

Ellen Aasum

List of Publications by Year in descending order

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Version: 2024-02-01

47
papers

2,615
citations

186265

28
h-index

214800

47
g-index

48
all docs

48
docs citations

48
times ranked

3166
citing authors

#	ARTICLE	IF	CITATIONS
1	Overexpression of NOX2 Exacerbates AngII-Mediated Cardiac Dysfunction and Metabolic Remodelling. <i>Antioxidants</i> , 2022, 11, 143.	5.1	2
2	Diet-induced obese mouse hearts tolerate an acute high-fatty acid exposure that also increases ischemic tolerance. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2020, 319, H682-H693.	3.2	6
3	NADPH Oxidase 2 Mediates Myocardial Oxygen Wasting in Obesity. <i>Antioxidants</i> , 2020, 9, 171.	5.1	10
4	3-Weeks of Exercise Training Increases Ischemic-Tolerance in Hearts From High-Fat Diet Fed Mice. <i>Frontiers in Physiology</i> , 2019, 10, 1274.	2.8	6
5	The role of NADPH oxidases in diabetic cardiomyopathy. <i>Biochimica Et Biophysica Acta - Molecular Basis of Disease</i> , 2018, 1864, 1908-1913.	3.8	60
6	Isolated perfused working hearts provide valuable additional information during phenotypic assessment of the diabetic mouse heart. <i>PLoS ONE</i> , 2018, 13, e0204843.	2.5	7
7	Exercise of obese mice induces cardioprotection and oxygen sparing in hearts exposed to high-fat load. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2017, 313, H1054-H1062.	3.2	18
8	Nox4 reprograms cardiac substrate metabolism via protein O-GlcNAcylation to enhance stress adaptation. <i>JCI Insight</i> , 2017, 2, .	5.0	42
9	Increased oxidative metabolism following hypoxia in the type 2 diabetic heart, despite normal hypoxia signalling and metabolic adaptation. <i>Journal of Physiology</i> , 2016, 594, 307-320.	2.9	40
10	On the pivotal role of PPAR α in adaptation of the heart to hypoxia and why fat in the diet increases hypoxic injury. <i>FASEB Journal</i> , 2016, 30, 2684-2697.	0.5	54
11	Dietary Calanus oil antagonizes angiotensin II-induced hypertension and tissue wasting in diet-induced obese mice. <i>Prostaglandins Leukotrienes and Essential Fatty Acids</i> , 2016, 108, 13-21.	2.2	7
12	Response to Letter Regarding Article, "Myosin Activator Omecamtiv Mecarbil Increases Myocardial Oxygen Consumption and Impairs Cardiac Efficiency Mediated by Resting Myosin ATPase Activity": Circulation: Heart Failure, 2015, 8, 1142-1142.	3.9	3
13	How Exercise May Amend Metabolic Disturbances in Diabetic Cardiomyopathy. <i>Antioxidants and Redox Signaling</i> , 2015, 22, 1587-1605.	5.4	57
14	Myosin Activator Omecamtiv Mecarbil Increases Myocardial Oxygen Consumption and Impairs Cardiac Efficiency Mediated by Resting Myosin ATPase Activity. <i>Circulation: Heart Failure</i> , 2015, 8, 766-775.	3.9	48
15	Wax Esters from the Marine Copepod <i>Calanus finmarchicus</i> Reduce Diet-Induced Obesity and Obesity-Related Metabolic Disorders in Mice. <i>Journal of Nutrition</i> , 2014, 144, 164-169.	2.9	28
16	High- and Moderate-Intensity Training Normalizes Ventricular Function and Mechanoenergetics in Mice With Diet-Induced Obesity. <i>Diabetes</i> , 2013, 62, 2287-2294.	0.6	79
17	Oil from the marine zooplankton <i>Calanus finmarchicus</i> improves the cardiometabolic phenotype of diet-induced obese mice. <i>British Journal of Nutrition</i> , 2013, 110, 2186-2193.	2.3	29
18	Cardiomyocyte-restricted inhibition of G protein-coupled receptor kinase-3 attenuates cardiac dysfunction after chronic pressure overload. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2012, 303, H66-H74.	3.2	13

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19	Cardioprotective effect of the PPAR ligand tetradecylthioacetic acid in type 2 diabetic mice. American Journal of Physiology - Heart and Circulatory Physiology, 2011, 300, H2116-H2122.	3.2	12
20	Chronic and acute exposure of mouse hearts to fatty acids increases oxygen cost of excitation-contraction coupling. American Journal of Physiology - Heart and Circulatory Physiology, 2011, 300, H1631-H1636.	3.2	14
21	Interval Training Normalizes Cardiomyocyte Function, Diastolic Ca ²⁺ Control, and SR Ca ²⁺ Release Synchronicity in a Mouse Model of Diabetic Cardiomyopathy. Circulation Research, 2009, 105, 527-536.	4.5	173
22	Cardiac peroxisome proliferator-activated receptor- α activation causes increased fatty acid oxidation, reducing efficiency and post-ischaemic functional loss. Cardiovascular Research, 2009, 83, 519-526.	3.8	56
23	Increased O ₂ cost of basal metabolism and excitation-contraction coupling in hearts from type 2 diabetic mice. American Journal of Physiology - Heart and Circulatory Physiology, 2009, 296, H1373-H1379.	3.2	42
24	Activation of a HIF1 α -PPAR α Axis Underlies the Integration of Glycolytic and Lipid Anabolic Pathways in Pathologic Cardiac Hypertrophy. Cell Metabolism, 2009, 9, 512-524.	16.2	342
25	Metabolic (In)Flexibility of the Diabetic Heart. Cardiovascular Drugs and Therapy, 2008, 22, 91-95.	2.6	33
26	Novel MRI method to detect altered left ventricular ejection and filling patterns in rodent models of disease. Magnetic Resonance in Medicine, 2008, 60, 582-587.	3.0	37
27	Fenofibrate modulates cardiac and hepatic metabolism and increases ischemic tolerance in diet-induced obese mice. Journal of Molecular and Cellular Cardiology, 2008, 44, 201-209.	1.9	50
28	Myocardial mechanical dysfunction and calcium overload following rewarming from experimental hypothermia in vivo. Cryobiology, 2008, 56, 15-21.	0.7	73
29	Cardiac-restricted Expression of the Carboxyl-terminal Fragment of GRK3 Uncovers Distinct Functions of GRK3 in Regulation of Cardiac Contractility and Growth. Journal of Biological Chemistry, 2008, 283, 10601-10610.	3.4	30
30	Glucose and insulin improve cardiac efficiency and postischemic functional recovery in perfused hearts from type 2 diabetic (db/db) mice. American Journal of Physiology - Endocrinology and Metabolism, 2007, 292, E1288-E1294.	3.5	64
31	Overexpression of angiotensinogen in the myocardium induces downregulation of the fatty acid oxidation pathway. Journal of Molecular and Cellular Cardiology, 2006, 41, 459-466.	1.9	54
32	Cardiac metabolism in mice: tracer method developments and in vivo application revealing profound metabolic inflexibility in diabetes. American Journal of Physiology - Endocrinology and Metabolism, 2006, 290, E870-E881.	3.5	45
33	Perfused hearts from Type 2 diabetic (db/db) mice show metabolic responsiveness to insulin. American Journal of Physiology - Heart and Circulatory Physiology, 2006, 290, H1763-H1769.	3.2	62
34	Increased Myocardial Oxygen Consumption Reduces Cardiac Efficiency in Diabetic Mice. Diabetes, 2006, 55, 466-473.	0.6	219
35	Effect of BM 17.0744, a PPAR α ligand, on the metabolism of perfused hearts from control and diabetic mice. Canadian Journal of Physiology and Pharmacology, 2005, 83, 183-190.	1.4	35
36	Influence of substrate supply on cardiac efficiency, as measured by pressure-volume analysis in ex vivo mouse hearts. American Journal of Physiology - Heart and Circulatory Physiology, 2005, 288, H2979-H2985.	3.2	88

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37	Endogenous glycogen prevents Ca ²⁺ -overload and hypercontracture in harp seal myocardial cells during simulated ischemia. <i>Journal of Molecular and Cellular Cardiology</i> , 2004, 37, 43-50.	1.9	19
38	Treatment of type 2 diabetic db/db mice with a novel PPAR δ agonist improves cardiac metabolism but not contractile function. <i>American Journal of Physiology - Endocrinology and Metabolism</i> , 2004, 286, E449-E455.	3.5	83
39	Change in substrate metabolism in isolated mouse hearts following ischemia-reperfusion. <i>Molecular and Cellular Biochemistry</i> , 2003, 249, 97-103.	3.1	15
40	Age-Dependent Changes in Metabolism, Contractile Function, and Ischemic Sensitivity in Hearts From db/db Mice. <i>Diabetes</i> , 2003, 52, 434-441.	0.6	247
41	Changes in substrate metabolism in isolated mouse hearts following ischemia-reperfusion. <i>Molecular and Cellular Biochemistry</i> , 2003, 249, 97-103.	3.1	9
42	Cardiac function and metabolism in Type 2 diabetic mice after treatment with BM 17.0744, a novel PPAR δ activator. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2002, 283, H949-H957.	3.2	145
43	Glucose-insulin-potassium reduces infarct size when administered during reperfusion. <i>Cardiovascular Drugs and Therapy</i> , 2000, 14, 615-623.	2.6	100
44	Different Tolerance to Hypothermia and Rewarming of Isolated Rat and Guinea Pig Hearts. <i>Cryobiology</i> , 1999, 38, 243-249.	0.7	8
45	The Role of Glycolysis in Myocardial Calcium Control. <i>Journal of Molecular and Cellular Cardiology</i> , 1998, 30, 1703-1712.	1.9	17
46	Stimulation of Carbohydrate Metabolism Reduces Hypothermia-induced Calcium Load in Fatty Acid-perfused Rat Hearts. <i>Journal of Molecular and Cellular Cardiology</i> , 1997, 29, 527-534.	1.9	8
47	Effects of fatty acids on myocardial calcium control during hypothermic perfusion. <i>Journal of Thoracic and Cardiovascular Surgery</i> , 1994, 107, 233-241.	0.8	20