

Vincent J De Beer

List of Publications by Year in descending order

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Version: 2024-02-01

21
papers

337
citations

933264

10
h-index

1281743

11
g-index

21
all docs

21
docs citations

21
times ranked

618
citing authors

#	ARTICLE	IF	CITATIONS
1	Cellular, mitochondrial and molecular alterations associate with early left ventricular diastolic dysfunction in a porcine model of diabetic metabolic derangement. <i>Scientific Reports</i> , 2020, 10, 13173.	1.6	15
2	Differential impact of severe familial hypercholesterolemia on regional skeletal muscle and organ blood flows during exercise: Effects of PDE 5 inhibition. <i>Microcirculation</i> , 2019, 26, e12539.	1.0	0
3	Differential impact of severe familial hypercholesterolemia on regional skeletal muscle and organ blood flows during exercise: effects of PDE5 inhibition. <i>FASEB Journal</i> , 2019, 33, lb457.	0.2	0
4	Multiple common comorbidities produce left ventricular diastolic dysfunction associated with coronary microvascular dysfunction, oxidative stress, and myocardial stiffening. <i>Cardiovascular Research</i> , 2018, 114, 954-964.	1.8	148
5	Intermittent pacing therapy favorably modulates infarct remodeling. <i>Basic Research in Cardiology</i> , 2017, 112, 28.	2.5	3
6	Altered purinergic signaling in uridine adenosine tetraphosphate-induced coronary relaxation in swine with metabolic derangement. <i>Purinergic Signalling</i> , 2017, 13, 319-329.	1.1	12
7	Coronary microvascular dysfunction after long-term diabetes and hypercholesterolemia. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2016, 311, H1339-H1351.	1.5	52
8	Severe familial hypercholesterolemia impairs the regulation of coronary blood flow and oxygen supply during exercise. <i>Basic Research in Cardiology</i> , 2016, 111, 61.	2.5	24
9	What can we learn about treating heart failure from the heart's response to acute exercise? Focus on the coronary microcirculation. <i>Journal of Applied Physiology</i> , 2015, 119, 934-943.	1.2	20
10	Familial hypercholesterolemia impairs exercise-induced systemic vasodilation due to reduced NO bioavailability. <i>Journal of Applied Physiology</i> , 2013, 115, 1767-1776.	1.2	12
11	Cytochrome P450 2C9 contributes to pulmonary vasoconstriction in exercising swine. <i>FASEB Journal</i> , 2013, 27, 898.1.	0.2	0
12	Phosphodiesterase ϵ 5 activity exerts a coronary vasoconstrictor influence in awake swine that is partly mediated via an increase in endothelin production. <i>FASEB Journal</i> , 2013, 27, 1185.5.	0.2	0
13	Reduced NO Bioavailability Hampers Exercise Induced Vasodilation in Familial Hypercholesterolemic Swine. <i>FASEB Journal</i> , 2012, 26, 1138.25.	0.2	0
14	Blunted exercise-induced vasodilation in familial hypercholesterolemic swine does not involve enhanced ET ϵ 1-mediated vasoconstriction. <i>FASEB Journal</i> , 2012, 26, 1138.22.	0.2	0
15	Enhanced myofilament responsiveness upon β ² -adrenergic stimulation in post-infarct remodeled myocardium. <i>Journal of Molecular and Cellular Cardiology</i> , 2011, 50, 487-499.	0.9	24
16	Prostanoids suppress the coronary vasoconstrictor influence of endothelin after myocardial infarction. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2011, 301, H1080-H1089.	1.5	16
17	Blunted pulmonary dilation during exercise in familial hypercholesterolemic swine does not involve endothelin ϵ 1. <i>FASEB Journal</i> , 2011, 25, 1102.10.	0.2	0
18	Exercise reduces production of endothelin in the coronary circulation. <i>FASEB Journal</i> , 2009, 23, LB49.	0.2	0

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19	Effect of phosphodiesterase 5 inhibition on systemic hemodynamics and regional blood flows in exercising swine. FASEB Journal, 2009, 23, 948.17.	0.2	0
20	Integrative control of coronary resistance vessel tone by endothelin and angiotensin II is altered in swine with a recent myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2008, 294, H2069-H2077.	1.5	11
21	Phosphodiesterase 5 activity is reduced in the coronary circulation of swine after myocardial infarction. FASEB Journal, 2007, 21, A1382.	0.2	0