

Merry L Lindsey

List of Publications by Citations

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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 | Targeted deletion of matrix metalloproteinase-9 attenuates left ventricular enlargement and collagen accumulation after experimental myocardial infarction. <i>Journal of Clinical Investigation</i> , 2000 , 106, 55-62 | 15.9 | 626 |
| 239 | Resident cardiac mast cells degranulate and release preformed TNF-alpha, initiating the cytokine cascade in experimental canine myocardial ischemia/reperfusion. <i>Circulation</i> , 1998 , 98, 699-710 | 16.7 | 393 |
| 238 | Endothelial nitric oxide synthase limits left ventricular remodeling after myocardial infarction in mice. <i>Circulation</i> , 2001 , 104, 1286-91 | 16.7 | 263 |
| 237 | Macrophage roles following myocardial infarction. <i>International Journal of Cardiology</i> , 2008 , 130, 147-58 | 3.2 | 258 |
| 236 | 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 |
| 235 | Matrix metalloproteinase-9: Many shades of function in cardiovascular disease. <i>Physiology</i> , 2013 , 28, 391-403 | 9.8 | 248 |
| 234 | IL-10 is induced in the reperfused myocardium and may modulate the reaction to injury. <i>Journal of Immunology</i> , 2000 , 165, 2798-808 | 5.3 | 230 |
| 233 | Matrix-dependent mechanism of neutrophil-mediated release and activation of matrix metalloproteinase 9 in myocardial ischemia/reperfusion. <i>Circulation</i> , 2001 , 103, 2181-7 | 16.7 | 194 |
| 232 | Temporal neutrophil polarization following myocardial infarction. <i>Cardiovascular Research</i> , 2016 , 110, 51-61 | 9.9 | 177 |
| 231 | 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 |
| 230 | Selective matrix metalloproteinase inhibition reduces left ventricular remodeling but does not inhibit angiogenesis after myocardial infarction. <i>Circulation</i> , 2002 , 105, 753-8 | 16.7 | 168 |
| 229 | Matrix metalloproteinase-9 gene deletion facilitates angiogenesis after myocardial infarction. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2006 , 290, H232-9 | 5.2 | 162 |
| 228 | Stem cell factor induction is associated with mast cell accumulation after canine myocardial ischemia and reperfusion. <i>Circulation</i> , 1998 , 98, 687-98 | 16.7 | 153 |
| 227 | 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 |
| 226 | 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 |
| 225 | 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 |
| 224 | Cytokines and the microcirculation in ischemia and reperfusion. <i>Journal of Molecular and Cellular Cardiology</i> , 1998 , 30, 2567-76 | 5.8 | 150 |

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| 223 | Guidelines for measuring cardiac physiology in mice. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2018 , 314, H733-H752 | 5.2 | 137 |
| 222 | Age-dependent changes in myocardial matrix metalloproteinase/tissue inhibitor of metalloproteinase profiles and fibroblast function. <i>Cardiovascular Research</i> , 2005 , 66, 410-9 | 9.9 | 131 |
| 221 | Induction of monocyte chemoattractant protein-1 in the small veins of the ischemic and reperfused canine myocardium. <i>Circulation</i> , 1997 , 95, 693-700 | 16.7 | 129 |
| 220 | Matrix metalloproteinase-7 affects connexin-43 levels, electrical conduction, and survival after myocardial infarction. <i>Circulation</i> , 2006 , 113, 2919-28 | 16.7 | 121 |
| 219 | Mapping macrophage polarization over the myocardial infarction time continuum. <i>Basic Research in Cardiology</i> , 2018 , 113, 26 | 11.8 | 120 |
| 218 | Streptococcus pneumoniae translocates into the myocardium and forms unique microlesions that disrupt cardiac function. <i>PLoS Pathogens</i> , 2014 , 10, e1004383 | 7.6 | 119 |
| 217 | Matrix metalloproteinase-9 deletion attenuates myocardial fibrosis and diastolic dysfunction in ageing mice. <i>Cardiovascular Research</i> , 2012 , 96, 444-55 | 9.9 | 118 |
| 216 | 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 |
| 215 | Matrix Metalloproteinases in Myocardial Infarction and Heart Failure. <i>Progress in Molecular Biology and Translational Science</i> , 2017 , 147, 75-100 | 4 | 116 |
| 214 | Neutrophil roles in left ventricular remodeling following myocardial infarction. <i>Fibrogenesis and Tissue Repair</i> , 2013 , 6, 11 | | 113 |
| 213 | 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 |
| 212 | 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 |
| 211 | Deletion of thioredoxin-interacting protein in mice impairs mitochondrial function but protects the myocardium from ischemia-reperfusion injury. <i>Journal of Clinical Investigation</i> , 2012 , 122, 267-79 | 15.9 | 112 |
| 210 | The impact of aging on cardiac extracellular matrix. <i>GeroScience</i> , 2017 , 39, 7-18 | 8.9 | 109 |
| 209 | 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 |
| 208 | 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 |
| 207 | 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 |
| 206 | Extracellular matrix remodeling following myocardial injury. <i>Annals of Medicine</i> , 2003 , 35, 316-26 | 1.5 | 100 |

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|-----|--|------|----|
| 205 | 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 |
| 204 | Altered fibroblast function following myocardial infarction. <i>Journal of Molecular and Cellular Cardiology</i> , 2005 , 39, 699-707 | 5.8 | 99 |
| 203 | Cardiac Fibroblast Activation Post-Myocardial Infarction: Current Knowledge Gaps. <i>Trends in Pharmacological Sciences</i> , 2017 , 38, 448-458 | 13.2 | 94 |
| 202 | Extracellular matrix turnover and signaling during cardiac remodeling following MI: causes and consequences. <i>Journal of Molecular and Cellular Cardiology</i> , 2010 , 48, 558-63 | 5.8 | 83 |
| 201 | beta-blockade prevents sustained metalloproteinase activation and diastolic stiffening induced by angiotensin II combined with evolving cardiac dysfunction. <i>Circulation Research</i> , 2000 , 86, 807-15 | 15.7 | 82 |
| 200 | Proteomic analysis identifies in vivo candidate matrix metalloproteinase-9 substrates in the left ventricle post-myocardial infarction. <i>Proteomics</i> , 2010 , 10, 2214-23 | 4.8 | 81 |
| 199 | MMP induction and inhibition in myocardial infarction. <i>Heart Failure Reviews</i> , 2004 , 9, 7-19 | 5 | 79 |
| 198 | Extracellular matrix roles during cardiac repair. <i>Life Sciences</i> , 2010 , 87, 391-400 | 6.8 | 77 |
| 197 | Age-related cardiac muscle sarcopenia: Combining experimental and mathematical modeling to identify mechanisms. <i>Experimental Gerontology</i> , 2008 , 43, 296-306 | 4.5 | 77 |
| 196 | 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 |
| 195 | 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 |
| 194 | Long-lived ames dwarf mice are resistant to chemical stressors. <i>Journals of Gerontology - Series A Biological Sciences and Medical Sciences</i> , 2009 , 64, 819-27 | 6.4 | 70 |
| 193 | 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 |
| 192 | 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 |
| 191 | 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 |
| 190 | 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 |
| 189 | Understanding cardiac extracellular matrix remodeling to develop biomarkers of myocardial infarction outcomes. <i>Matrix Biology</i> , 2019 , 75-76, 43-57 | 11.4 | 64 |
| 188 | Myocardial matrix metalloproteinase-2: inside out and upside down. <i>Journal of Molecular and Cellular Cardiology</i> , 2014 , 77, 64-72 | 5.8 | 63 |

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| 187 | Texas 3-step decellularization protocol: looking at the cardiac extracellular matrix. <i>Journal of Proteomics</i> , 2013 , 86, 43-52 | 3.9 | 62 |
| 186 | Toll-like receptor (TLR) 2 and TLR4 differentially regulate doxorubicin induced cardiomyopathy in mice. <i>PLoS ONE</i> , 2012 , 7, e40763 | 3.7 | 62 |
| 185 | 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 |
| 184 | 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 |
| 183 | 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 |
| 182 | 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 |
| 181 | Assigning matrix metalloproteinase roles in ischaemic cardiac remodelling. <i>Nature Reviews Cardiology</i> , 2018 , 15, 471-479 | 14.8 | 58 |
| 180 | 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 |
| 179 | Extracellular matrix and fibroblast communication following myocardial infarction. <i>Journal of Cardiovascular Translational Research</i> , 2012 , 5, 848-57 | 3.3 | 55 |
| 178 | SPARC mediates early extracellular matrix remodeling following myocardial infarction. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2011 , 301, H497-505 | 5.2 | 54 |
| 177 | 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 |
| 176 | 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 |
| 175 | Multi-analyte profiling reveals matrix metalloproteinase-9 and monocyte chemoattractant protein-1 as plasma biomarkers of cardiac aging. <i>Circulation: Cardiovascular Genetics</i> , 2011 , 4, 455-62 | | 51 |
| 174 | Atherosclerosis exacerbates arrhythmia following myocardial infarction: Role of myocardial inflammation. <i>Heart Rhythm</i> , 2015 , 12, 169-78 | 6.7 | 50 |
| 173 | CC chemokine receptor 5 deletion impairs macrophage activation and induces adverse remodeling following myocardial infarction. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2011 , 300, H1418-26 | 5.2 | 49 |
| 172 | Harnessing the heart of big data. <i>Circulation Research</i> , 2015 , 116, 1115-9 | 15.7 | 47 |
| 171 | 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 |
| 170 | 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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| 169 | Effects of deletion of the matrix metalloproteinase 9 gene on development of murine thoracic aortic aneurysms. <i>Circulation</i> , 2005 , 112, 1242-8 | 16.7 | 47 |
| 168 | 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 |
| 167 | Statistical considerations in reporting cardiovascular research. <i>American Journal of Physiology - Heart and Circulatory Physiology</i> , 2018 , 315, H303-H313 | 5.2 | 45 |
| 166 | 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 |
| 165 | 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 |
| 164 | Caveolin-1 modulates TGF- β signaling in cardiac remodeling. <i>Matrix Biology</i> , 2011 , 30, 318-29 | 11.4 | 44 |
| 163 | In vivo matrix metalloproteinase-7 substrates identified in the left ventricle post-myocardial infarction using proteomics. <i>Journal of Proteome Research</i> , 2010 , 9, 2649-57 | 5.6 | 44 |
| 162 | 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 |
| 161 | Neutrophil proteome shifts over the myocardial infarction time continuum. <i>Basic Research in Cardiology</i> , 2019 , 114, 37 | 11.8 | 41 |
| 160 | Alterations in cultured myocardial fibroblast function following the development of left ventricular failure. <i>Journal of Molecular and Cellular Cardiology</i> , 2006 , 40, 474-83 | 5.8 | 41 |
| 159 | Antiarrhythmic effects of interleukin 1 inhibition after myocardial infarction. <i>Heart Rhythm</i> , 2017 , 14, 727-736 | 6.7 | 40 |
| 158 | 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 |
| 157 | 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 |
| 156 | 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, H284-97 | 5.2 | 39 |
| 155 | Heart failure with preserved ejection fraction: emerging drug strategies. <i>Journal of Cardiovascular Pharmacology</i> , 2013 , 62, 13-21 | 3.1 | 39 |
| 154 | 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 |
| 153 | Combining experimental and mathematical modeling to reveal mechanisms of macrophage-dependent left ventricular remodeling. <i>BMC Systems Biology</i> , 2011 , 5, 60 | 3.5 | 37 |
| 152 | Fibroblasts: The arbiters of extracellular matrix remodeling. <i>Matrix Biology</i> , 2020 , 91-92, 1-7 | 11.4 | 36 |

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| 151 | Walking the oxidative stress tightrope: a perspective from the naked mole-rat, the longest-living rodent. <i>Current Pharmaceutical Design</i> , 2011 , 17, 2290-307 | 3.3 | 36 |
| 150 | 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 |
| 149 | P. gingivalis 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 |
| 148 | Plasma Glycoproteomics Reveals Sepsis Outcomes Linked to Distinct Proteins in Common Pathways. <i>Critical Care Medicine</i> , 2015 , 43, 2049-2058 | 1.4 | 34 |
| 147 | Age and SPARC change the extracellular matrix composition of the left ventricle. <i>BioMed Research International</i> , 2014 , 2014, 810562 | 3 | 33 |
| 146 | Osteopontin is proteolytically processed by matrix metalloproteinase 9. <i>Canadian Journal of Physiology and Pharmacology</i> , 2015 , 93, 879-86 | 2.4 | 32 |
| 145 | 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 |
| 144 | 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 |
| 143 | A multidimensional proteomic approach to identify hypertrophy-associated proteins. <i>Proteomics</i> , 2006 , 6, 2225-35 | 4.8 | 32 |
| 142 | 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 |
| 141 | Matrix metalloproteinases: drug targets for myocardial infarction. <i>Current Drug Targets</i> , 2013 , 14, 276-86 | | 31 |
| 140 | The circular relationship between matrix metalloproteinase-9 and inflammation following myocardial infarction. <i>IUBMB Life</i> , 2015 , 67, 611-8 | 4.7 | 30 |
| 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 | 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 |
| 137 | The left ventricle proteome differentiates middle-aged and old left ventricles in mice. <i>Journal of Proteome Research</i> , 2008 , 7, 756-65 | 5.6 | 29 |
| 136 | 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 |
| 135 | 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 |
| 134 | Effect of a cleavage-resistant collagen mutation on left ventricular remodeling. <i>Circulation Research</i> , 2003 , 93, 238-45 | 15.7 | 28 |

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|-----|---|------|----|
| 133 | 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 |
| 132 | Proteomic analysis reveals late exercise effects on cardiac remodeling following myocardial infarction. <i>Journal of Proteomics</i> , 2010 , 73, 2041-9 | 3.9 | 27 |
| 131 | Applications of miRNA technology for atherosclerosis. <i>Current Atherosclerosis Reports</i> , 2014 , 16, 386 | 6 | 26 |
| 130 | Mechanisms to inhibit matrix metalloproteinase activity: where are we in the development of clinically relevant inhibitors?. <i>Recent Patents on Anti-Cancer Drug Discovery</i> , 2007 , 2, 135-42 | 2.6 | 26 |
| 129 | Matrix Metalloproteinases: Drug Targets for Myocardial Infarction. <i>Current Drug Targets</i> , 2013 , 14, 276-286 | | 26 |
| 128 | Adapting extracellular matrix proteomics for clinical studies on cardiac remodeling post-myocardial infarction. <i>Clinical Proteomics</i> , 2016 , 13, 19 | 5 | 25 |
| 127 | Effects of surface-modified scaffolds on the growth and differentiation of mouse adipose-derived stromal cells. <i>Journal of Tissue Engineering and Regenerative Medicine</i> , 2007 , 1, 211-7 | 4.4 | 25 |
| 126 | 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 |
| 125 | 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 |
| 124 | Cardiac extracellular proteome profiling and membrane topology analysis using glycoproteomics. <i>Proteomics - Clinical Applications</i> , 2014 , 8, 595-602 | 3.1 | 23 |
| 123 | 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 |
| 122 | Matrix metalloproteinase (MMP)-7 activates MMP-8 but not MMP-13. <i>Medicinal Chemistry</i> , 2006 , 2, 523-6 | 1.8 | 23 |
| 121 | Interleukin 6 induction in the canine myocardium after cardiopulmonary bypass. <i>Journal of Thoracic and Cardiovascular Surgery</i> , 2000 , 120, 256-63 | 1.5 | 23 |
| 120 | Cardiac fibroblast activation during myocardial infarction wound healing: Fibroblast polarization after MI. <i>Matrix Biology</i> , 2020 , 91-92, 109-116 | 11.4 | 23 |
| 119 | Effects of early and late chronic pressure overload on extracellular matrix remodeling. <i>Hypertension Research</i> , 2008 , 31, 1225-31 | 4.7 | 22 |
| 118 | Proteomic analysis of the cardiac extracellular matrix: clinical research applications. <i>Expert Review of Proteomics</i> , 2018 , 15, 105-112 | 4.2 | 21 |
| 117 | ACE inhibitors to block MMP-9 activity: new functions for old inhibitors. <i>Journal of Molecular and Cellular Cardiology</i> , 2007 , 43, 664-6 | 5.8 | 21 |
| 116 | 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 |

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| 115 | Intercellular adhesion molecule-1 regulation in the canine lung after cardiopulmonary bypass. <i>Journal of Thoracic and Cardiovascular Surgery</i> , 1998 , 115, 689-98; discussion 698-9 | 1.5 | 20 |
| 114 | Using proteomics to uncover extracellular matrix interactions during cardiac remodeling. <i>Proteomics - Clinical Applications</i> , 2013 , 7, 516-27 | 3.1 | 18 |
| 113 | Bayesian parameter estimation for nonlinear modelling of biological pathways. <i>BMC Systems Biology</i> , 2011 , 5 Suppl 3, S9 | 3.5 | 18 |
| 112 | Alterations of pulse pressure stimulate arterial wall matrix remodeling. <i>Journal of Biomechanical Engineering</i> , 2009 , 131, 101011 | 2.1 | 18 |
| 111 | Chronic and intermittent hypoxia differentially regulate left ventricular inflammatory and extracellular matrix responses. <i>Hypertension Research</i> , 2012 , 35, 811-8 | 4.7 | 18 |
| 110 | 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 |
| 109 | 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 |
| 108 | Negative elongation factor controls energy homeostasis in cardiomyocytes. <i>Cell Reports</i> , 2014 , 7, 79-85 | 10.6 | 17 |
| 107 | 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 |
| 106 | Inhibiting metalloproteases with PD 166793 in heart failure: impact on cardiac remodeling and beyond. <i>Cardiovascular Drug Reviews</i> , 2008 , 26, 24-37 | | 16 |
| 105 | Stability analysis of genetic regulatory network with additive noises. <i>BMC Genomics</i> , 2008 , 9 Suppl 1, S21 | 4.5 | 16 |
| 104 | Plasma monitoring of the myocardial specific tissue inhibitor of metalloproteinase-4 after alcohol septal ablation in hypertrophic obstructive cardiomyopathy. <i>Journal of Cardiac Failure</i> , 2005 , 11, 124-30 | 3.3 | 16 |
| 103 | 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 |
| 102 | Neutrophil signaling during myocardial infarction wound repair. <i>Cellular Signalling</i> , 2021 , 77, 109816 | 4.9 | 15 |
| 101 | Alterations in Pulse Pressure Affect Artery Function. <i>Cellular and Molecular Bioengineering</i> , 2012 , 5, 474-487 | 3.9 | 13 |
| 100 | Titin phosphorylation: myocardial passive stiffness regulated by the intracellular giant. <i>Circulation Research</i> , 2009 , 105, 611-3 | 15.7 | 13 |
| 99 | Using extracellular matrix proteomics to understand left ventricular remodeling. <i>Circulation: Cardiovascular Genetics</i> , 2012 , 5, o1-7 | | 13 |
| 98 | Tissue Inhibitor of Metalloproteinase-1: Actions beyond Matrix Metalloproteinase Inhibition. <i>Cardiology</i> , 2015 , 132, 147-50 | 1.6 | 12 |

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|----|---|------|----|
| 97 | 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 |
| 96 | 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 |
| 95 | A conceptual cellular interaction model of left ventricular remodeling post-MI: dynamic network with exit-entry competition strategy. <i>BMC Systems Biology</i> , 2010 , 4 Suppl 1, S5 | 3.5 | 12 |
| 94 | 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 |
| 93 | Mathematical modeling of left ventricular dimensional changes in mice during aging. <i>BMC Systems Biology</i> , 2012 , 6 Suppl 3, S10 | 3.5 | 11 |
| 92 | Novel strategies to delineate matrix metalloproteinase (MMP)-substrate relationships and identify targets to block MMP activity. <i>Mini-Reviews in Medicinal Chemistry</i> , 2006 , 6, 1243-8 | 3.2 | 11 |
| 91 | 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 |
| 90 | 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 |
| 89 | 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 |
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