

Merry L Lindsey

List of Publications by Year in Descending Order

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The third column is the impact factor (IF) of the journal, and the fourth column is the number of citations of the article.

240
papers

10,827
citations

59
h-index

96
g-index

281
ext. papers

12,844
ext. citations

5.9
avg, IF

6.5
L-index

#	Paper	IF	Citations
240	Faster skin wound healing predicts survival after myocardial infarction.. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2022 ,	5.2	1
239	Macrophages secrete murinoglobulin-1 and galectin-3 to regulate neutrophil degranulation after myocardial infarction.. <i>Molecular Omics</i> , 2022 ,	4.4	1
238	S100A9 is a functional effector of infarct wall thinning after myocardial infarction. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2021 ,	5.2	2
237	Guidelines for in vivo mouse models of myocardial infarction. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2021 , 321, H1056-H1073	5.2	7
236	Infarct in the Heart: What's MMP-9 Got to Do with It?. <i>Biomolecules</i> , 2021 , 11,	5.9	9
235	Transient ACE (Angiotensin-Converting Enzyme) Inhibition Suppresses Future Fibrogenic Capacity and Heterogeneity of Cardiac Fibroblast Subpopulations. <i>Hypertension</i> , 2021 , 77, 904-918	8.5	3
234	Dysbiosis and Intestinal Barrier Dysfunction in Pediatric Congenital Heart Disease Is Exacerbated Following Cardiopulmonary Bypass. <i>JACC Basic To Translational Science</i> , 2021 , 6, 311-327	8.7	3
233	Neutrophil signaling during myocardial infarction wound repair. <i>Cellular Signalling</i> , 2021 , 77, 109816	4.9	15
232	CD4 T Cell-Specific Proteomic Pathways Identified in Progression of Hypertension Across Postmenopausal Transition. <i>Journal of the American Heart Association</i> , 2021 , 10, e018038	6	2
231	Network Analysis Reveals a Distinct Axis of Macrophage Activation in Response to Conflicting Inflammatory Cues. <i>Journal of Immunology</i> , 2021 , 206, 883-891	5.3	11
230	Effect of genetic depletion of MMP-9 on neurological manifestations of hypertension-induced intracerebral hemorrhages in aged mice. <i>GeroScience</i> , 2021 , 43, 2611-2619	8.9	1
229	The HEART Camp Exercise Intervention Improves Exercise Adherence, Physical Function, and Patient-Reported Outcomes in Adults With Preserved Ejection Fraction Heart Failure. <i>Journal of Cardiac Failure</i> , 2021 ,	3.3	1
228	Chronic lipopolysaccharide induces adverse myocardial infarction wound healing through activation of CD8 T cells. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2021 , 321, H948-H962	5.2	4
227	Exogenous IL-4 shuts off pro-inflammation in neutrophils while stimulating anti-inflammation in macrophages to induce neutrophil phagocytosis following myocardial infarction. <i>Journal of Molecular and Cellular Cardiology</i> , 2020 , 145, 112-121	5.8	12
226	Fibroblasts: The arbiters of extracellular matrix remodeling. <i>Matrix Biology</i> , 2020 , 91-92, 1-7	11.4	36
225	Loss of in the Dahl Salt-Sensitive Rat Protects Against Hypertension-Induced Renal Injury. <i>Hypertension</i> , 2020 , 75, 1012-1024	8.5	5
224	The compendium of matrix metalloproteinase expression in the left ventricle of mice following myocardial infarction. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2020 , 318, H706-H714	5.2	7

223	COVID-19 and cardiovascular disease: What we know, what we think we know, and what we need to know. <i>Journal of Molecular and Cellular Cardiology</i> , 2020 , 144, 12-14	5.8	4
222	Exogenous IL-4 Promotes Myocardial Infarction Repair by Turning off Pro-Inflammation in Neutrophils while Stimulating Anti-Inflammation in Macrophages to Induce Neutrophil Phagocytosis. <i>FASEB Journal</i> , 2020 , 34, 1-1	0.9	
221	Focusing Heart Failure Research on Myocardial Fibrosis to Prioritize Translation. <i>Journal of Cardiac Failure</i> , 2020 , 26, 876-884	3.3	3
220	Cardiac fibroblast activation during myocardial infarction wound healing: Fibroblast polarization after MI. <i>Matrix Biology</i> , 2020 , 91-92, 109-116	11.4	23
219	Somewhere over the sex differences rainbow of myocardial infarction remodeling: hormones, chromosomes, inflammasome, oh my. <i>Expert Review of Proteomics</i> , 2019 , 16, 933-940	4.2	3
218	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. <i>European Journal of Heart Failure</i> , 2019 , 21, 272-285	12.3	99
217	Common pathways and communication between the brain and heart: connecting post-traumatic stress disorder and heart failure. <i>Stress</i> , 2019 , 22, 530-547	3	11
216	Menopause and FOXP3 Treg cell depletion eliminate female protection against T cell-mediated angiotensin II hypertension. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2019 , 317, H415-H423	5.2	15
215	Extracellular matrix roles in cardiorenal fibrosis: Potential therapeutic targets for CVD and CKD in the elderly. <i>Pharmacology & Therapeutics</i> , 2019 , 193, 99-120	13.9	20
214	Neutrophil proteome shifts over the myocardial infarction time continuum. <i>Basic Research in Cardiology</i> , 2019 , 114, 37	11.8	41
213	Physiological Omics Identifies Mechanisms that Attenuate Renal Injury and Blood Pressure in Dahl salt-sensitive Arhgef11 ^{+/+} Rats. <i>FASEB Journal</i> , 2019 , 33, 571.1	0.9	
212	Myocardial infarction-relevant MMP-12 interactions identified by correlation analysis. <i>FASEB Journal</i> , 2019 , 33, 530.2	0.9	
211	Mapping neutrophil polarization over the myocardial infarction time continuum. <i>FASEB Journal</i> , 2019 , 33, 690.12	0.9	
210	Understanding the mechanisms that determine extracellular matrix remodeling in the infarcted myocardium. <i>Biochemical Society Transactions</i> , 2019 , 47, 1679-1687	5.1	4
209	Exogenous CXCL4 infusion inhibits macrophage phagocytosis by limiting CD36 signalling to enhance post-myocardial infarction cardiac dilation and mortality. <i>Cardiovascular Research</i> , 2019 , 115, 395-408	9.9	18
208	Identifying the molecular and cellular signature of cardiac dilation following myocardial infarction. <i>Biochimica Et Biophysica Acta - Molecular Basis of Disease</i> , 2019 , 1865, 1845-1852	6.9	3
207	Using Peptidomics to Identify Extracellular Matrix-Derived Peptides as Novel Therapeutics for Cardiac Disease. <i>Molecular and Translational Medicine</i> , 2019 , 349-365	0.4	
206	Matrix Metalloproteinase-9-Dependent Mechanisms of Reduced Contractility and Increased Stiffness in the Aging Heart. <i>Molecular and Translational Medicine</i> , 2019 , 335-347	0.4	1

205	Fibroblast polarization over the myocardial infarction time continuum shifts roles from inflammation to angiogenesis. <i>Basic Research in Cardiology</i> , 2019 , 114, 6	11.8	72
204	Glycoproteomic Profiling Provides Candidate Myocardial Infarction Predictors of Later Progression to Heart Failure. <i>ACS Omega</i> , 2019 , 4, 1272-1280	3.9	5
203	Understanding cardiac extracellular matrix remodeling to develop biomarkers of myocardial infarction outcomes. <i>Matrix Biology</i> , 2019 , 75-76, 43-57	11.4	64
202	Physiological proteomics of heart failure. <i>Current Opinion in Physiology</i> , 2018 , 1, 185-197	2.6	1
201	Proteomic analysis of the cardiac extracellular matrix: clinical research applications. <i>Expert Review of Proteomics</i> , 2018 , 15, 105-112	4.2	21
200	Macrophage overexpression of matrix metalloproteinase-9 in aged mice improves diastolic physiology and cardiac wound healing after myocardial infarction. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2018 , 314, H224-H235	5.2	27
199	Guidelines for experimental models of myocardial ischemia and infarction. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2018 , 314, H812-H838	5.2	249
198	Statistical considerations in reporting cardiovascular research. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2018 , 315, H303-H313	5.2	45
197	Myocardial infarction remodeling that progresses to heart failure: a signaling misunderstanding. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2018 , 315, H71-H79	5.2	39
196	Guidelines for measuring cardiac physiology in mice. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2018 , 314, H733-H752	5.2	137
195	Assigning matrix metalloproteinase roles in ischaemic cardiac remodelling. <i>Nature Reviews Cardiology</i> , 2018 , 15, 471-479	14.8	58
194	LXR/RXR signaling and neutrophil phenotype following myocardial infarction classify sex differences in remodeling. <i>Basic Research in Cardiology</i> , 2018 , 113, 40	11.8	64
193	Mapping macrophage polarization over the myocardial infarction time continuum. <i>Basic Research in Cardiology</i> , 2018 , 113, 26	11.8	120
192	The Mouse Heart Attack Research Tool (mHART) 1.0 Database. <i>FASEB Journal</i> , 2018 , 32, 848.5	0.9	
191	CD8 T-cells have a biphasic role during post-myocardial infarction cardiac remodeling. <i>FASEB Journal</i> , 2018 , 32, 718.5	0.9	
190	Day 1 Post-Myocardial Infarction Cardiac Macrophage Transcriptomic Signatures that Link to LV Infarct Wall Thinning. <i>FASEB Journal</i> , 2018 , 32, 717.11	0.9	
189	Cardiac macrophage biology in the steady-state heart, the aging heart, and following myocardial infarction. <i>Translational Research</i> , 2018 , 191, 15-28	11	153
188	Extracellular matrix in cardiovascular pathophysiology. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2018 , 315, H1687-H1690	5.2	7

187	Guidelines for authors and reviewers on antibody use in physiology studies. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2018 , 314, H724-H732	5.2	46
186	Death of an antioxidant brings heart failure with preserved ejection fraction to life: 5-oxoproline and post-ischaemic cardio-renal dysfunction. <i>Cardiovascular Research</i> , 2018 , 114, 1819-1821	9.9	4
185	Matrix metalloproteinase-12 as an endogenous resolution promoting factor following myocardial infarction. <i>Pharmacological Research</i> , 2018 , 137, 252-258	10.2	7
184	The Mouse Heart Attack Research Tool 1.0 database. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2018 , 315, H522-H530	5.2	11
183	Antiarrhythmic effects of interleukin 1 inhibition after myocardial infarction. <i>Heart Rhythm</i> , 2017 , 14, 727-736	6.7	40
182	The impact of aging on cardiac extracellular matrix. <i>GeroScience</i> , 2017 , 39, 7-18	8.9	109
181	Glucose regulates the intrinsic inflammatory response of the heart to surgically induced hypothermic ischemic arrest and reperfusion. <i>Physiological Genomics</i> , 2017 , 49, 37-52	3.6	6
180	Transgenic overexpression of macrophage matrix metalloproteinase-9 exacerbates age-related cardiac hypertrophy, vessel rarefaction, inflammation, and fibrosis. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2017 , 312, H375-H383	5.2	40
179	Dentin Sialoprotein is a Novel Substrate of Matrix Metalloproteinase 9 in vitro and in vivo. <i>Scientific Reports</i> , 2017 , 7, 42449	4.9	7
178	IL-10 improves cardiac remodeling after myocardial infarction by stimulating M2 macrophage polarization and fibroblast activation. <i>Basic Research in Cardiology</i> , 2017 , 112, 33	11.8	172
177	Cardiac Fibroblast Activation Post-Myocardial Infarction: Current Knowledge Gaps. <i>Trends in Pharmacological Sciences</i> , 2017 , 38, 448-458	13.2	94
176	Elevated serum osteoprotegerin is associated with increased left ventricular mass index and myocardial stiffness. <i>Journal of Cardiovascular Medicine</i> , 2017 , 18, 954-961	1.9	3
175	Matrix Metalloproteinases in Myocardial Infarction and Heart Failure. <i>Progress in Molecular Biology and Translational Science</i> , 2017 , 147, 75-100	4	116
174	Matrix Metalloproteinases in Cardiovascular Diseases 2017 , 187-225		2
173	Periodontal-induced chronic inflammation triggers macrophage secretion of Ccl12 to inhibit fibroblast-mediated cardiac wound healing. <i>JCI Insight</i> , 2017 , 2,	9.9	45
172	Myocardial Infarction Superimposed on Aging: MMP-9 Deletion Promotes M2 Macrophage Polarization. <i>Journals of Gerontology - Series A Biological Sciences and Medical Sciences</i> , 2016 , 71, 475-83	6.4	53
171	MMP-9 signaling in the left ventricle following myocardial infarction. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2016 , 311, H190-8	5.2	64
170	Adapting extracellular matrix proteomics for clinical studies on cardiac remodeling post-myocardial infarction. <i>Clinical Proteomics</i> , 2016 , 13, 19	5	25

169	Early matrix metalloproteinase-9 inhibition post-myocardial infarction worsens cardiac dysfunction by delaying inflammation resolution. <i>Journal of Molecular and Cellular Cardiology</i> , 2016 , 100, 109-117	5.8	42
168	Knowledge gaps to understanding cardiac macrophage polarization following myocardial infarction. <i>Biochimica Et Biophysica Acta - Molecular Basis of Disease</i> , 2016 , 1862, 2288-2292	6.9	31
167	Temporal neutrophil polarization following myocardial infarction. <i>Cardiovascular Research</i> , 2016 , 110, 51-61	9.9	177
166	The crossroads of inflammation, fibrosis, and arrhythmia following myocardial infarction. <i>Journal of Molecular and Cellular Cardiology</i> , 2016 , 91, 114-22	5.8	116
165	Matrix metalloproteinases as input and output signals for post-myocardial infarction remodeling. <i>Journal of Molecular and Cellular Cardiology</i> , 2016 , 91, 134-40	5.8	67
164	CD36 Is a Matrix Metalloproteinase-9 Substrate That Stimulates Neutrophil Apoptosis and Removal During Cardiac Remodeling. <i>Circulation: Cardiovascular Genetics</i> , 2016 , 9, 14-25		61
163	Increased ADAMTS1 mediates SPARC-dependent collagen deposition in the aging myocardium. <i>American Journal of Physiology - Endocrinology and Metabolism</i> , 2016 , 310, E1027-35	6	29
162	Defining the sham environment for post-myocardial infarction studies in mice. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2016 , 311, H822-36	5.2	24
161	How to Design a Cardiovascular Proteomics Experiment 2016 , 33-57		2
160	Synergizing Proteomic and Metabolomic Data to Study Cardiovascular Systems 2016 , 365-388		
159	Building a better infarct: Modulation of collagen cross-linking to increase infarct stiffness and reduce left ventricular dilation post-myocardial infarction. <i>Journal of Molecular and Cellular Cardiology</i> , 2015 , 85, 229-39	5.8	52
158	Osteopontin is proteolytically processed by matrix metalloproteinase 9. <i>Canadian Journal of Physiology and Pharmacology</i> , 2015 , 93, 879-86	2.4	32
157	Cardiac aging: Send in the vinculin reinforcements. <i>Science Translational Medicine</i> , 2015 , 7, 292fs26	17.5	4
156	Deriving a cardiac ageing signature to reveal MMP-9-dependent inflammatory signalling in senescence. <i>Cardiovascular Research</i> , 2015 , 106, 421-31	9.9	61
155	Early matrix metalloproteinase-12 inhibition worsens post-myocardial infarction cardiac dysfunction by delaying inflammation resolution. <i>International Journal of Cardiology</i> , 2015 , 185, 198-208 ^{3.2}		66
154	Harnessing the heart of big data. <i>Circulation Research</i> , 2015 , 116, 1115-9	15.7	47
153	Matrix Metalloproteinase 9 (MMP-9) 2015 , 237-259		1
152	Secreted protein acidic and rich in cysteine facilitates age-related cardiac inflammation and macrophage M1 polarization. <i>American Journal of Physiology - Cell Physiology</i> , 2015 , 308, C972-82	5.4	34

151	The circular relationship between matrix metalloproteinase-9 and inflammation following myocardial infarction. <i>IUBMB Life</i> , 2015 , 67, 611-8	4.7	30
150	Tissue Inhibitor of Metalloproteinase-1: Actions beyond Matrix Metalloproteinase Inhibition. <i>Cardiology</i> , 2015 , 132, 147-50	1.6	12
149	Transformative Impact of Proteomics on Cardiovascular Health and Disease: A Scientific Statement From the American Heart Association. <i>Circulation</i> , 2015 , 132, 852-72	16.7	112
148	A Novel Collagen Matricryptin Reduces Left Ventricular Dilation Post-Myocardial Infarction by Promoting Scar Formation and Angiogenesis. <i>Journal of the American College of Cardiology</i> , 2015 , 66, 1364-74	15.1	101
147	Cross Talk Between Inflammation and Extracellular Matrix Following Myocardial Infarction 2015 , 67-79		2
146	Obesity superimposed on aging magnifies inflammation and delays the resolving response after myocardial infarction. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2015 , 308, H269-80	5.2	59
145	Atherosclerosis exacerbates arrhythmia following myocardial infarction: Role of myocardial inflammation. <i>Heart Rhythm</i> , 2015 , 12, 169-78	6.7	50
144	Systems analysis of gene ontology and biological pathways involved in post-myocardial infarction responses. <i>BMC Genomics</i> , 2015 , 16 Suppl 7, S18	4.5	7
143	Plasma Glycoproteomics Reveals Sepsis Outcomes Linked to Distinct Proteins in Common Pathways. <i>Critical Care Medicine</i> , 2015 , 43, 2049-2058	1.4	34
142	Using the laws of thermodynamics to understand how matrix metalloproteinases coordinate the myocardial response to injury. <i>Metalloproteinases in Medicine</i> , 2015 , 2, 75-82	0.7	4
141	Myofibroblasts and the extracellular matrix network in post-myocardial infarction cardiac remodeling. <i>Pflugers Archiv European Journal of Physiology</i> , 2014 , 466, 1113-27	4.6	70
140	Applications of miRNA technology for atherosclerosis. <i>Current Atherosclerosis Reports</i> , 2014 , 16, 386	6	26
139	Aliskiren and valsartan mediate left ventricular remodeling post-myocardial infarction in mice through MMP-9 effects. <i>Journal of Molecular and Cellular Cardiology</i> , 2014 , 72, 326-35	5.8	30
138	Negative elongation factor controls energy homeostasis in cardiomyocytes. <i>Cell Reports</i> , 2014 , 7, 79-85	10.6	17
137	Cardiac aging is initiated by matrix metalloproteinase-9-mediated endothelial dysfunction. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2014 , 306, H1398-407	5.2	44
136	And the beat goes on: maintained cardiovascular function during aging in the longest-lived rodent, the naked mole-rat. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2014 , 307, H284-91	5.2	39
135	Caveolin-1 deletion exacerbates cardiac interstitial fibrosis by promoting M2 macrophage activation in mice after myocardial infarction. <i>Journal of Molecular and Cellular Cardiology</i> , 2014 , 76, 84-93	5.8	56
134	Myocardial matrix metalloproteinase-2: inside out and upside down. <i>Journal of Molecular and Cellular Cardiology</i> , 2014 , 77, 64-72	5.8	63

133	Cardiac function of the naked mole-rat: ecophysiological responses to working underground. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2014 , 306, H730-7	5.2	23
132	Heavy hitting: Using water to label humans. <i>Proteomics - Clinical Applications</i> , 2014 , 8, 477-9	3.1	1
131	Cardiac extracellular proteome profiling and membrane topology analysis using glycoproteomics. <i>Proteomics - Clinical Applications</i> , 2014 , 8, 595-602	3.1	23
130	<i>P. gingivalis</i> lipopolysaccharide intensifies inflammation post-myocardial infarction through matrix metalloproteinase-9. <i>Journal of Molecular and Cellular Cardiology</i> , 2014 , 76, 218-26	5.8	34
129	Using systems biology approaches to understand cardiac inflammation and extracellular matrix remodeling in the setting of myocardial infarction. <i>Wiley Interdisciplinary Reviews: Systems Biology and Medicine</i> , 2014 , 6, 77-91	6.6	12
128	43Matrix metalloproteinase-9 deletion shifts macrophage polarization towards M2 phenotype in aged left ventricles post-myocardial infarction. <i>Cardiovascular Research</i> , 2014 , 103, S6.3-S6	9.9	2
127	Translating Koch's postulates to identify matrix metalloproteinase roles in postmyocardial infarction remodeling: cardiac metalloproteinase actions (CarMA) postulates. <i>Circulation Research</i> , 2014 , 114, 860-71	15.7	32
126	Age and SPARC change the extracellular matrix composition of the left ventricle. <i>BioMed Research International</i> , 2014 , 2014, 810562	3	33
125	Integrative computational and experimental approaches to establish a post-myocardial infarction knowledge map. <i>PLoS Computational Biology</i> , 2014 , 10, e1003472	5	9
124	<i>Streptococcus pneumoniae</i> translocates into the myocardium and forms unique microlesions that disrupt cardiac function. <i>PLoS Pathogens</i> , 2014 , 10, e1004383	7.6	119
123	Citrate synthase is a novel in vivo matrix metalloproteinase-9 substrate that regulates mitochondrial function in the postmyocardial infarction left ventricle. <i>Antioxidants and Redox Signaling</i> , 2014 , 21, 1974-85	8.4	29
122	Artery buckling stimulates cell proliferation and NF- κ B signaling. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2014 , 307, H542-51	5.2	10
121	Cardiac assessment in pediatric mice: strain analysis as a diagnostic measurement. <i>Echocardiography</i> , 2014 , 31, 375-84	1.5	7
120	Modifying matrix remodeling to prevent heart failure 2014 , 41-60		1
119	The tell-tale heart: molecular and cellular responses to childhood anthracycline exposure. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2014 , 307, H1379-89	5.2	16
118	Using plasma matrix metalloproteinase-9 and monocyte chemoattractant protein-1 to predict future cardiovascular events in subjects with carotid atherosclerosis. <i>Atherosclerosis</i> , 2014 , 232, 231-3	3.1	23
117	Monoamine oxidase B prompts mitochondrial and cardiac dysfunction in pressure overloaded hearts. <i>Antioxidants and Redox Signaling</i> , 2014 , 20, 267-80	8.4	112
116	Aging-Related Changes in Extracellular Matrix: Implications for Ventricular Remodeling Following Myocardial Infarction 2014 , 377-389		2

115	Obesity superimposed on aging magnifies the inflammatory and plasma lipid mediator responses following myocardial infarction (1155.1). <i>FASEB Journal</i> , 2014 , 28, 1155.1	0.9	
114	Collagen C-peptide roles in post-myocardial infarction remodeling (867.15). <i>FASEB Journal</i> , 2014 , 28, 867.15	0.9	
113	Obese and diabetic KKAY mice show increased mortality but improved cardiac function following myocardial infarction. <i>Cardiovascular Pathology</i> , 2013 , 22, 481-7	3.8	10
112	Matrix metalloproteinase (MMP)-9: a proximal biomarker for cardiac remodeling and a distal biomarker for inflammation. <i>Pharmacology & Therapeutics</i> , 2013 , 139, 32-40	13.9	152
111	Matrix metalloproteinase-28 deletion exacerbates cardiac dysfunction and rupture after myocardial infarction in mice by inhibiting M2 macrophage activation. <i>Circulation Research</i> , 2013 , 112, 675-88	15.7	150
110	Texas 3-step decellularization protocol: looking at the cardiac extracellular matrix. <i>Journal of Proteomics</i> , 2013 , 86, 43-52	3.9	62
109	Neutrophil roles in left ventricular remodeling following myocardial infarction. <i>Fibrogenesis and Tissue Repair</i> , 2013 , 6, 11		113
108	Reduced BDNF attenuates inflammation and angiogenesis to improve survival and cardiac function following myocardial infarction in mice. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2013 , 305, H1830-42	5.2	47
107	Heart failure with preserved ejection fraction: emerging drug strategies. <i>Journal of Cardiovascular Pharmacology</i> , 2013 , 62, 13-21	3.1	39
106	Matrix metalloproteinase-9: Many shades of function in cardiovascular disease. <i>Physiology</i> , 2013 , 28, 391-403	9.8	248
105	Left ventricular remodeling: one small step for the extracellular matrix will translate to a giant leap for the myocardium. <i>Congestive Heart Failure</i> , 2013 , 19, E5-8		7
104	A two-for-one bargain: using cilnidipine to treat hypertension and its comorbidities. <i>Journal of Clinical Hypertension</i> , 2013 , 15, 455-7	2.3	4
103	Circulating Porphyromonas gingivalis lipopolysaccharide resets cardiac homeostasis in mice through a matrix metalloproteinase-9-dependent mechanism. <i>Physiological Reports</i> , 2013 , 1, e00079	2.6	32
102	Using proteomics to uncover extracellular matrix interactions during cardiac remodeling. <i>Proteomics - Clinical Applications</i> , 2013 , 7, 516-27	3.1	18
101	Matrix Metalloproteinases: Drug Targets for Myocardial Infarction. <i>Current Drug Targets</i> , 2013 , 14, 276-286		4
100	Matrix metalloproteinases: drug targets for myocardial infarction. <i>Current Drug Targets</i> , 2013 , 14, 276-86		31
99	Matrix Metalloproteinases: Drug Targets for Myocardial Infarction. <i>Current Drug Targets</i> , 2013 , 14, 276-286		26
98	Cardiac wound healing post-myocardial infarction: a novel method to target extracellular matrix remodeling in the left ventricle. <i>Methods in Molecular Biology</i> , 2013 , 1037, 313-24	1.4	18

97	Proteomic analysis of the left ventricle post-myocardial infarction to identify in vivo candidate matrix metalloproteinase substrates. <i>Methods in Molecular Biology</i> , 2013 , 1066, 185-99	1.4	3
96	MMP-9 dependent proteins regulate left ventricular remodeling following myocardial infarction. <i>FASEB Journal</i> , 2013 , 27, 1129.4	0.9	
95	Reduced BDNF attenuates inflammation and angiogenesis to improve survival and cardiac function following myocardial infarction in mice. <i>FASEB Journal</i> , 2013 , 27, 1085.6	0.9	
94	Extracellular Matrix Biomarkers of Adverse Remodeling After Myocardial Infarction 2013 , 383-412		1
93	Temporal and spatial expression of matrix metalloproteinases and tissue inhibitors of metalloproteinases following myocardial infarction. <i>Cardiovascular Therapeutics</i> , 2012 , 30, 31-41	3.3	100
92	Transgenic overexpression of matrix metalloproteinase-9 in macrophages attenuates the inflammatory response and improves left ventricular function post-myocardial infarction. <i>Journal of Molecular and Cellular Cardiology</i> , 2012 , 53, 599-608	5.8	60
91	The history of matrix metalloproteinases: milestones, myths, and misperceptions. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2012 , 303, H919-30	5.2	101
90	Getting to the heart of the matter: age-related changes in diastolic heart function in the longest-lived rodent, the naked mole rat. <i>Journals of Gerontology - Series A Biological Sciences and Medical Sciences</i> , 2012 , 67, 384-94	6.4	28
89	Is isolated systolic hypertension worse than combined systolic/diastolic hypertension?. <i>Journal of Clinical Hypertension</i> , 2012 , 14, 808-9	2.3	5
88	Extracellular matrix and fibroblast communication following myocardial infarction. <i>Journal of Cardiovascular Translational Research</i> , 2012 , 5, 848-57	3.3	55
87	Alterations in Pulse Pressure Affect Artery Function. <i>Cellular and Molecular Bioengineering</i> , 2012 , 5, 474-487	4.9	13
86	DHA derivatives of fish oil as dietary supplements: a nutrition-based drug discovery approach for therapies to prevent metabolic cardiotoxicity. <i>Expert Opinion on Drug Discovery</i> , 2012 , 7, 711-21	6.2	10
85	Mathematical modeling and stability analysis of macrophage activation in left ventricular remodeling post-myocardial infarction. <i>BMC Genomics</i> , 2012 , 13 Suppl 6, S21	4.5	38
84	Mathematical modeling of left ventricular dimensional changes in mice during aging. <i>BMC Systems Biology</i> , 2012 , 6 Suppl 3, S10	3.5	11
83	Using extracellular matrix proteomics to understand left ventricular remodeling. <i>Circulation: Cardiovascular Genetics</i> , 2012 , 5, 01-7		13
82	A biclustering approach to analyze drug effects on extracellular matrix remodeling post-myocardial infarction 2012 ,		4
81	Chronic and intermittent hypoxia differentially regulate left ventricular inflammatory and extracellular matrix responses. <i>Hypertension Research</i> , 2012 , 35, 811-8	4.7	18
80	Women are different: the role of coupling factor 6 in blood pressure regulation. <i>Hypertension Research</i> , 2012 , 35, 485-6	4.7	

79	Plasma fractionation enriches post-myocardial infarction samples prior to proteomics analysis. <i>International Journal of Proteomics</i> , 2012 , 2012, 397103		5
78	Matrix metalloproteinase-9 deletion attenuates myocardial fibrosis and diastolic dysfunction in ageing mice. <i>Cardiovascular Research</i> , 2012 , 96, 444-55	9.9	118
77	Matrix metalloproteinase-28 deletion amplifies inflammatory and extracellular matrix responses to cardiac aging. <i>Microscopy and Microanalysis</i> , 2012 , 18, 81-90	0.5	47
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