activation promotes downregulation of β-adrenoceptor-mediated effects in the guinea pig heart independently of structural remodeling and systolic dysfunction. Pflugers Archiv European Journal of Physiology, 2011, 462, 529-543.	1.3	20
27	Na+ channel distribution and electrophysiological heterogeneities in guinea pig ventricular wall. American Journal of Physiology - Heart and Circulatory Physiology, 2011, 300, H989-H1002.	1.5	18
28	Mechanisms of hypokalemiaâ€induced ventricular arrhythmogenicity. Fundamental and Clinical Pharmacology, 2010, 24, 547-559.	1.0	119
29	Predictive value of electrical restitution in hypokalemia-induced ventricular arrhythmogenicity. American Journal of Physiology - Heart and Circulatory Physiology, 2010, 298, H210-H220.	1.5	29
30	Activation of big conductance Ca2+-activated K+ channels (BK) protects the heart against ischemia–reperfusion injury. Pflugers Archiv European Journal of Physiology, 2009, 457, 979-988.	1.3	84
31	Chamberâ€ <b>s</b> pecific effects of hypokalaemia on ventricular arrhythmogenicity in isolated, perfused guineaâ€pig heart. Experimental Physiology, 2009, 94, 434-446.	0.9	15
32	Electrophysiological determinants of hypokalaemiaâ€induced arrhythmogenicity in the guineaâ€pig heart. Acta Physiologica, 2009, 197, 273-287.	1.8	26
33	Subtype-selective blockade of cardiac muscarinic receptors inhibits vagal chronotropic responses in cats. Pflugers Archiv European Journal of Physiology, 2008, 455, 819-828.	1.3	1
34	Susceptibility to systolic dysfunction in the myocardium from chronically infarcted spontaneously hypertensive rats. American Journal of Physiology - Heart and Circulatory Physiology, 2008, 294, H372-H378.	1.5	5
35	Cardiac dilatation and pump dysfunction without intrinsic myocardial systolic failure following chronic Î <sup>2</sup> -adrenoreceptor activation. American Journal of Physiology - Heart and Circulatory Physiology, 2007, 292, H1898-H1905.	1.5	55
36	Rat strain-related differences in myocardial adrenergic tone and the impact on cardiac fibrosis, adrenergic responsiveness and myocardial structure and function. Pharmacological Research, 2007, 55, 287-294.	3.1	18

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#	Article	IF	CITATIONS
37	Cardiac hypertrophy induced by sustained β-adrenoreceptor activation: pathophysiological aspects. Heart Failure Reviews, 2007, 12, 66-86.	1.7	147
38	Myocardial Phosphodiesterases and Regulation of Cardiac Contractility in Health and Cardiac Disease. Cardiovascular Drugs and Therapy, 2007, 21, 171-194.	1.3	75
39	Temporal changes in myocardial adrenergic regulation with the progression to pump dysfunction after chronic β-adrenoreceptor activation in rats. Pflugers Archiv European Journal of Physiology, 2007, 455, 251-260.	1.3	5
40	Neurotensin-Induced Myocardial Noradrenergic Effects in Spontaneously Hypertensive Rats. Journal of Cardiovascular Pharmacology, 2006, 47, 221-227.	0.8	16
41	Contractile responses to selective phosphodiesterase inhibitors following chronic β-adrenoreceptor activation. Pflugers Archiv European Journal of Physiology, 2006, 452, 155-163.	1.3	19
42	Phosphodiesterase inhibition promotes the transition from compensated hypertrophy to cardiac dilatation in rats. Pflugers Archiv European Journal of Physiology, 2006, 451, 526-533.	1.3	6
43	Impact of chronic β-adrenoceptor activation on neurotensin-induced myocardial effects in rats. European Journal of Pharmacology, 2006, 553, 246-253.	1.7	6
44	Inotropic responses to phosphodiesterase inhibitors in cardiac hypertrophy in rats. European Journal of Pharmacology, 2005, 514, 201-208.	1.7	10
45	Impact and mechanisms of action of neurotensin on cardiac contractility in the rat left ventricle. European Journal of Pharmacology, 2005, 520, 108-117.	1.7	17
46	Aldosterone Receptor Blockade Prevents the Transition to Cardiac Pump Dysfunction Induced by β-Adrenoreceptor Activation. Hypertension, 2005, 45, 914-920.	1.3	31
47	Mechanisms of preserved baseline cardiac systolic function in rats with adrenergic inotropic downregulation. Life Sciences, 2005, 78, 366-375.	2.0	16
48	Chronic Î <sup>2</sup> -adrenoreceptor activation increases cardiac cavity size through chamber remodeling and not via modifications in myocardial material properties. American Journal of Physiology - Heart and Circulatory Physiology, 2004, 287, H2762-H2767.	1.5	18