Merry L Lindsey

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/4354119/publications.pdf

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278 papers

14,438 citations

64 h-index 24961 109 g-index

281 all docs

281 docs citations

times ranked

281

15410 citing authors

#	Article	IF	CITATIONS
1	S100A9 is a functional effector of infarct wall thinning after myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2022, 322, H145-H155.	1.5	11
2	Faster skin wound healing predicts survival after myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2022, 322, H537-H548.	1.5	7
3	Macrophages secrete murinoglobulin-1 and galectin-3 to regulate neutrophil degranulation after myocardial infarction. Molecular Omics, 2022, 18, 186-195.	1.4	9
4	Metabolic Transformation of Fat in Obesity Determines the Inflammation Resolving Capacity of Splenocardiac and Cardiorenal Networks in Heart Failure. American Journal of Physiology - Heart and Circulatory Physiology, 2022, , .	1.5	5
5	Faster skin wound healing predicts survival after myocardial infarction. FASEB Journal, 2022, 36, .	0.2	O
6	Cardiac Fibroblasts Secrete Galectinâ€1 After Myocardial Infarction to Communicate With Macrophages. FASEB Journal, 2022, 36, .	0.2	0
7	MMP-12 polarizes neutrophil signalome towards an apoptotic signature. Journal of Proteomics, 2022, 264, 104636.	1.2	4
8	Neutrophil signaling during myocardial infarction wound repair. Cellular Signalling, 2021, 77, 109816.	1.7	44
9	CD4 ⁺ T Cellâ€Specific Proteomic Pathways Identified in Progression of Hypertension Across Postmenopausal Transition. Journal of the American Heart Association, 2021, 10, e018038.	1.6	8
10	Network Analysis Reveals a Distinct Axis of Macrophage Activation in Response to Conflicting Inflammatory Cues. Journal of Immunology, 2021, 206, 883-891.	0.4	26
11	Infarct in the Heart: What's MMP-9 Got to Do with It?. Biomolecules, 2021, 11, 491.	1.8	37
12	Transient ACE (Angiotensin-Converting Enzyme) Inhibition Suppresses Future Fibrogenic Capacity and Heterogeneity of Cardiac Fibroblast Subpopulations. Hypertension, 2021, 77, 904-918.	1.3	13
13	Dysbiosis and Intestinal Barrier Dysfunction in Pediatric Congenital Heart Disease Is Exacerbated Following Cardiopulmonary Bypass. JACC Basic To Translational Science, 2021, 6, 311-327.	1.9	18
14	We are the change we seek. American Journal of Physiology - Heart and Circulatory Physiology, 2021, 320, H1411-H1414.	1.5	4
15	An American Physiological Society cross-journal Call for Papers on "Inter-Organ Communication in Homeostasis and Disease― American Journal of Physiology - Lung Cellular and Molecular Physiology, 2021, 321, L42-L49.	1.3	13
16	Proteomics Reveals Neutrophil Markers of Infarct Wall Thinning. FASEB Journal, 2021, 35, .	0.2	0
17	Reperfused vs. nonreperfused myocardial infarction: when to use which model. American Journal of Physiology - Heart and Circulatory Physiology, 2021, 321, H208-H213.	1.5	29
18	Effect of genetic depletion of MMP-9 on neurological manifestations of hypertension-induced intracerebral hemorrhages in aged mice. GeroScience, 2021, 43, 2611-2619.	2.1	10

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19	The HEART Camp Exercise Intervention Improves Exercise Adherence, Physical Function, and Patient-Reported Outcomes in Adults With Preserved Ejection Fraction Heart Failure. Journal of Cardiac Failure, 2021, , .	0.7	6
20	Reinforcing rigor and reproducibility expectations for use of sex and gender in cardiovascular research. American Journal of Physiology - Heart and Circulatory Physiology, 2021, 321, H819-H824.	1.5	49
21	Chronic <i>Porphyromonas gingivalis</i> lipopolysaccharide induces adverse myocardial infarction wound healing through activation of CD8 ⁺ T cells. American Journal of Physiology - Heart and Circulatory Physiology, 2021, 321, H948-H962.	1.5	15
22	Guidelines for in vivo mouse models of myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2021, 321, H1056-H1073.	1.5	53
23	Neutrophil crosstalk during cardiac wound healing after myocardial infarction. Current Opinion in Physiology, 2021, 24, 100485.	0.9	6
24	Secrets of Cardiac Remodeling Revealed in the Secretome. Circulation, 2020, 141, 1645-1647.	1.6	0
25	Using an Investigative Journalism Approach to Design Mechanistic Experiments in Physiology. Physiology, 2020, 35, 218-219.	1.6	0
26	Exogenous IL-4 shuts off pro-inflammation in neutrophils while stimulating anti-inflammation in macrophages to induce neutrophil phagocytosis following myocardial infarction. Journal of Molecular and Cellular Cardiology, 2020, 145, 112-121.	0.9	38
27	Fibroblasts: The arbiters of extracellular matrix remodeling. Matrix Biology, 2020, 91-92, 1-7.	1.5	75
28	Loss of $\langle i \rangle$ Arhgef11 $\langle i \rangle$ in the Dahl Salt-Sensitive Rat Protects Against Hypertension-Induced Renal Injury. Hypertension, 2020, 75, 1012-1024.	1.3	15
29	The compendium of matrix metalloproteinase expression in the left ventricle of mice following myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2020, 318, H706-H714.	1.5	16
30	COVID-19 and cardiovascular disease: What we know, what we think we know, and what we need to know. Journal of Molecular and Cellular Cardiology, 2020, 144, 12-14.	0.9	7
31	Focusing Heart Failure Research on Myocardial Fibrosis to Prioritize Translation. Journal of Cardiac Failure, 2020, 26, 876-884.	0.7	4
32	Cardiac fibroblast activation during myocardial infarction wound healing. Matrix Biology, 2020, 91-92, 109-116.	1.5	61
33	Exogenous ILâ€4 Promotes Myocardial Infarction Repair by Turning off Proâ€Inflammation in Neutrophils while Stimulating Antiâ€Inflammation in Macrophages to Induce Neutrophil Phagocytosis. FASEB Journal, 2020, 34, 1-1.	0.2	0
34	Extracellular matrix roles in cardiorenal fibrosis: Potential therapeutic targets for CVD and CKD in the elderly., 2019, 193, 99-120.		28
35	Neutrophil proteome shifts over the myocardial infarction time continuum. Basic Research in Cardiology, 2019, 114, 37.	2.5	78
36	Somewhere over the sex differences rainbow of myocardial infarction remodeling: hormones, chromosomes, inflammasome, oh my. Expert Review of Proteomics, 2019, 16, 933-940.	1.3	8

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37	Towards better definition, quantification and treatment of fibrosis in heart failure. A scientific roadmap by the Committee of Translational Research of the Heart Failure Association (HFA) of the European Society of Cardiology. European Journal of Heart Failure, 2019, 21, 272-285.	2.9	182
38	Common pathways and communication between the brain and heart: connecting post-traumatic stress disorder and heart failure. Stress, 2019, 22, 530-547.	0.8	22
39	Menopause and FOXP3+ Treg cell depletion eliminate female protection against T cell-mediated angiotensin II hypertension. American Journal of Physiology - Heart and Circulatory Physiology, 2019, 317, H415-H423.	1.5	31
40	Connecting the Dots for Connective Tissue Growth Factor Roles in CardiacÂWound Healing After MyocardialÂInfarction. JACC Basic To Translational Science, 2019, 4, 95-97.	1.9	0
41	Exogenous CXCL4 infusion inhibits macrophage phagocytosis by limiting CD36 signalling to enhance post-myocardial infarction cardiac dilation and mortality. Cardiovascular Research, 2019, 115, 395-408.	1.8	36
42	Identifying the molecular and cellular signature of cardiac dilation following myocardial infarction. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2019, 1865, 1845-1852.	1.8	6
43	Using Peptidomics to Identify Extracellular Matrix-Derived Peptides as Novel Therapeutics for Cardiac Disease. Molecular and Translational Medicine, 2019, , 349-365.	0.4	0
44	Matrix Metalloproteinase-9-Dependent Mechanisms of Reduced Contractility and Increased Stiffness in the Aging Heart. Molecular and Translational Medicine, 2019, , 335-347.	0.4	1
45	Fibroblast polarization over the myocardial infarction time continuum shifts roles from inflammation to angiogenesis. Basic Research in Cardiology, 2019, 114, 6.	2.5	118
46	Glycoproteomic Profiling Provides Candidate Myocardial Infarction Predictors of Later Progression to Heart Failure. ACS Omega, 2019, 4, 1272-1280.	1.6	10
47	Understanding cardiac extracellular matrix remodeling to develop biomarkers of myocardial infarction outcomes. Matrix Biology, 2019, 75-76, 43-57.	1.5	106
48	Understanding the mechanisms that determine extracellular matrix remodeling in the infarcted myocardium. Biochemical Society Transactions, 2019, 47, 1679-1687.	1.6	12
49	Physiological Omics Identifies Mechanisms that Attenuate Renal Injury and Blood Pressure in Dahl saltâ€sensitive Arhgef11 â^'/â^' Rats. FASEB Journal, 2019, 33, 571.1.	0.2	0
50	Myocardial infarctionâ€relevant MMPâ€12 interactions identified by correlation analysis. FASEB Journal, 2019, 33, 530.2.	0.2	0
51	Mapping neutrophil polarization over the myocardial infarction time continuum. FASEB Journal, 2019, 33, 690.12.	0.2	0
52	Physiological proteomics of heart failure. Current Opinion in Physiology, 2018, 1, 185-197.	0.9	1
53	Proteomic analysis of the cardiac extracellular matrix: clinical research applications. Expert Review of Proteomics, 2018, 15, 105-112.	1.3	40
54	Adding Reg $3\hat{l}^2$ to the acute coronary syndrome prognostic marker list. International Journal of Cardiology, 2018, 258, 24-25.	0.8	3

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55	Macrophage overexpression of matrix metalloproteinase-9 in aged mice improves diastolic physiology and cardiac wound healing after myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2018, 314, H224-H235.	1.5	37
56	Cardiac macrophage biology in the steady-state heart, the aging heart, and following myocardial infarction. Translational Research, 2018, 191, 15-28.	2.2	275
57	Exogenous CXCL4 Infusion Inhibits Macrophage Phagocytosis by Limiting CD36 Signaling to Enhance Post-myocardial Infarction Cardiac Dilation. Journal of Molecular and Cellular Cardiology, 2018, 124, 101-102.	0.9	0
58	Extracellular matrix in cardiovascular pathophysiology. American Journal of Physiology - Heart and Circulatory Physiology, 2018, 315, H1687-H1690.	1.5	18
59	Guidelines for authors and reviewers on antibody use in physiology studies. American Journal of Physiology - Heart and Circulatory Physiology, 2018, 314, H724-H732.	1.5	68
60	Death of an antioxidant brings heart failure with preserved ejection fraction to life: 5-oxoproline and post-ischaemic cardio-renal dysfunction. Cardiovascular Research, 2018, 114, 1819-1821.	1.8	4
61	Matrix metalloproteinase-12 as an endogenous resolution promoting factor following myocardial infarction. Pharmacological Research, 2018, 137, 252-258.	3.1	14
62	The Mouse Heart Attack Research Tool 1.0 database. American Journal of Physiology - Heart and Circulatory Physiology, 2018, 315, H522-H530.	1.5	14
63	Guidelines for experimental models of myocardial ischemia and infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2018, 314, H812-H838.	1.5	372
64	Statistical considerations in reporting cardiovascular research. American Journal of Physiology - Heart and Circulatory Physiology, 2018, 315, H303-H313.	1.5	58
65	Myocardial infarction remodeling that progresses to heart failure: a signaling misunderstanding. American Journal of Physiology - Heart and Circulatory Physiology, 2018, 315, H71-H79.	1.5	61
66	Guidelines for measuring cardiac physiology in mice. American Journal of Physiology - Heart and Circulatory Physiology, 2018, 314, H733-H752.	1.5	220
67	Assigning matrix metalloproteinase roles in ischaemic cardiac remodelling. Nature Reviews Cardiology, 2018, 15, 471-479.	6.1	87
68	LXR/RXR signaling and neutrophil phenotype following myocardial infarction classify sex differences in remodeling. Basic Research in Cardiology, 2018, 113, 40.	2.5	86
69	Mapping macrophage polarization over the myocardial infarction time continuum. Basic Research in Cardiology, 2018, 113, 26.	2.5	189
70	Reg-ulating macrophage infiltration to alter wound healing following myocardial infarction. Cardiovascular Research, 2018, 114, 1571-1572.	1.8	2
71	The Mouse Heart Attack Research Tool (mHART) 1.0 Database. FASEB Journal, 2018, 32, 848.5.	0.2	0
72	CD8 Tâ€cells have a biphasic role during postâ€myocardial infarction cardiac remodeling. FASEB Journal, 2018, 32, 718.5.	0.2	0

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73	Day 1 Postâ€Myocardial Infarction Cardiac Macrophage Transcriptomic Signatures that Link to LV Infarct Wall Thinning. FASEB Journal, 2018, 32, 717.11.	0.2	O
74	Antiarrhythmic effects of interleukin 1 inhibition after myocardial infarction. Heart Rhythm, 2017, 14, 727-736.	0.3	61
75	The impact of aging on cardiac extracellular matrix. GeroScience, 2017, 39, 7-18.	2.1	168
76	Glucose regulates the intrinsic inflammatory response of the heart to surgically induced hypothermic ischemic arrest and reperfusion. Physiological Genomics, 2017, 49, 37-52.	1.0	7
77	Transgenic overexpression of macrophage matrix metalloproteinase-9 exacerbates age-related cardiac hypertrophy, vessel rarefaction, inflammation, and fibrosis. American Journal of Physiology - Heart and Circulatory Physiology, 2017, 312, H375-H383.	1.5	51
78	Dentin Sialoprotein is a Novel Substrate of Matrix Metalloproteinase 9 in vitro and in vivo. Scientific Reports, 2017, 7, 42449.	1.6	15
79	IL-10 improves cardiac remodeling after myocardial infarction by stimulating M2 macrophage polarization and fibroblast activation. Basic Research in Cardiology, 2017, 112, 33.	2.5	278
80	Cardiac Fibroblast Activation Post-Myocardial Infarction: Current Knowledge Gaps. Trends in Pharmacological Sciences, 2017, 38, 448-458.	4.0	151
81	The physics of an academic career. American Journal of Physiology - Advances in Physiology Education, 2017, 41, 493-497.	0.8	2
82	Elevated serum osteoprotegerin is associated with increased left ventricular mass index and myocardial stiffness. Journal of Cardiovascular Medicine, 2017, 18, 954-961.	0.6	10
83	Why publish in the <i>American Journal of Physiology-Heart and Circulatory Physiology</i> ?. American Journal of Physiology - Heart and Circulatory Physiology, 2017, 313, H221-H223.	1.5	4
84	Matrix Metalloproteinases in Myocardial Infarction and Heart Failure. Progress in Molecular Biology and Translational Science, 2017, 147, 75-100.	0.9	188
85	Matrix Metalloproteinases in Cardiovascular Diseases. , 2017, , 187-225.		3
86	Periodontal-induced chronic inflammation triggers macrophage secretion of Ccl12 to inhibit fibroblast-mediated cardiac wound healing. JCI Insight, 2017, 2, .	2.3	55
87	Increased ADAMTS1 mediates SPARC-dependent collagen deposition in the aging myocardium. American Journal of Physiology - Endocrinology and Metabolism, 2016, 310, E1027-E1035.	1.8	40
88	Defining the sham environment for post-myocardial infarction studies in mice. American Journal of Physiology - Heart and Circulatory Physiology, 2016, 311, H822-H836.	1.5	27
89	Crossing Into the Next Frontier of Cardiac Extracellular Matrix Research. Circulation Research, 2016, 119, 1040-1045.	2.0	50
90	How to Design a Cardiovascular Proteomics Experiment. , 2016, , 33-57.		2

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91	Synergizing Proteomic and Metabolomic Data to Study Cardiovascular Systems. , 2016, , 365-388.		O
92	MMP-9 signaling in the left ventricle following myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2016, 311, H190-H198.	1.5	92
93	Adapting extracellular matrix proteomics for clinical studies on cardiac remodeling post-myocardial infarction. Clinical Proteomics, 2016, 13, 19.	1.1	31
94	Early matrix metalloproteinase-9 inhibition post-myocardial infarction worsens cardiac dysfunction by delaying inflammation resolution. Journal of Molecular and Cellular Cardiology, 2016, 100, 109-117.	0.9	52
95	Knowledge gaps to understanding cardiac macrophage polarization following myocardial infarction. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2016, 1862, 2288-2292.	1.8	39
96	Clinical and Translational Proteomics Focused on Tissue Damage, Repair, and Regeneration. Proteomics - Clinical Applications, 2016, 10, 6-7.	0.8	0
97	Temporal neutrophil polarization following myocardial infarction. Cardiovascular Research, 2016, 110, 51-61.	1.8	253
98	The crossroads of inflammation, fibrosis, and arrhythmia following myocardial infarction. Journal of Molecular and Cellular Cardiology, 2016, 91, 114-122.	0.9	181
99	Matrix metalloproteinases as input and output signals for post-myocardial infarction remodeling. Journal of Molecular and Cellular Cardiology, 2016, 91, 134-140.	0.9	88
100	CD36 Is a Matrix Metalloproteinase-9 Substrate That Stimulates Neutrophil Apoptosis and Removal During Cardiac Remodeling. Circulation: Cardiovascular Genetics, 2016, 9, 14-25.	5.1	78
101	Myocardial Infarction Superimposed on Aging: MMP-9 Deletion Promotes M2 Macrophage Polarization. Journals of Gerontology - Series A Biological Sciences and Medical Sciences, 2016, 71, 475-483.	1.7	62
102	Systems analysis of gene ontology and biological pathways involved in post-myocardial infarction responses. BMC Genomics, 2015, 16, S18.	1.2	9
103	Plasma Glycoproteomics Reveals Sepsis Outcomes Linked to Distinct Proteins in Common Pathways*. Critical Care Medicine, 2015, 43, 2049-2058.	0.4	46
104	Using the laws of thermodynamics to understand how matrix metalloproteinases coordinate the myocardial response to injury. Metalloproteinases in Medicine, 2015, 2, 75.	1.0	5
105	Building a better infarct: Modulation of collagen cross-linking to increase infarct stiffness and reduce left ventricular dilation post-myocardial infarction. Journal of Molecular and Cellular Cardiology, 2015, 85, 229-239.	0.9	59
106	Osteopontin is proteolytically processed by matrix metalloproteinase 9. Canadian Journal of Physiology and Pharmacology, 2015, 93, 879-886.	0.7	46
107	Cardiac aging: Send in the vinculin reinforcements. Science Translational Medicine, 2015, 7, 292fs26.	5.8	4
108	Deriving a cardiac ageing signature to reveal MMP-9-dependent inflammatory signalling in senescence. Cardiovascular Research, 2015, 106, 421-431.	1.8	79

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109	Syndecan-4: a novel regulator of collagen synthesis and deposition in the pressure-overloaded myocardium. Cardiovascular Research, 2015, 106, 178-179.	1.8	0
110	Early matrix metalloproteinase-12 inhibition worsens post-myocardial infarction cardiac dysfunction by delaying inflammation resolution. International Journal of Cardiology, 2015, 185, 198-208.	0.8	85
111	Harnessing the Heart of Big Data. Circulation Research, 2015, 116, 1115-1119.	2.0	54
112	Matrix Metalloproteinase 9 (MMP-9)., 2015,, 237-259.		1
113	Secreted protein acidic and rich in cysteine facilitates age-related cardiac inflammation and macrophage M1 polarization. American Journal of Physiology - Cell Physiology, 2015, 308, C972-C982.	2.1	46
114	The circular relationship between matrix metalloproteinaseâ€9 and inflammation following myocardial infarction. IUBMB Life, 2015, 67, 611-618.	1.5	38
115	Tissue Inhibitor of Metalloproteinase-1: Actions beyond Matrix Metalloproteinase Inhibition. Cardiology, 2015, 132, 147-150.	0.6	13
116	Transformative Impact of Proteomics on Cardiovascular Health and Disease. Circulation, 2015, 132, 852-872.	1.6	140
117	A Novel Collagen Matricryptin Reduces Left Ventricular Dilation Post-Myocardial Infarction by Promoting Scar Formation and Angiogenesis. Journal of the American College of Cardiology, 2015, 66, 1364-1374.	1.2	145
118	Cross Talk Between Inflammation and Extracellular Matrix Following Myocardial Infarction. , 2015, , 67-79.		9
119	Obesity superimposed on aging magnifies inflammation and delays the resolving response after myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2015, 308, H269-H280.	1.5	70
120	Atherosclerosis exacerbates arrhythmia following myocardial infarction: Role of myocardial inflammation. Heart Rhythm, 2015, 12, 169-178.	0.3	67
121	Translating Koch's Postulates to Identify Matrix Metalloproteinase Roles in Postmyocardial Infarction Remodeling. Circulation Research, 2014, 114, 860-871.	2.0	41
122	Age and SPARC Change the Extracellular Matrix Composition of the Left Ventricle. BioMed Research International, 2014, 2014, 1-7.	0.9	39
123	Integrative Computational and Experimental Approaches to Establish a Post-Myocardial Infarction Knowledge Map. PLoS Computational Biology, 2014, 10, e1003472.	1.5	10
124	Streptococcus pneumoniae Translocates into the Myocardium and Forms Unique Microlesions That Disrupt Cardiac Function. PLoS Pathogens, 2014, 10, e1004383.	2.1	183
125	Citrate Synthase Is a Novel (i>In Vivo (i>Matrix Metalloproteinase-9 Substrate That Regulates Mitochondrial Function in the Postmyocardial Infarction Left Ventricle. Antioxidants and Redox Signaling, 2014, 21, 1974-1985.	2.5	38
126	Artery buckling stimulates cell proliferation and NF-κB signaling. American Journal of Physiology - Heart and Circulatory Physiology, 2014, 307, H542-H551.	1.5	10

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127	Cardiac Assessment in Pediatric Mice: Strain Analysis as a Diagnostic Measurement. Echocardiography, 2014, 31, 375-384.	0.3	9
128	Modifying matrix remodeling to prevent heart failure. , 2014, , 41-60.		2
129	The tell-tale heart: molecular and cellular responses to childhood anthracycline exposure. American Journal of Physiology - Heart and Circulatory Physiology, 2014, 307, H1379-H1389.	1.5	20
130	Using plasma matrix metalloproteinase-9 and monocyte chemoattractant protein-1 to predict future cardiovascular events in subjects with carotid atherosclerosis. Atherosclerosis, 2014, 232, 231-233.	0.4	25
131	Monoamine Oxidase B Prompts Mitochondrial and Cardiac Dysfunction in Pressure Overloaded Hearts. Antioxidants and Redox Signaling, 2014, 20, 267-280.	2.5	135
132	Myofibroblasts and the extracellular matrix network in post-myocardial infarction cardiac remodeling. Pflugers Archiv European Journal of Physiology, 2014, 466, 1113-27.	1.3	94
133	Applications of miRNA Technology for Atherosclerosis. Current Atherosclerosis Reports, 2014, 16, 386.	2.0	37
134	Aliskiren and valsartan mediate left ventricular remodeling post-myocardial infarction in mice through MMP-9 effects. Journal of Molecular and Cellular Cardiology, 2014, 72, 326-335.	0.9	33
135	Negative Elongation Factor Controls Energy Homeostasis in Cardiomyocytes. Cell Reports, 2014, 7, 79-85.	2.9	36
136	Cardiac aging is initiated by matrix metalloproteinase-9-mediated endothelial dysfunction. American Journal of Physiology - Heart and Circulatory Physiology, 2014, 306, H1398-H1407.	1.5	51
137	And the beat goes on: maintained cardiovascular function during aging in the longest-lived rodent, the naked mole-rat. American Journal of Physiology - Heart and Circulatory Physiology, 2014, 307, H284-H291.	1.5	46
138	Caveolin-1 deletion exacerbates cardiac interstitial fibrosis by promoting M2 macrophage activation in mice after myocardial infarction. Journal of Molecular and Cellular Cardiology, 2014, 76, 84-93.	0.9	67
139	Myocardial matrix metalloproteinase-2: inside out and upside down. Journal of Molecular and Cellular Cardiology, 2014, 77, 64-72.	0.9	89
140	Cardiac function of the naked mole-rat: ecophysiological responses to working underground. American Journal of Physiology - Heart and Circulatory Physiology, 2014, 306, H730-H737.	1.5	32
141	Heavy hitting: Using water to label humans. Proteomics - Clinical Applications, 2014, 8, 477-479.	0.8	2
142	Cardiac extracellular proteome profiling and membrane topology analysis using glycoproteomics. Proteomics - Clinical Applications, 2014, 8, 595-602.	0.8	27
143	P. gingivalis lipopolysaccharide intensifies inflammation post-myocardial infarction through matrix metalloproteinase-9. Journal of Molecular and Cellular Cardiology, 2014, 76, 218-226.	0.9	41
144	Using systems biology approaches to understand cardiac inflammation and extracellular matrix remodeling in the setting of myocardial infarction. Wiley Interdisciplinary Reviews: Systems Biology and Medicine, 2014, 6, 77-91.	6.6	14

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145	43Matrix metalloproteinase-9 deletion shifts macrophage polarization towards M2 phenotype in aged left ventricles post-myocardial infarction. Cardiovascular Research, 2014, 103, S6.3-S6.	1.8	2
146	Aging-Related Changes in Extracellular Matrix: Implications for Ventricular Remodeling Following Myocardial Infarction., 2014,, 377-389.		2
147	Obesity superimposed on aging magnifies the inflammatory and plasma lipid mediator responses following myocardial infarction (1155.1). FASEB Journal, 2014, 28, 1155.1.	0.2	0
148	Collagen Câ€peptide roles in postâ€myocardial infarction remodeling (867.15). FASEB Journal, 2014, 28, 867.15.	0.2	0
149	Parameter distribution estimation in first order ODE. , 2013, , .		0
150	Obese and diabetic KKAy mice show increased mortality but improved cardiac function following myocardial infarction. Cardiovascular Pathology, 2013, 22, 481-487.	0.7	14
151	Matrix metalloproteinase (MMP)-9: A proximal biomarker for cardiac remodeling and a distal biomarker for inflammation., 2013, 139, 32-40.		202
152	Matrix Metalloproteinase-28 Deletion Exacerbates Cardiac Dysfunction and Rupture After Myocardial Infarction in Mice by Inhibiting M2 Macrophage Activation. Circulation Research, 2013, 112, 675-688.	2.0	187
153	Texas 3-Step decellularization protocol: Looking at the cardiac extracellular matrix. Journal of Proteomics, 2013, 86, 43-52.	1.2	81
154	Neutrophil roles in left ventricular remodeling following myocardial infarction. Fibrogenesis and Tissue Repair, 2013, 6, 11.	3.4	157
155	Reduced BDNF attenuates inflammation and angiogenesis to improve survival and cardiac function following myocardial infarction in mice. American Journal of Physiology - Heart and Circulatory Physiology, 2013, 305, H1830-H1842.	1.5	62
156	Thrombospondin-1. Circulation Research, 2013, 113, 1272-1274.	2.0	13
157	Heart Failure with Preserved Ejection Fraction. Journal of Cardiovascular Pharmacology, 2013, 62, 13-21.	0.8	46
158	Matrix Metalloproteinase-9: Many Shades of Function in Cardiovascular Disease. Physiology, 2013, 28, 391-403.	1.6	385
159	Left Ventricular Remodeling: One Small Step for the Extracellular Matrix Will Translate to a Giant Leap for the Myocardium. Congestive Heart Failure, 2013, 19, E5-8.	2.0	9
160	A Twoâ€forâ€One Bargain: Using Cilnidipine to Treat Hypertension and Its Comorbidities. Journal of Clinical Hypertension, 2013, 15, 455-457.	1.0	6
161	CirculatingPorphyromonas gingivalislipopolysaccharide resets cardiac homeostasis in mice through a matrix metalloproteinase-9-dependent mechanism. Physiological Reports, 2013, 1, e00079.	0.7	37
162	Using proteomics to uncover extracellular matrix interactions during cardiac remodeling. Proteomics - Clinical Applications, 2013, 7, 516-527.	0.8	23

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163	Matrix Metalloproteinases: Drug Targets for Myocardial Infarction. Current Drug Targets, 2013, 14, 276-286.	1.0	4
164	Extracellular Matrix Biomarkers of Adverse Remodeling After Myocardial Infarction. , 2013, , 383-412.		2
165	Cardiac Wound Healing Post-myocardial Infarction: A Novel Method to Target Extracellular Matrix Remodeling in the Left Ventricle. Methods in Molecular Biology, 2013, 1037, 313-324.	0.4	22
166	Proteomic Analysis of the Left Ventricle Post-myocardial Infarction to Identify In Vivo Candidate Matrix Metalloproteinase Substrates. Methods in Molecular Biology, 2013, 1066, 185-199.	0.4	5
167	Matrix Metalloproteinases: Drug Targets for Myocardial Infarction. Current Drug Targets, 2013, 14, 276-286.	1.0	31
168	MMPâ€9 dependent proteins regulate left ventricular remodeling following myocardial infarction. FASEB Journal, 2013, 27, 1129.4.	0.2	0
169	Reduced BDNF attenuates inflammation and angiogenesis to improve survival and cardiac function following myocardial infarction in mice. FASEB Journal, 2013, 27, 1085.6.	0.2	0
170	Matrix metalloproteinases: drug targets for myocardial infarction. Current Drug Targets, 2013, 14, 276-86.	1.0	34
171	Using Extracellular Matrix Proteomics to Understand Left Ventricular Remodeling. Circulation: Cardiovascular Genetics, 2012, 5, o1-7.	5.1	17
172	A biclustering approach to analyze drug effects on extracellular matrix remodeling post-myocardial infarction. , $2012, \ldots$		4
173	Extracellular Matrix Proteomics in Cardiac Ischemia/Reperfusion. Circulation, 2012, 125, 746-748.	1.6	8
174	Chronic and intermittent hypoxia differentially regulate left ventricular inflammatory and extracellular matrix responses. Hypertension Research, 2012, 35, 811-818.	1.5	25
175	Women are different: the role of coupling factor 6 in blood pressure regulation. Hypertension Research, 2012, 35, 485-486.	1.5	0
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