List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Large animal model of vein grafts intimal hyperplasia: A systematic review. Perfusion (United Kingdom), 2023, 38, 894-930.	1.0	5
2	Nrf2-Keap-1 imbalance under acute shear stress induces inflammatory response in venous endothelial cells. Perfusion (United Kingdom), 2022, 37, 582-589.	1.0	7
3	Monitoring Cellular and in Plaques and. Methods in Molecular Biology, 2022, 2419, 507-519.	0.9	0
4	Investigation of Atherosclerotic Plaque Vulnerability. Methods in Molecular Biology, 2022, 2419, 521-535.	0.9	1
5	Monitoring Cellular Proliferation, Migration, and Apoptosis Associated with Atherosclerosis Plaques In Vitro. Methods in Molecular Biology, 2022, 2419, 133-167.	0.9	3
6	Use of Mouse Carotid Model of Intimal to Probe Vascular Smooth Muscle Remodeling and Function in. Methods in Molecular Biology, 2022, 2419, 537-560.	0.9	1
7	Next-Generation and Single-Cell Sequencing Approaches to Study Atherosclerosis and Vascular Inflammation Pathophysiology: A Systematic Review. Frontiers in Cardiovascular Medicine, 2022, 9, 849675.	2.4	5
8	Therapeutic potential of inhibiting mitochondrial fission to reduce abdominal aortic aneurysms. Cardiovascular Research, 2021, 117, 658-660.	3.8	0
9	Aneurysm severity is suppressed by deletion of CCN4. Journal of Cell Communication and Signaling, 2021, 15, 421-432.	3.4	5
10	Effective decellularisation of human saphenous veins for biocompatible arterial tissue engineering applications: Bench optimisation and feasibility in vivo testing. Journal of Tissue Engineering, 2021, 12, 204173142098752.	5.5	5
11	Disparate effects of MMP and TIMP modulation on coronary atherosclerosis and associated myocardial fibrosis. Scientific Reports, 2021, 11, 23081.	3.3	22
12	NF-κB inhibition prevents acute shear stress-induced inflammation in the saphenous vein graft endothelium. Scientific Reports, 2020, 10, 15133.	3.3	24
13	A Protocol for a Novel Human Ex Vivo Model of Aneurysm. STAR Protocols, 2020, 1, 100108.	1.2	1
14	Saphenous vein graft disease, pathophysiology, prevention, and treatment. A review of the literature. Journal of Cardiac Surgery, 2020, 35, 1314-1321.	0.7	22
15	Galectin-3 Identifies a Subset of Macrophages With a Potential Beneficial Role in Atherosclerosis. Arteriosclerosis, Thrombosis, and Vascular Biology, 2020, 40, 1491-1509.	2.4	49
16	Nonautologous Grafts in Coronary Artery Bypass Surgery: A Systematic Review. Annals of Thoracic Surgery, 2020, 112, 2094-2103.	1.3	2
17	Aging differentially modulates the Wnt proâ€survival signalling pathways in vascular smooth muscle cells. Aging Cell, 2019, 18, e12844.	6.7	23
18	The Human-Specific and Smooth Muscle Cell-Enriched LncRNA SMILR Promotes Proliferation by Regulating Mitotic CENPF mRNA and Drives Cell-Cycle Progression Which Can Be Targeted to Limit Vascular Remodeling. Circulation Research, 2019, 125, 535-551.	4.5	100

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19	Adipose tissue–derived WNT5A regulates vascular redox signaling in obesity via USP17/RAC1-mediated activation of NADPH oxidases. Science Translational Medicine, 2019, 11, .	12.4	54
20	BS26â€Ceneration of a tissue engineered conduit from human saphenous vein and porcine blood outgrowth endothelial cells. , 2019, , .		1
21	P5000Wnt/b-catenin signalling drives angiotensin II induced cardiac fibrosis via WISP-1. European Heart Journal, 2019, 40, .	2.2	1
22	The cardiac proteome in patients with congenital ventricular septal defect: A comparative study between right atria and right ventricles. Journal of Proteomics, 2019, 191, 107-113.	2.4	7
23	Lung Function, Inflammation, and Endothelinâ€1 in Congenital Heart Disease–Associated Pulmonary Arterial Hypertension. Journal of the American Heart Association, 2018, 7, .	3.7	17
24	Changes in contractile protein expression are linked to ventricular stiffness in infants with pulmonary hypertension or right ventricular hypertrophy due to congenital heart disease. Open Heart, 2018, 5, e000716.	2.3	15
25	Role of smooth muscle cells in coronary artery bypass grafting failure. Cardiovascular Research, 2018, 114, 601-610.	3.8	63
26	Carotid artery ligation induced intimal thickening and proliferation is unaffected by ageing. Journal of Cell Communication and Signaling, 2018, 12, 529-537.	3.4	8
27	O3â€∱MMP12 INHIBITION PROTECTS AGAINST ABDOMINAL AORTIC ANEURYSM PROGRESSION. Cardiovascular Research, 2018, 114, S1-S1.	3.8	0
28	Phosphorylation of PRH/HHEX by Protein Kinase CK2 Regulates Cell Proliferation and Cell Migration in Diverse Cell Types. , 2018, , .		0
29	P22â€∱PRO- AND ANTI-INFLAMMATORY MACROPHAGES DISPLAY DIVERGENT POLARISATION TOWARDS VASCULAR SMOOTH MUSCLE-LIKE AND ENDOTHELIAL-LIKE PHENOTYPES. Cardiovascular Research, 2018, 114, S7-S8.	3.8	0
30	P2 VALIDATION OF A NOVEL HUMAN EX-VIVO MODEL OF ANEURYSM TO SUPPLANT MOUSE MODELS. Cardiovascular Research, 2018, 114, S2-S2.	3.8	0
31	P3 MODULATION OF THE ACTIN CYTOSKELETON IN MACROPHAGE PHENOTYPES DIFFERENTIALLY AFFECTS THEIR BEHAVIOUR. Cardiovascular Research, 2018, 114, S2-S2.	3.8	0
32	28Development and characterisation of a human ex-vivo model of aneurysm. Cardiovascular Research, 2018, 114, S6-S7.	3.8	0
33	Targeting Wnt/β-Catenin Activated Cells with Dominant-Negative N-cadherin to Reduce Neointima Formation. Molecular Therapy - Methods and Clinical Development, 2017, 5, 191-199.	4.1	13
34	Contribution of the classical NF- <kappa>B pathway to venous endothelial inflammation following acute increases in shear stress: Implications for vein graft failure. Atherosclerosis, 2017, 263, e135.</kappa>	0.8	0
35	Activation and inflammation of the venous endothelium in vein graft disease. Atherosclerosis, 2017, 265, 266-274.	0.8	53
36	Inhibition of smooth muscle cell proliferation and intimal thickening with small peptide mimetics of soluble N-cadherin. Atherosclerosis, 2017, 263, e64-e65.	0.8	0

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37	Protein kinase CK2 inhibition suppresses neointima formation via a proline-rich homeodomain-dependent mechanism. Vascular Pharmacology, 2017, 99, 34-44.	2.1	10
38	Aneurysm Severity is Increased by Combined Mmp-7 Deletion and N-cadherin Mimetic (EC4-Fc) Over-Expression. Scientific Reports, 2017, 7, 17342.	3.3	13
39	MicroRNA-181b Controls Atherosclerosis and Aneurysms Through Regulation of TIMP-3 and Elastin. Circulation Research, 2017, 120, 49-65.	4.5	125
40	Evidence for the Involvement of Matrix-Degrading Metalloproteinases (MMPs) in Atherosclerosis. Progress in Molecular Biology and Translational Science, 2017, 147, 197-237.	1.7	44
41	Hypoxic Pulmonary Vasoconstriction in Humans: Tale or Myth. Open Cardiovascular Medicine Journal, 2017, 11, 1-13.	0.3	19
42	Suppression of neointima formation by targeting $\hat{l}^2$ -catenin/TCF pathway. Bioscience Reports, 2016, 36, .	2.4	9
43	Wnt2 and WISP-1/CCN4 Induce Intimal Thickening via Promotion of Smooth Muscle Cell Migration. Arteriosclerosis, Thrombosis, and Vascular Biology, 2016, 36, 1417-1424.	2.4	47
44	Differential effects of tissue inhibitor of metalloproteinase (TIMP)-1 and TIMP-2 on atherosclerosis and monocyte/macrophage invasion. Cardiovascular Research, 2016, 109, 318-330.	3.8	44
45	Response to Weintraub and Garratt. Circulation, 2016, 133, 1826-1826.	1.6	0
46	Should Chronic Total Occlusion Be Treated With Coronary Artery Bypass Grafting?. Circulation, 2016, 133, 1807-1816.	1.6	14
47	Soluble N-cadherin: A novel inhibitor of VSMC proliferation and intimal thickening. Vascular Pharmacology, 2016, 78, 53-62.	2.1	8
48	Cellular and molecular basis of RV hypertrophy in congenital heart disease. Heart, 2016, 102, 12-17.	2.9	33
49	Abstract 17136: MicroRNA-181b Inhibition Stabilises Abdominal Aortic Aneurysms by Promoting Collagen Accumulation and Elastin Deposition. Circulation, 2015, 132, .	1.6	0
50	EC4, a truncation of soluble N-cadherin, reduces vascular smooth muscle cell apoptosis and markers of atherosclerotic plaque instability. Molecular Therapy - Methods and Clinical Development, 2014, 1, 14004.	4.1	5
51	Wnt5a-Induced Wnt1-Inducible Secreted Protein-1 Suppresses Vascular Smooth Muscle Cell Apoptosis Induced by Oxidative Stress. Arteriosclerosis, Thrombosis, and Vascular Biology, 2014, 34, 2449-2456.	2.4	36
52	miRNA-21 is dysregulated in response to vein grafting in multiple models and genetic ablation in mice attenuates neointima formation. European Heart Journal, 2013, 34, 1636-1643.	2.2	61
53	Contractile, but not endothelial, dysfunction in early inflammatory arthritis: a possible role for matrix metalloproteinaseâ€9. British Journal of Pharmacology, 2012, 167, 505-514. 	5.4	19
54	Wnt signalling in smooth muscle cells and its role in cardiovascular disorders. Cardiovascular Research, 2012, 95, 233-240.	3.8	113

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55	Vein graft failure: current clinical practice and potential for gene therapeutics. Gene Therapy, 2012, 19, 630-636.	4.5	45
56	Metalloproteinases in atherosclerotic plaques — A matter of life or death. Vascular Pharmacology, 2012, 56, 336.	2.1	0
57	Extracellular Matrix and Smooth Muscle Cells. , 2012, , 435-460.		2
58	YIA 4 Wnt5a signalling promotes VSMC survival via WISP-1: consequences for VSMC viability in atherosclerotic plaques. Heart, 2011, 97, e7-e7.	2.9	4
59	Regulation of VSMC behavior by the cadherin-catenin complex. Frontiers in Bioscience - Landmark, 2011, 16, 644.	3.0	29
60	The Wnt pathways in vascular disease. Current Opinion in Lipidology, 2011, 22, 350-357.	2.7	70
61	Prevention of post-cardiopulmonary bypass acute kidney injury by endothelin A receptor blockade*. Critical Care Medicine, 2011, 39, 793-802.	0.9	65
62	A Selective Matrix Metalloproteinase-12 Inhibitor Retards Atherosclerotic Plaque Development in Apolipoprotein E–Knockout Mice. Arteriosclerosis, Thrombosis, and Vascular Biology, 2011, 31, 528-535.	2.4	144
63	Wnt4/β-Catenin Signaling Induces VSMC Proliferation and Is Associated With Intimal Thickening. Circulation Research, 2011, 108, 427-436.	4.5	140
64	Matrix Metalloproteinase (MMP)-3 Activates MMP-9 Mediated Vascular Smooth Muscle Cell Migration and Neointima Formation in Mice. Arteriosclerosis, Thrombosis, and Vascular Biology, 2011, 31, e35-44.	2.4	122
65	Sustained Reduction of Vein Graft Neointima Formation by Ex Vivo TIMP-3 Gene Therapy. Circulation, 2011, 124, S135-42.	1.6	65
66	Tissue-engineered vascular graft remodeling in a growing lamb model: expression of matrix metalloproteinases. European Journal of Cardio-thoracic Surgery, 2011, 41, 167-72.	1.4	33
67	MMP-7 mediates cleavage of N-cadherin and promotes smooth muscle cell apoptosis. Cardiovascular Research, 2010, 87, 137-146.	3.8	90
68	Dysregulation of cadherins in the intercalated disc of the spontaneously hypertensive stroke-prone rat. Journal of Molecular and Cellular Cardiology, 2010, 48, 1121-1128.	1.9	4
69	Increased expression of Wnt5A in unstable atherosclerotic plaques is associated with increased MMP expression and may contribute to instability. Atherosclerosis, 2010, 213, e12.	0.8	6
70	In Situ Zymography. Methods in Molecular Biology, 2010, 622, 271-277.	0.9	21
71	MMP-9 and -12 cause N-cadherin shedding and thereby β-catenin signalling and vascular smooth muscle cell proliferation. Cardiovascular Research, 2009, 81, 178-186.	3.8	124
72	Soluble N-Cadherin Overexpression Reduces Features of Atherosclerotic Plaque Instability. Arteriosclerosis, Thrombosis, and Vascular Biology, 2009, 29, 195-201.	2.4	46

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73	Effect of broad-spectrum matrix metalloproteinase inhibition on atherosclerotic plaque stability. Cardiovascular Research, 2006, 71, 586-595.	3.8	70
74	Suppression of Atherosclerotic Plaque Progression and Instability by Tissue Inhibitor of Metalloproteinase-2. Circulation, 2006, 113, 2435-2444.	1.6	142
75	Regulation of Smooth Muscle Cell Proliferation by β-Catenin/T-Cell Factor Signaling Involves Modulation of Cyclin D1 and p21 Expression. Circulation Research, 2006, 99, 1329-1337.	4.5	125
76	Plaque Rupture After Short Periods of Fat Feeding in the Apolipoprotein E–Knockout Mouse. Circulation, 2005, 111, 1422-1430.	1.6	235
77	Divergent effects of matrix metalloproteinases 3, 7, 9, and 12 on atherosclerotic plaque stability in mouse brachiocephalic arteries. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 15575-15580.	7.1	308
78	Transforming Growth Factor-β Is Activated by Plasmin and Inhibits Smooth Muscle Cell Death in Human Saphenous Vein. Journal of Vascular Research, 2005, 42, 247-254.	1.4	25
79	N-Cadherin–Dependent Cell–Cell Contacts Promote Human Saphenous Vein Smooth Muscle Cell Survival. Arteriosclerosis, Thrombosis, and Vascular Biology, 2005, 25, 982-988.	2.4	64
80	R-Cadherin:β-Catenin Complex and Its Association With Vascular Smooth Muscle Cell Proliferation. Arteriosclerosis, Thrombosis, and Vascular Biology, 2004, 24, 1204-1210.	2.4	77
81	Dismantling of Cadherin-Mediated Cell-Cell Contacts Modulates Smooth Muscle Cell Proliferation. Circulation Research, 2003, 92, 1314-1321.	4.5	129
82	Relationship between type IV collagen degradation, metalloproteinase activity and smooth muscle cell migration and proliferation in cultured human saphenous vein. Cardiovascular Research, 2003, 58, 679-688.	3.8	52
83	Gene Transfer to the Vasculature. Molecular Biotechnology, 2002, 22, 153-163.	2.4	5
84	Wild-type p53 gene transfer inhibits neointima formation in human saphenous vein by modulation of smooth muscle cell migration and induction of apoptosis. Gene Therapy, 2001, 8, 668-676.	4.5	80
85	Plasmin-Mediated Fibroblast Growth Factor-2 Mobilisation Supports Smooth Muscle Cell Proliferation in Human Saphenous Vein. Journal of Vascular Research, 2001, 38, 492-501.	1.4	34
86	Injury Induces Dedifferentiation of Smooth Muscle Cells and Increased Matrix-Degrading Metalloproteinase Activity in Human Saphenous Vein. Arteriosclerosis, Thrombosis, and Vascular Biology, 2001, 21, 1146-1151.	2.4	90
87	Inhibition of Late Vein Graft Neointima Formation in Human and Porcine Models by Adenovirus-Mediated Overexpression of Tissue Inhibitor of Metalloproteinase-3. Circulation, 2000, 101, 296-304.	1.6	203
88	The association of platelet-derived growth factor receptor expression, plaque morphology and histological features with symptoms in carotid atherosclerosis. Vascular, 2000, 8, 121-129.	0.5	11
89	Gene transfer of tissue inhibitor of metalloproteinase-2 inhibits metalloproteinase activity and neointima formation in human saphenous veins. Gene Therapy, 1998, 5, 1552-1560.	4.5	144
90	External stenting reduces long-term medial and neointimal thickening and platelet derived growth factor expression in a pig model of arteriovenous bypass grafting. Nature Medicine, 1998, 4, 235-239.	30.7	145

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91	Adenovirus-Mediated Gene Transfer of the Human TIMP-1 Gene Inhibits Smooth Muscle Cell Migration and Neointimal Formation in Human Saphenous Vein. Human Gene Therapy, 1998, 9, 867-877.	2.7	201
92	Activation of Matrix-Degrading Metalloproteinases by Mast Cell Proteases in Atherosclerotic Plaques. Arteriosclerosis, Thrombosis, and Vascular Biology, 1998, 18, 1707-1715.	2.4	234
93	Tissue inhibitors of metalloproteinases and metalloproteinases in atherosclerosis. Current Opinion in Lipidology, 1998, 9, 413-423.	2.7	70
94	Divergent effects of tissue inhibitor of metalloproteinase-1, -2, or -3 overexpression on rat vascular smooth muscle cell invasion, proliferation, and death in vitro. TIMP-3 promotes apoptosis Journal of Clinical Investigation, 1998, 101, 1478-1487.	8.2	416
95	Surgical preparative injury and neointima formation increase MMP-9 expression and MMP-2 activation in human saphenous vein. Cardiovascular Research, 1997, 33, 447-459.	3.8	116
96	Short-term Exposure to Thapsigargin Inhibits Neointima Formation in Human Saphenous Vein. Arteriosclerosis, Thrombosis, and Vascular Biology, 1997, 17, 2500-2506.	2.4	31
97	An essential role for platelet-derived growth factor in neointima formation in human saphenous vein in vitro. Atherosclerosis, 1996, 120, 227-240.	0.8	57
98	Neointimal fibrosis in vascular pathologies: role of growth factors and metalloproteinases in vascular smooth muscle proliferation. Experimental Nephrology, 1995, 3, 108-13.	0.4	8