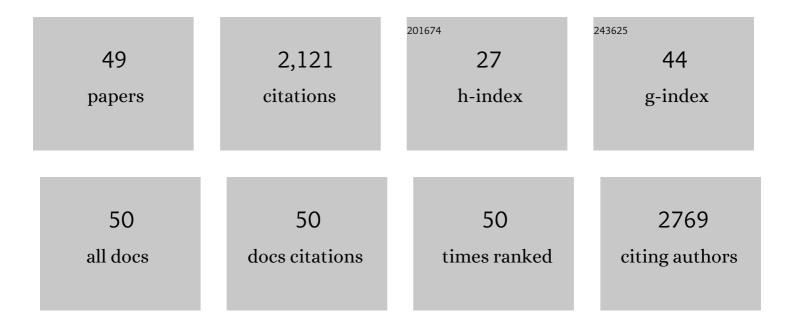
Mark J Kohr

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Cysteine 203 of Cyclophilin D Is Critical for Cyclophilin D Activation of the Mitochondrial Permeability Transition Pore. Journal of Biological Chemistry, 2011, 286, 40184-40192.	3.4	163
2	Simultaneous Measurement of Protein Oxidation and <i>S</i> -Nitrosylation During Preconditioning and Ischemia/Reperfusion Injury With Resin-Assisted Capture. Circulation Research, 2011, 108, 418-426.	4.5	150
3	Nitric oxide signaling and the regulation of myocardial function. Journal of Molecular and Cellular Cardiology, 2008, 45, 625-632.	1.9	130
4	Characterization of potential <i>S</i> -nitrosylation sites in the myocardium. American Journal of Physiology - Heart and Circulatory Physiology, 2011, 300, H1327-H1335.	3.2	129
5	S-nitrosylation: A radical way to protect the heart. Journal of Molecular and Cellular Cardiology, 2012, 52, 568-577.	1.9	92
6	Unlocking the Secrets of Mitochondria in the Cardiovascular System. Circulation, 2019, 140, 1205-1216.	1.6	91
7	Regulation of myocyte contraction via neuronal nitric oxide synthase: role of ryanodine receptor <i>S</i> â€nitrosylation. Journal of Physiology, 2010, 588, 2905-2917.	2.9	80
8	Signaling by S-nitrosylation in the heart. Journal of Molecular and Cellular Cardiology, 2014, 73, 18-25.	1.9	79
9	Ischaemic preconditioning preferentially increases protein S-nitrosylation in subsarcolemmal mitochondria. Cardiovascular Research, 2015, 106, 227-236.	3.8	74
10	Divergent Effects of miRâ€181 Family Members on Myocardial Function Through Protective Cytosolic and Detrimental Mitochondrial microRNA Targets. Journal of the American Heart Association, 2017, 6, .	3.7	74
11	Endothelial nitric oxide synthase decreases β-adrenergic responsiveness via inhibition of the L-type Ca ²⁺ current. American Journal of Physiology - Heart and Circulatory Physiology, 2008, 294, H1473-H1480.	3.2	70
12	Measurement of <i>S</i> -Nitrosylation Occupancy in the Myocardium With Cysteine-Reactive Tandem Mass Tags. Circulation Research, 2012, 111, 1308-1312.	4.5	70
13	Disruption of Caveolae Blocks Ischemic Preconditioning-Mediated S-Nitrosylation of Mitochondrial Proteins. Antioxidants and Redox Signaling, 2012, 16, 45-56.	5.4	61
14	S-nitrosylation of TRIM72 at cysteine 144 is critical for protection against oxidation-induced protein degradation and cell death. Journal of Molecular and Cellular Cardiology, 2014, 69, 67-74.	1.9	61
15	Essential role of nitric oxide in acute ischemic preconditioning: S-Nitros(yl)ation versus sGC/cGMP/PKG signaling?. Free Radical Biology and Medicine, 2013, 54, 105-112.	2.9	59
16	Neuronal nitric oxide synthase signaling within cardiac myocytes targets phospholamban. American Journal of Physiology - Cell Physiology, 2008, 294, C1566-C1575.	4.6	58
17	<i>S</i> -Nitrosylation: Specificity, Occupancy, and Interaction with Other Post-Translational Modifications. Antioxidants and Redox Signaling, 2013, 19, 1209-1219.	5.4	56
18	Postconditioning leads to an increase in protein <i>S</i> -nitrosylation. American Journal of Physiology - Heart and Circulatory Physiology, 2014, 306, H825-H832.	3.2	48

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19	Sustained elevation of MG53 in the bloodstream increases tissue regenerative capacity without compromising metabolic function. Nature Communications, 2019, 10, 4659.	12.8	47
20	Glyceraldehyde-3-Phosphate Dehydrogenase Acts as a Mitochondrial Trans-S-Nitrosylase in the Heart. PLoS ONE, 2014, 9, e111448.	2.5	45
21	Phosphodiesterase 5 restricts NOS3/Soluble guanylate cyclase signaling to L-type Ca2+ current in cardiac myocytes. Journal of Molecular and Cellular Cardiology, 2009, 47, 304-314.	1.9	42
22	Targeting of phospholamban by peroxynitrite decreases β-adrenergic stimulation in cardiomyocytes. Cardiovascular Research, 2008, 77, 353-361.	3.8	41
23	Nitroxyl enhances myocyte Ca2 transients by exclusively targeting SR Ca2 -cycling. Frontiers in Bioscience - Elite, 2010, E2, 614-626.	1.8	36
24	Characterization of the sex-dependent myocardial <i>S</i> -nitrosothiol proteome. American Journal of Physiology - Heart and Circulatory Physiology, 2016, 310, H505-H515.	3.2	35
25	<i>S</i> -Nitrosoglutathione Reductase Is Essential for Protecting the Female Heart From Ischemia-Reperfusion Injury. Circulation Research, 2018, 123, 1232-1243.	4.5	35
26	cAMP-independent activation of protein kinase A by the peroxynitrite generator SIN-1 elicits positive inotropic effects in cardiomyocytes. Journal of Molecular and Cellular Cardiology, 2010, 48, 645-648.	1.9	34
27	Mechanism of Cardioprotection: What Can We Learn from Females?. Pediatric Cardiology, 2011, 32, 354-359.	1.3	30
28	Peroxynitrite increases protein phosphatase activity and promotes the interaction of phospholamban with protein phosphatase 2a in the myocardium. Nitric Oxide - Biology and Chemistry, 2009, 20, 217-221.	2.7	27
29	Synthesis and biological testing of aminoxyls designed for long-term retention by living cells. Organic and Biomolecular Chemistry, 2005, 3, 645.	2.8	25
30	Effect of Sodium Nitrite on Ischaemia and Reperfusion-Induced Arrhythmias in Anaesthetized Dogs: Is Protein S-Nitrosylation Involved?. PLoS ONE, 2015, 10, e0122243.	2.5	19
31	Biphasic effect of SIN-1 is reliant upon cardiomyocyte contractile state. Free Radical Biology and Medicine, 2008, 45, 73-80.	2.9	18
32	miRâ€181c Activates Mitochondrial Calcium Uptake by Regulating MICU1 in the Heart. Journal of the American Heart Association, 2019, 8, e012919.	3.7	18
33	An emerging perspective on sex differences: Intersecting S-nitrosothiol and aldehyde signaling in the heart. Redox Biology, 2020, 31, 101441.	9.0	18
34	Adenosine A1 receptor activation increases myocardial protein S-nitrosothiols and elicits protection from ischemia-reperfusion injury in male and female hearts. PLoS ONE, 2017, 12, e0177315.	2.5	18
35	Inorganic arsenic exposure induces sex-disparate effects and exacerbates ischemia-reperfusion injury in the female heart. American Journal of Physiology - Heart and Circulatory Physiology, 2019, 316, H1053-H1064.	3.2	16
36	Modulation of myocardial contraction by peroxynitrite. Frontiers in Physiology, 2012, 3, 468.	2.8	15

#	Article	IF	CITATIONS
37	Inorganic arsenic induces sex-dependent pathological hypertrophy in the heart. American Journal of Physiology - Heart and Circulatory Physiology, 2021, 320, H1321-H1336.	3.2	15
38	Nuclear-mitochondrial communication involving miR-181c plays an important role in cardiac dysfunction during obesity. Journal of Molecular and Cellular Cardiology, 2020, 144, 87-96.	1.9	12
39	Cardioprotective Role of Caveolae in Ischemia-Reperfusion Injury. Translational Medicine (Sunnyvale,) Tj ETQq1 1	0.784314 0.4	rgBT /Over
40	Prolonged Action Potential and After depolarizations Are Not due to Changes in Potassium Currents in NOS3 Knockout Ventricular Myocytes. Journal of Signal Transduction, 2012, 2012, 1-8.	2.0	6
41	Kcnj11 Ablation Is Associated With Increased Nitro-Oxidative Stress During Ischemia-Reperfusion Injury. Circulation: Heart Failure, 2017, 10, .	3.9	6
42	A knock-in mutation at cysteine 144 of TRIM72 is cardioprotective and reduces myocardial TRIM72 release. Journal of Molecular and Cellular Cardiology, 2019, 136, 95-101.	1.9	5
43	Mitsugumin-53: Potential biomarker and therapeutic for myocardial ischemic injury?. Journal of Molecular and Cellular Cardiology, 2015, 81, 46-48.	1.9	4
44	Sâ€nitrosylation of cyclophilin D alters mitochondrial permeability transition pore. FASEB Journal, 2011, 25, 1033.1.	0.5	2
45	Nitroxyl (HNO) Modifies Cysteine Residues in Phospholamban to Increase Myocyte Ca2+-Cycling and Contractility. Biophysical Journal, 2009, 96, 515a-516a.	0.5	Ο
46	Cysteine 203 of cyclophilin D is critical for cyclophilin D activation of the mitochondrial permeability transition pore Journal of Biological Chemistry, 2012, 287, 34496-34498.	3.4	0
47	Post-translational Modifications in the Cardiovascular Proteome. , 2016, , 293-320.		0
48	Identifying Regulators of Oâ€GlcNAcylation in the Broken Heart. FASEB Journal, 2020, 34, 1-1.	0.5	0
49	Abstract 16165: Inorganic Arsenic Induces Sex-Dependent Pathological Cardiac Hypertrophy. Circulation, 2020, 142, .	1.6	0

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