Merry L Lindsey

List of Publications by Year
in descending order

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278 papers	14,438 citations	16437 64 h-index	24961 109 g-index
281 all docs	281 docs citations	281 times ranked	15410 citing authors

#	Article	IF	Citations
1	Targeted deletion of matrix metalloproteinase-9 attenuates left ventricular enlargement and collagen accumulation after experimental myocardial infarction. Journal of Clinical Investigation, 2000, 106, 55-62.	3.9	724
2	Resident Cardiac Mast Cells Degranulate and Release Preformed TNF-Î±, Initiating the Cytokine Cascade in Experimental Canine Myocardial Ischemia/Reperfusion. Circulation, 1998, 98, 699-710.	1.6	459
3	Matrix Metalloproteinase-9: Many Shades of Function in Cardiovascular Disease. Physiology, 2013, 28, 391-403.	1.6	385
4	Guidelines for experimental models of myocardial ischemia and infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2018, 314, H812-H838.	1.5	372
5	Macrophage roles following myocardial infarction. International Journal of Cardiology, 2008, 130, 147-158.	0.8	302
6	Endothelial Nitric Oxide Synthase Limits Left Ventricular Remodeling After Myocardial Infarction in Mice. Circulation, 2001, 104, 1286-1291.	1.6	282
7	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
8	Cardiac macrophage biology in the steady-state heart, the aging heart, and following myocardial infarction. Translational Research, 2018, 191, 15-28.	2.2	275
9	IL-10 Is Induced in the Reperfused Myocardium and May Modulate the Reaction to Injury. Journal of Immunology, 2000, 165, 2798-2808.	0.4	261
10	Temporal neutrophil polarization following myocardial infarction. Cardiovascular Research, 2016, 110, 51-61.	1.8	253
11	Matrix-Dependent Mechanism of Neutrophil-Mediated Release and Activation of Matrix Metalloproteinase 9 in Myocardial Ischemia/Reperfusion. Circulation, 2001, 103, 2181-2187.	1.6	221
12	Guidelines for measuring cardiac physiology in mice. American Journal of Physiology - Heart and Circulatory Physiology, 2018, 314, H733-H752.	1.5	220
13	Matrix metalloproteinase (MMP)-9: A proximal biomarker for cardiac remodeling and a distal biomarker for inflammation. , 2013, 139, 32-40.		202
14	Mapping macrophage polarization over the myocardial infarction time continuum. Basic Research in Cardiology, 2018, 113, 26.	2.5	189
15	Matrix Metalloproteinases in Myocardial Infarction and Heart Failure. Progress in Molecular Biology and Translational Science, 2017, 147, 75-100.	0.9	188
16	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
17	Streptococcus pneumoniae Translocates into the Myocardium and Forms Unique Microlesions That Disrupt Cardiac Function. PLoS Pathogens, 2014, 10, e1004383.	2.1	183
18	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

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19	The crossroads of inflammation, fibrosis, and arrhythmia following myocardial infarction. Journal of Molecular and Cellular Cardiology, 2016, 91, 114-122.	0.9	181
20	Selective Matrix Metalloproteinase Inhibition Reduces Left Ventricular Remodeling but Does Not Inhibit Angiogenesis After Myocardial Infarction. Circulation, 2002, 105, 753-758.	1.6	180
21	Matrix metalloproteinase-9 gene deletion facilitates angiogenesis after myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2006, 290, H232-H239.	1.5	178
22	Stem Cell Factor Induction Is Associated With Mast Cell Accumulation After Canine Myocardial Ischemia and Reperfusion. Circulation, 1998, 98, 687-698.	1.6	170
23	Cytokines and the Microcirculation in Ischemia and Reperfusion. Journal of Molecular and Cellular Cardiology, 1998, 30, 2567-2576.	0.9	168
24	The impact of aging on cardiac extracellular matrix. GeroScience, 2017, 39, 7-18.	2.1	168
25	Neutrophil roles in left ventricular remodeling following myocardial infarction. Fibrogenesis and Tissue Repair, 2013, 6, 11.	3.4	157
26	Age-dependent changes in myocardial matrix metalloproteinase/tissue inhibitor of metalloproteinase profiles and fibroblast function. Cardiovascular Research, 2005, 66, 410-419.	1.8	151
27	Cardiac Fibroblast Activation Post-Myocardial Infarction: Current Knowledge Gaps. Trends in Pharmacological Sciences, 2017, 38, 448-458.	4.0	151
28	Induction of Monocyte Chemoattractant Protein-1 in the Small Veins of the Ischemic and Reperfused Canine Myocardium. Circulation, 1997, 95, 693-700.	1.6	147
29	Matrix Metalloproteinase-7 Affects Connexin-43 Levels, Electrical Conduction, and Survival After Myocardial Infarction. Circulation, 2006, 113, 2919-2928.	1.6	145
30	Matrix metalloproteinase-9 deletion attenuates myocardial fibrosis and diastolic dysfunction in ageing mice. Cardiovascular Research, 2012, 96, 444-455.	1.8	145
31	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
32	Transformative Impact of Proteomics on Cardiovascular Health and Disease. Circulation, 2015, 132, 852-872.	1.6	140
33	Monoamine Oxidase B Prompts Mitochondrial and Cardiac Dysfunction in Pressure Overloaded Hearts. Antioxidants and Redox Signaling, 2014, 20, 267-280.	2.5	135
34	Deletion of thioredoxin-interacting protein in mice impairs mitochondrial function but protects the myocardium from ischemia-reperfusion injury. Journal of Clinical Investigation, 2012, 122, 267-279.	3.9	135
35	The history of matrix metalloproteinases: milestones, myths, and misperceptions. American Journal of Physiology - Heart and Circulatory Physiology, 2012, 303, H919-H930.	1.5	134
36	Temporal and Spatial Expression of Matrix Metalloproteinases and Tissue Inhibitors of Metalloproteinases Following Myocardial Infarction. Cardiovascular Therapeutics, 2012, 30, 31-41.	1.1	124

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37	Fibroblast polarization over the myocardial infarction time continuum shifts roles from inflammation to angiogenesis. Basic Research in Cardiology, 2019, 114, 6.	2.5	118
38	Extracellular matrix remodeling following myocardial injury. Annals of Medicine, 2003, 35, 316-326.	1.5	117
39	Altered fibroblast function following myocardial infarction. Journal of Molecular and Cellular Cardiology, 2005, 39, 699-707.	0.9	115
40	Understanding cardiac extracellular matrix remodeling to develop biomarkers of myocardial infarction outcomes. Matrix Biology, 2019, 75-76, 43-57.	1.5	106
41	Age-related cardiac muscle sarcopenia: Combining experimental and mathematical modeling to identify mechanisms. Experimental Gerontology, 2008, 43, 296-306.	1.2	99
42	MMP Induction and Inhibition in Myocardial Infarction. Heart Failure Reviews, 2004, 9, 7-19.	1.7	97
43	Extracellular matrix turnover and signaling during cardiac remodeling following MI: Causes and consequences. Journal of Molecular and Cellular Cardiology, 2010, 48, 558-563.	0.9	95
44	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
45	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
46	Î²-Blockade Prevents Sustained Metalloproteinase Activation and Diastolic Stiffening Induced by Angiotensin II Combined With Evolving Cardiac Dysfunction. Circulation Research, 2000, 86, 807-815.	2.0	90
47	Extracellular matrix roles during cardiac repair. Life Sciences, 2010, 87, 391-400.	2.0	89
48	Myocardial matrix metalloproteinase-2: inside out and upside down. Journal of Molecular and Cellular Cardiology, 2014, 77, 64-72.	0.9	89
49	Proteomic analysis identifies in vivo candidate matrix metalloproteinaseâ€9 substrates in the left ventricle postâ€myocardial infarction. Proteomics, 2010, 10, 2214-2223.	1.3	88
50	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
51	Assigning matrix metalloproteinase roles in ischaemic cardiac remodelling. Nature Reviews Cardiology, 2018, 15, 471-479.	6.1	87
52	LXR/RXR signaling and neutrophil phenotype following myocardial infarction classify sex differences in remodeling. Basic Research in Cardiology, 2018, 113, 40.	2.5	86
53	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
54	Texas 3-Step decellularization protocol: Looking at the cardiac extracellular matrix. Journal of Proteomics, 2013, 86, 43-52.	1.2	81

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55	Toll-Like Receptor (TLR) 2 and TLR4 Differentially Regulate Doxorubicin Induced Cardiomyopathy in Mice. PLoS ONE, 2012, 7, e40763.	1.1	81
56	Deriving a cardiac ageing signature to reveal MMP-9-dependent inflammatory signalling in senescence. Cardiovascular Research, 2015, 106, 421-431.	1.8	79
57	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
58	Neutrophil proteome shifts over the myocardial infarction time continuum. Basic Research in Cardiology, 2019, 114, 37.	2.5	78
59	Long-Lived Ames Dwarf Mice Are Resistant to Chemical Stressors. Journals of Gerontology - Series A Biological Sciences and Medical Sciences, 2009, 64A, 819-827.	1.7	75
60	Fibroblasts: The arbiters of extracellular matrix remodeling. Matrix Biology, 2020, 91-92, 1-7.	1.5	75
61	Multi-Analyte Profiling Reveals Matrix Metalloproteinase-9 and Monocyte Chemotactic Protein-1 as Plasma Biomarkers of Cardiac Aging. Circulation: Cardiovascular Genetics, 2011, 4, 455-462.	5.1	71
62	Transgenic overexpression of matrix metalloproteinase-9 in macrophages attenuates the inflammatory response and improves left ventricular function post-myocardial infarction. Journal of Molecular and Cellular Cardiology, 2012, 53, 599-608.	0.9	70
63	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
64	Extracellular Matrix and Fibroblast Communication Following Myocardial Infarction. Journal of Cardiovascular Translational Research, 2012, 5, 848-857.	1.1	68
65	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
66	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
67	Atherosclerosis exacerbates arrhythmia following myocardial infarction: Role of myocardial inflammation. Heart Rhythm, 2015, 12, 169-178.	0.3	67
68	SPARC mediates early extracellular matrix remodeling following myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2011, 301, H497-H505.	1.5	66
69	Mathematical modeling and stability analysis of macrophage activation in left ventricular remodeling post-myocardial infarction. BMC Genomics, 2012, 13, S21.	1.2	62
70	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
71	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
72	Antiarrhythmic effects of interleukin 1 inhibition after myocardial infarction. Heart Rhythm, 2017, 14, 727-736.	0.3	61

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73	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
74	Cardiac fibroblast activation during myocardial infarction wound healing. Matrix Biology, 2020, 91-92, 109-116.	1.5	61
75	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
76	Statistical considerations in reporting cardiovascular research. American Journal of Physiology - Heart and Circulatory Physiology, 2018, 315, H303-H313.	1.5	58
77	Combining experimental and mathematical modeling to reveal mechanisms of macrophage-dependent left ventricular remodeling. BMC Systems Biology, 2011, 5, 60.	3.0	56
78	Matrix Metalloproteinase-28 Deletion Amplifies Inflammatory and Extracellular Matrix Responses to Cardiac Aging. Microscopy and Microanalysis, 2012, 18, 81-90.	0.2	56
79	Effects of Deletion of the Matrix Metalloproteinase 9 Gene on Development of Murine Thoracic Aortic Aneurysms. Circulation, 2005, 112, I242-8.	1.6	55
80	Periodontal-induced chronic inflammation triggers macrophage secretion of Ccl12 to inhibit fibroblast-mediated cardiac wound healing. JCI Insight, 2017, 2, .	2.3	55
81	Harnessing the Heart of Big Data. Circulation Research, 2015, 116, 1115-1119.	2.0	54
82	In vivoMatrix Metalloproteinase-7 Substrates Identified in the Left Ventricle Post-Myocardial Infarction Using Proteomics. Journal of Proteome Research, 2010, 9, 2649-2657.	1.8	53
83	Guidelines for in vivo mouse models of myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2021, 321, H1056-H1073.	1.5	53
84	CC chemokine receptor 5 deletion impairs macrophage activation and induces adverse remodeling following myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2011, 300, H1418-H1426.	1.5	52
85	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
86	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
87	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
88	Caveolin-1 modulates TGF-Î²1 signaling in cardiac remodeling. Matrix Biology, 2011, 30, 318-329.	1.5	50
89	Crossing Into the Next Frontier of Cardiac Extracellular Matrix Research. Circulation Research, 2016, 119, 1040-1045.	2.0	50
90	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

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91	Heart Failure with Preserved Ejection Fraction. Journal of Cardiovascular Pharmacology, 2013, 62, 13-21.	0.8	46
92	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
93	Plasma Glycoproteomics Reveals Sepsis Outcomes Linked to Distinct Proteins in Common Pathways*. Critical Care Medicine, 2015, 43, 2049-2058.	0.4	46
94	Osteopontin is proteolytically processed by matrix metalloproteinase 9. Canadian Journal of Physiology and Pharmacology, 2015, 93, 879-886.	0.7	46
95	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
96	Alterations inÂcultured myocardial fibroblast function following theÂdevelopment ofÂleft ventricular failure. Journal of Molecular and Cellular Cardiology, 2006, 40, 474-483.	0.9	44
97	Walking the Oxidative Stress Tightrope: A Perspective from the Naked Mole-Rat, the Longest-Living Rodent. Current Pharmaceutical Design, 2011, 17, 2290-2307.	0.9	44
98	Neutrophil signaling during myocardial infarction wound repair. Cellular Signalling, 2021, 77, 109816.	1.7	44
99	Translating Kochâ€™s Postulates to Identify Matrix Metalloproteinase Roles in Postmyocardial Infarction Remodeling. Circulation Research, 2014, 114, 860-871.	2.0	41
100	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
101	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
102	Proteomic analysis of the cardiac extracellular matrix: clinical research applications. Expert Review of Proteomics, 2018, 15, 105-112.	1.3	40
103	Bayesian parameter estimation for nonlinear modelling of biological pathways. BMC Systems Biology, 2011, 5, S9.	3.0	39
104	Age and SPARC Change the Extracellular Matrix Composition of the Left Ventricle. BioMed Research International, 2014, 2014, 1-7.	0.9	39
105	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
106	Citrate Synthase Is a NovelIn VivoMatrix Metalloproteinase-9 Substrate That Regulates Mitochondrial Function in the Postmyocardial Infarction Left Ventricle. Antioxidants and Redox Signaling, 2014, 21, 1974-1985.	2.5	38
107	The circular relationship between matrix metalloproteinaseâ€9 and inflammation following myocardial infarction. IUBMB Life, 2015, 67, 611-618.	1.5	38
108	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

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109	CirculatingPorphyromonas gingivalislipopolysaccharide resets cardiac homeostasis in mice through a matrix metalloproteinase-9-dependent mechanism. Physiological Reports, 2013, 1, e00079.	0.7	37
110	Applications of miRNA Technology for Atherosclerosis. Current Atherosclerosis Reports, 2014, 16, 386.	2.0	37
111	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
112	Infarct in the Heart: Whatâ€™s MMP-9 Got to Do with It?. Biomolecules, 2021, 11, 491.	1.8	37
113	Negative Elongation Factor Controls Energy Homeostasis in Cardiomyocytes. Cell Reports, 2014, 7, 79-85.	2.9	36
114	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
115	Effect of a Cleavage-Resistant Collagen Mutation on Left Ventricular Remodeling. Circulation Research, 2003, 93, 238-245.	2.0	35
116	A multidimensional proteomic approach to identify hypertrophy-associated proteins. Proteomics, 2006, 6, 2225-2235.	1.3	35
117	Proteomic analysis reveals late exercise effects on cardiac remodeling following myocardial infarction. Journal of Proteomics, 2010, 73, 2041-2049.	1.2	35
118	Matrix metalloproteinases: drug targets for myocardial infarction. Current Drug Targets, 2013, 14, 276-86.	1.0	34
119	Getting to the Heart of the Matter: Age-related Changes in Diastolic Heart Function in the Longest-lived Rodent, the Naked Mole Rat. Journals of Gerontology - Series A Biological Sciences and Medical Sciences, 2012, 67A, 384-394.	1.7	33
120	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
121	Mechanisms to Inhibit Matrix Metalloproteinase Activity: Where are we in the Development of Clinically Relevant Inhibitors?. Recent Patents on Anti-Cancer Drug Discovery, 2007, 2, 135-142.	0.8	32
122	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
123	The Left Ventricle Proteome Differentiates Middle-Aged and Old Left Ventricles in Mice. Journal of Proteome Research, 2008, 7, 756-765.	1.8	31
124	Adapting extracellular matrix proteomics for clinical studies on cardiac remodeling post-myocardial infarction. Clinical Proteomics, 2016, 13, 19.	1.1	31
125	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
126	Matrix Metalloproteinases: Drug Targets for Myocardial Infarction. Current Drug Targets, 2013, 14, 276-286.	1.0	31

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127	Interleukin 6 induction in the canine myocardium after cardiopulmonary bypass. Journal of Thoracic and Cardiovascular Surgery, 2000, 120, 256-263.	0.4	30
128	Effects of Early and Late Chronic Pressure Overload on Extracellular Matrix Remodeling. Hypertension Research, 2008, 31, 1225-1231.	1.5	29
129	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
130	Extracellular matrix roles in cardiorenal fibrosis: Potential therapeutic targets for CVD and CKD in the elderly. , 2019, 193, 99-120.		28
131	Matrix Metalloproteinase (MMP)-7 Activates MMP-8 But Not MMP-13. Medicinal Chemistry, 2006, 2, 523-526.	0.7	27
132	Effects of surface-modified scaffolds on the growth and differentiation of mouse adipose-derived stromal cells. Journal of Tissue Engineering and Regenerative Medicine, 2007, 1, 211-217.	1.3	27
133	Cardiac extracellular proteome profiling and membrane topology analysis using glycoproteomics. Proteomics - Clinical Applications, 2014, 8, 595-602.	0.8	27
134	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
135	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
136	ACE inhibitors to block MMP-9 activity: New functions for old inhibitors. Journal of Molecular and Cellular Cardiology, 2007, 43, 664-666.	0.9	25
137	Chronic and intermittent hypoxia differentially regulate left ventricular inflammatory and extracellular matrix responses. Hypertension Research, 2012, 35, 811-818.	1.5	25
138	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
139	Using proteomics to uncover extracellular matrix interactions during cardiac remodeling. Proteomics - Clinical Applications, 2013, 7, 516-527.	0.8	23
140	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
141	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
142	Intercellular Adhesion Molecule-1 Regulation In The Canine Lung After Cardiopulmonary Bypass. Journal of Thoracic and Cardiovascular Surgery, 1998, 115, 689-699.	0.4	21
143	A conceptual cellular interaction model of left ventricular remodelling post-MI: dynamic network with exit-entry competition strategy. BMC Systems Biology, 2010, 4, S5.	3.0	21
144	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

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145	Inhibiting Metalloproteases with PD 166793 in Heart Failure: Impact on Cardiac Remodeling and Beyond. Cardiovascular Drug Reviews, 2008, 26, 24-37.	4.4	19
146	Stability analysis of genetic regulatory network with additive noises. BMC Genomics, 2008, 9, S21.	1.2	19
147	Alterations of Pulse Pressure Stimulate Arterial Wall Matrix Remodeling. Journal of Biomechanical Engineering, 2009, 131, 101011.	0.6	19
148	Extracellular matrix in cardiovascular pathophysiology. American Journal of Physiology - Heart and Circulatory Physiology, 2018, 315, H1687-H1690.	1.5	18
149	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
150	Titin Phosphorylation. Circulation Research, 2009, 105, 611-613.	2.0	17
151	Understanding the role of the extracellular matrix in cardiovascular development and disease: Where do we go from here?. Journal of Molecular and Cellular Cardiology, 2010, 48, 431-432.	0.9	17
152	Using Extracellular Matrix Proteomics to Understand Left Ventricular Remodeling. Circulation: Cardiovascular Genetics, 2012, 5, o1-7.	5.1	17
153	Plasma Monitoring of the Myocardial Specific Tissue Inhibitor of Metalloproteinase-4 After Alcohol Septal Ablation in Hypertrophic Obstructive Cardiomyopathy. Journal of Cardiac Failure, 2005, 11, 124-130.	0.7	16
154	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
155	Alterations in Pulse Pressure Affect Artery Function. Cellular and Molecular Bioengineering, 2012, 5, 474-487.	1.0	15
156	Mathematical modeling of left ventricular dimensional changes in mice during aging. BMC Systems Biology, 2012, 6, S10.	3.0	15
157	Dentin Sialoprotein is a Novel Substrate of Matrix Metalloproteinase 9 in vitro and in vivo. Scientific Reports, 2017, 7, 42449.	1.6	15
158	Loss of Arhgef11 in the Dahl Salt-Sensitive Rat Protects Against Hypertension-Induced Renal Injury. Hypertension, 2020, 75, 1012-1024.	1.3	15
159	Chronic Porphyromonas gingivalis 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
160	Obese and diabetic KKAy mice show increased mortality but improved cardiac function following myocardial infarction. Cardiovascular Pathology, 2013, 22, 481-487.	0.7	14
161	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
162	Matrix metalloproteinase-12 as an endogenous resolution promoting factor following myocardial infarction. Pharmacological Research, 2018, 137, 252-258.	3.1	14

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163	The Mouse Heart Attack Research Tool 1.0 database. American Journal of Physiology - Heart and Circulatory Physiology, 2018, 315, H522-H530.	1.5	14
164	Roles of saturated vs. polyunsaturated fat in heart failure survival: not all fats are created equal. Cardiovascular Research, 2012, 93, 4-5.	1.8	13
165	Thrombospondin-1. Circulation Research, 2013, 113, 1272-1274.	2.0	13
166	Tissue Inhibitor of Metalloproteinase-1: Actions beyond Matrix Metalloproteinase Inhibition. Cardiology, 2015, 132, 147-150.	0.6	13
167	Transient ACE (Angiotensin-Converting Enzyme) Inhibition Suppresses Future Fibrogenic Capacity and Heterogeneity of Cardiac Fibroblast Subpopulations. Hypertension, 2021, 77, 904-918.	1.3	13
168	An American Physiological Society cross-journal Call for Papers on â€œInter-Organ Communication in Homeostasis and Diseaseâ€• American Journal of Physiology - Lung Cellular and Molecular Physiology, 2021, 321, L42-L49.	1.3	13
169	Novel Strategies to Delineate Matrix Metalloproteinase (MMP)-Substrate Relationships and Identify Targets to Block MMP Activity. Mini-Reviews in Medicinal Chemistry, 2006, 6, 1243-1248.	1.1	12
170	Understanding the mechanisms that determine extracellular matrix remodeling in the infarcted myocardium. Biochemical Society Transactions, 2019, 47, 1679-1687.	1.6	12
171	DHA derivatives of fish oil as dietary supplements: a nutrition-based drug discovery approach for therapies to prevent metabolic cardiotoxicity. Expert Opinion on Drug Discovery, 2012, 7, 711-721.	2.5	11
172	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
173	Estrogen effects on MMP-13 and MMP-14 regulation of left ventricular mass in Dahl salt-induced hypertension. Gender Medicine, 2008, 5, 74-85.	1.4	10
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