Howard A Rockman

List of Publications by Year
in descending order

108 papers	10,507 citations	31976 53 h-index	32842 100 g-index
110 all docs	110 docs citations	110 times ranked	9520 citing authors

#	Article	IF	Citations
1	Proximity labeling for investigating protein-protein interactions. Methods in Cell Biology, 2022, , 237-266.	1.1	7
2	Late onset cardiovascular dysfunction in adult mice resulting from galactic cosmic ray exposure. IScience, 2022, 25, 104086.	4.1	9
3	G protein-coupled receptor signaling: transducers and effectors. American Journal of Physiology - Cell Physiology, 2022, 323, C731-C748.	4.6	22
4	Mapping Angiotensin II Type 1 Receptor-Biased Signaling Using Proximity Labeling and Proteomics Identifies Diverse Actions of Biased Agonists. Journal of Proteome Research, 2021, 20, 3256-3267.	3.7	11
5	<i>Î²</i>-Arrestinâ€“Biased Allosteric Modulator Potentiates Carvedilol-Stimulated <i>Î²</i> Adrenergic Receptor Cardioprotection. Molecular Pharmacology, 2021, 100, 568-579.	2.3	24
6	Synthetic nanobodies as angiotensin receptor blockers. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 20284-20291.	7.1	35
7	The Î²-arrestin-biased Î²-adrenergic receptor blocker carvedilol enhances skeletal muscle contractility. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 12435-12443.	7.1	19
8	Î²-Arrestinâ€“Biased Angiotensin II Receptor Agonists for COVID-19. Circulation, 2020, 142, 318-320.	1.6	19
9	A murine model of increased coronary sinus pressure induces myocardial edema with cardiac lymphatic dilation and fibrosis. American Journal of Physiology - Heart and Circulatory Physiology, 2020, 318, H895-H907.	3.2	11
10	The deubiquitinase ubiquitinâ€“specific protease 20 is a positive modulator of myocardial Î²1-adrenergic receptor expression and signaling. Journal of Biological Chemistry, 2019, 294, 2500-2518.	3.4	17
11	Mechanoactivation of the angiotensin II type 1 receptor induces Î²â€arrestinâ€biased signaling through GÎ±_i coupling. Journal of Cellular Biochemistry, 2018, 119, 3586-3597.	2.6	39
12	Î²-arrestin 1 regulates Î²2-adrenergic receptor-mediated skeletal muscle hypertrophy and contractility. Skeletal Muscle, 2018, 8, 39.	4.2	37
13	Manifold roles of Î²-arrestins in GPCR signaling elucidated with siRNA and CRISPR/Cas9. Science Signaling, 2018, 11, .	3.6	169
14	G-Proteinâ€“Coupled Receptors in Heart Disease. Circulation Research, 2018, 123, 716-735.	4.5	184
15	Biased G Proteinâ€“Coupled Receptor Signaling. Circulation, 2018, 137, 2315-2317.	1.6	69
16	The two-pore domain potassium channel TREK-1 mediates cardiac fibrosis and diastolic dysfunction. Journal of Clinical Investigation, 2018, 128, 4843-4855.	8.2	62
17	GÎ±i is required for carvedilol-induced Î²1 adrenergic receptor Î²-arrestin biased signaling. Nature Communications, 2017, 8, 1706.	12.8	83
18	Mdm2 regulates cardiac contractility by inhibiting GRK2-mediated desensitization of Î²-adrenergic receptor signaling. JCI Insight, 2017, 2, .	5.0	17

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19	G Proteinâ€“coupled Receptor Biased Agonism. Journal of Cardiovascular Pharmacology, 2016, 67, 193-202.	1.9	41
20	Phosphorylation of Src by phosphoinositide 3-kinase regulates beta-adrenergic receptor-mediated EGFR transactivation. Cellular Signalling, 2016, 28, 1580-1592.	3.6	21
21	Î²-Arrestin mediates the Frankâ€“Starling mechanism of cardiac contractility. Proceedings of the National Academy of Sciences of the United States of America, 2016, 113, 14426-14431.	7.1	46
22	The role of Î²-arrestin2-dependent signaling in thoracic aortic aneurysm formation in a murine model of Marfan syndrome. American Journal of Physiology - Heart and Circulatory Physiology, 2015, 309, H1516-H1527.	3.2	17
23	Circulating Exosomes Induced by Cardiac Pressure Overload Contain Functional Angiotensin II Type 1 Receptors. Circulation, 2015, 131, 2120-2130.	1.6	177
24	Lethal Cardiomyopathy in Mice Lacking Transferrin Receptor in the Heart. Cell Reports, 2015, 13, 533-545.	6.4	213
25	Abstract 274: Carvedilol Stimulated GÎ±i-Î²-Arrestin Biased Î²1 Adrenergic Receptor Signaling. Circulation Research, 2015, 117, .	4.5	0
26	Galactokinase Is a Novel Modifier of Calcineurin-Induced Cardiomyopathy in <i>Drosophila</i>. Genetics, 2014, 198, 591-603.	2.9	10
27	Allosteric Modulation of Î²-Arrestin-biased Angiotensin II Type 1 Receptor Signaling by Membrane Stretch. Journal of Biological Chemistry, 2014, 289, 28271-28283.	3.4	55
28	Î²-Arrestin1â€“Biased Î² ₁ -Adrenergic Receptor Signaling Regulates MicroRNA Processing. Circulation Research, 2014, 114, 833-844.	4.5	60
29	Modulating G Protein-Coupled Receptors to Effect Reverse Cardiac Remodeling. , 2013, , 159-177.		0
30	Animal Models of Heart Failure. Circulation Research, 2012, 111, 131-150.	4.5	378
31	Deletion of Siah-interacting protein gene in Drosophila causes cardiomyopathy. Molecular Genetics and Genomics, 2012, 287, 351-360.	2.1	3
32	Î²-Arrestin-biased AT1R stimulation promotes cell survival during acute cardiac injury. American Journal of Physiology - Heart and Circulatory Physiology, 2012, 303, H1001-H1010.	3.2	105
33	Heart fails without pump partner. Nature, 2011, 477, 546-547.	27.8	4
34	Î²-Arrestin: A signaling molecule and potential therapeutic target for heart failure. Journal of Molecular and Cellular Cardiology, 2011, 51, 534-541.	1.9	79
35	Introduction to the Series on Novel Aspects of Cardiovascular G-Protein-Coupled Receptor Signaling. Circulation Research, 2011, 109, 202-204.	4.5	4
36	Drosophila, Genetic Screens, and Cardiac Function. Circulation Research, 2011, 109, 794-806.	4.5	51

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37	Î²-Arrestin mediates oxytocin receptor signaling, which regulates uterine contractility and cellular migration. American Journal of Physiology - Endocrinology and Metabolism, 2011, 300, E468-E477.	3.5	37
38	Cardiomyopathy Is Associated with Ribosomal Protein Gene Haplo-Insufficiency in<i>Drosophila melanogaster</i>. Genetics, 2011, 189, 861-870.	2.9	23
39	Increased intracellular creatine content is not harmful to the mouse heart. FASEB Journal, 2011, 25, 1033.10.	0.5	0
40	Gene Deletion Screen for Cardiomyopathy in Adult Drosophila Identifies a New Notch Ligand. Circulation Research, 2010, 106, 1233-1243.	4.5	43
41	Functional Selectivity in Adrenergic and Angiotensin Signaling Systems. Molecular Pharmacology, 2010, 78, 983-992.	2.3	71
42	Î²-Arrestinâ€"dependent activation of Ca2+/calmodulin kinase II after Î²1â€"adrenergic receptor stimulation. Journal of Cell Biology, 2010, 189, 573-587.	5.2	142
43	Î²-Arrestinâ€"Biased Agonism of the Angiotensin Receptor Induced by Mechanical Stress. Science Signaling, 2010, 3, ra46.	3.6	163
44	AT1 A Râ€•Î²â€•arrestin signaling confers PPARÎ³ agonistâ€•mediated myocyte contractility. FASEB Journal, 2010, 24, 586.3.	0.5	0
45	Î²-Arrestin Mediates Î²1-Adrenergic Receptor-Epidermal Growth Factor Receptor Interaction and Downstream Signaling. Journal of Biological Chemistry, 2009, 284, 20375-20386.	3.4	92
46	TRPC1 Channels Are Critical for Hypertrophic Signaling in the Heart. Circulation Research, 2009, 105, 1023-1030.	4.5	202
47	Î²₁-Adrenergic receptors stimulate cardiac contractility and CaMKII activation in vivo and enhance cardiac dysfunction following myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2009, 297, H1377-H1386.	3.2	85
48	Decreased beta-adrenergic responsiveness following hypertrophy occurs only in cardiomyocytes that also re-express beta-myosin heavy chain. European Journal of Heart Failure, 2009, 11, 648-652.	7.1	10
49	An N-ethyl-N-nitrosourea mutagenesis recessive screen identifies two candidate regions for murine cardiomyopathy that map to chromosomes 1 and 15. Mammalian Genome, 2009, 20, 296-304.	2.2	6
50	Physiologic and cardiac roles of Î²-arrestins. Journal of Molecular and Cellular Cardiology, 2009, 46, 300-308.	1.9	50
51	Increased myocardial contractility and enhanced exercise function in transgenic mice overexpressing either adenylyl cyclase 5 or 8. Basic Research in Cardiology, 2008, 103, 22-30.	5.9	26
52	Î²-Blockers alprenolol and carvedilol stimulate Î²-arrestin-mediated EGFR transactivation. Proceedings of the National Academy of Sciences of the United States of America, 2008, 105, 14555-14560.	7.1	241
53	Beta-Arrestin-Mediated Signaling in the Heart. Circulation Journal, 2008, 72, 1725-1729.	1.6	46
54	Receptor Signaling Pathways in Heart Failure: Transgenic Mouse Models. , 2008, , 89-111.		0

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55	[beta]1 adrenergic receptor ([beta]1AR)â€epidermal growth factor receptor (EGFR) interaction regulates ERK cellular activity. FASEB Journal, 2008, 22, 723.1.	0.5	0
56	Left Ventricular Functional Assessment in Mice: Feasibility of High Spatial and Temporal Resolution ECG-gated Blood Pool SPECT. Radiology, 2007, 245, 440-448.	7.3	23
57	Reduced life span with heart and muscle dysfunction in Drosophila sarcoglycan mutants. Human Molecular Genetics, 2007, 16, 2933-2943.	2.9	61
58	Reversal of cardiac remodeling by modulation of adrenergic receptors: a new frontier in heart failure. Current Opinion in Cardiology, 2007, 22, 443-449.	1.8	35
59	Cardiac GPCRs: GPCR signaling in healthy and failing hearts. Biochimica Et Biophysica Acta - Biomembranes, 2007, 1768, 1006-1018.	2.6	165
60	Regulation of Î²-Adrenergic Receptor Signaling by S-Nitrosylation of G-Protein-Coupled Receptor Kinase 2. Cell, 2007, 129, 511-522.	28.9	274
61	Î²-Arrestinâ€"mediated Î²1-adrenergic receptor transactivation of the EGFR confers cardioprotection. Journal of Clinical Investigation, 2007, 117, 2445-2458.	8.2	405
62	Role of Î²-adrenergic receptor signaling and desensitization in heart failure: new concepts and prospects for treatment. Expert Review of Cardiovascular Therapy, 2006, 4, 417-432.	1.5	70
63	JNK1 is required to preserve cardiac function in the early response to pressure overload. Biochemical and Biophysical Research Communications, 2006, 343, 1060-1066.	2.1	60
64	Competitive displacement of phosphoinositide 3-kinase from Î²-adrenergic receptor kinase-1 improves postinfarction adverse myocardial remodeling. American Journal of Physiology - Heart and Circulatory Physiology, 2006, 291, H1754-H1760.	3.2	29
65	Lessons From Overexpressed Mouse Models. , 2006, , 293-320.		0
66	Targeted inhibition of phosphoinositide 3-kinase activity as a novel strategy to normalize Î²-adrenergic receptor function in heart failure. Vascular Pharmacology, 2006, 45, 77-85.	2.1	16
67	beta-Arrestin2-mediated inotropic effects of the angiotensin II type 1A receptor in isolated cardiac myocytes. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103, 16284-16289.	7.1	208
68	From The Cover: Drosophila as a model for the identification of genes causing adult human heart disease. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103, 1394-1399.	7.1	226
69	Intermittent pressure overload triggers hypertrophy-independent cardiac dysfunction and vascular rarefaction. Journal of Clinical Investigation, 2006, 116, 1547-1560.	8.2	220
70	Protein kinase activity of phosphoinositide 3-kinase regulates Î²-adrenergic receptor endocytosis. Nature Cell Biology, 2005, 7, 785-796.	10.3	125
71	Methods for the Detection of Altered Î²-Adrenergic Receptor Signaling Pathways in Hypertrophied Hearts. , 2005, 112, 353-362.		4
72	Restoration of Î²-Adrenergic Receptor Signaling and Contractile Function in Heart Failure by Disruption of the Î²ARK1/Phosphoinositide 3-Kinase Complex. Circulation, 2005, 111, 2579-2587.	1.6	72

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73	Level of Î²-Adrenergic Receptor Kinase 1 Inhibition Determines Degree of Cardiac Dysfunction After Chronic Pressure Overloadâ€"Induced Heart Failure. Circulation, 2005, 111, 591-597.	1.6	65
74	Receptor-Signaling Pathways in Heart Failure. , 2005, , 123-143.		0
75	Targeted Inhibition of Î²-Adrenergic Receptor Kinase-1â€"Associated Phosphoinositide-3 Kinase Activity Preserves Î²-Adrenergic Receptor Signaling and Prolongs Survival in Heart Failure Induced by Calsequestrin Overexpression. Journal of the American College of Cardiology, 2005, 45, 1862-1870.	2.8	48
76	When 7 transmembrane receptors are not G protein-coupled receptors. Journal of Clinical Investigation, 2005, 115, 2971-2974.	8.2	88
77	Network integration of the adrenergic system in cardiac hypertrophy. Cardiovascular Research, 2004, 63, 391-402.	3.8	81
78	The role of G-protein-coupled receptors in heart failure. Drug Discovery Today Disease Mechanisms, 2004, 1, 37-43.	0.8	1
79	Role of Phosphoinositide 3-Kinase in Cardiac Function and Heart Failure. Trends in Cardiovascular Medicine, 2003, 13, 206-212.	4.9	41
80	Sensing heart stress. Nature Medicine, 2003, 9, 19-20.	30.7	16
81	Multiple quantitative trait loci modify the heart failure phenotype in murine cardiomyopathy. Human Molecular Genetics, 2003, 12, 3097-3107.	2.9	32
82	Dual Inhibition of Î²-Adrenergic and Angiotensin II Receptors by a Single Antagonist. Circulation, 2003, 108, 1611-1618.	1.6	236
83	Protein Kinase A and G Protein-coupled Receptor Kinase Phosphorylation Mediates Î²-1 Adrenergic Receptor Endocytosis through Different Pathways. Journal of Biological Chemistry, 2003, 278, 35403-35411.	3.4	140
84	Cardiac hypertrophy and altered Î²-adrenergic signaling in transgenic mice that express the amino terminus of Î²-ARK1. American Journal of Physiology - Heart and Circulatory Physiology, 2003, 285, H2201-H2211.	3.2	21
85	Differential myocardial gene expression in the development and rescue of murine heart failure. Physiological Genomics, 2003, 15, 105-114.	2.3	70
86	Inhibition of receptor-localized PI3K preserves cardiac Î²-adrenergic receptor function and ameliorates pressure overload heart failure. Journal of Clinical Investigation, 2003, 112, 1067-1079.	8.2	117
87	Phosphoinositide 3-kinase regulates Î²2-adrenergic receptor endocytosis by AP-2 recruitment to the receptor/Î²-arrestin complex. Journal of Cell Biology, 2002, 158, 563-575.	5.2	178
88	Genetic Alterations That Inhibit In Vivo Pressure-Overload Hypertrophy Prevent Cardiac Dysfunction Despite Increased Wall Stress. Circulation, 2002, 105, 85-92.	1.6	352
89	Seven-transmembrane-spanning receptors and heart function. Nature, 2002, 415, 206-212.	27.8	862
90	Regulation of myocardial Î²ARK1 expression in catecholamine-induced cardiac hypertrophy in transgenic mice overexpressing Î±1B-adrenergic receptors. Journal of the American College of Cardiology, 2001, 38, 534-540.	2.8	62

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91	Important role of endogenous norepinephrine and epinephrine in the development of in vivo pressure-overload cardiac hypertrophy. Journal of the American College of Cardiology, 2001, 38, 876-882.	2.8	94
92	Augmentation of Cardiac Contractility Mediated by the Human Î² ₃ -Adrenergic Receptor Overexpressed in the Hearts of Transgenic Mice. Circulation, 2001, 104, 2485-2491.	1.6	85
93	Î²-Adrenergic axis and heart disease. Trends in Genetics, 2001, 17, S44-S49.	6.7	38
94	Agonist-dependent Recruitment of Phosphoinositide 3-Kinase to the Membrane by Î²-Adrenergic Receptor Kinase 1. Journal of Biological Chemistry, 2001, 276, 18953-18959.	3.4	168
95	Cardiac Overexpression of a G _q Inhibitor Blocks Induction of Extracellular Signalâ€"Regulated Kinase and c-Jun NH ₂ -Terminal Kinase Activity in In Vivo Pressure Overload. Circulation, 2001, 103, 1453-1458.	1.6	130
96	Cellular and functional defects in a mouse model of heart failure. American Journal of Physiology - Heart and Circulatory Physiology, 2000, 279, H3101-H3112.	3.2	108
97	Dilated cardiomyopathy in transgenic mice expressing a mutant A subunit of protein phosphatase 2A. American Journal of Physiology - Heart and Circulatory Physiology, 2000, 279, H1307-H1318.	3.2	41
98	GÎ²Î³-dependent Phosphoinositide 3-Kinase Activation in Hearts with in Vivo Pressure Overload Hypertrophy. Journal of Biological Chemistry, 2000, 275, 4693-4698.	3.4	183
99	Enhanced Contractility and Decreased Î²-Adrenergic Receptor Kinase-1 in Mice Lacking Endogenous Norepinephrine and Epinephrine. Circulation, 1999, 99, 2702-2707.	1.6	46
100	Defective Î²-Adrenergic Receptor Signaling Precedes the Development of Dilated Cardiomyopathy in Transgenic Mice with Calsequestrin Overexpression. Journal of Biological Chemistry, 1999, 274, 22251-22256.	3.4	113
101	Î²-adrenergic receptor desensitization in cardiac hypertrophy and heart failure. Cell Biochemistry and Biophysics, 1999, 31, 321-329.	1.8	20
102	Role of Î²â€Adrenoceptor Desensitization in Heart Failure. Cardiovascular Drug Reviews, 1999, 17, 384-394.	4.1	2
103	Targeting the Receptor-Gq Interface to Inhibit in Vivo Pressure Overload Myocardial Hypertrophy. Science, 1998, 280, 574-577.	12.6	442
104	Control of Myocardial Contractile Function by the Level of Î²-Adrenergic Receptor Kinase 1 in Gene-targeted Mice. Journal of Biological Chemistry, 1998, 273, 18180-18184.	3.4	153
105	Transgenic Mice with Cardiac Overexpression of Î±1B-Adrenergic Receptors. Journal of Biological Chemistry, 1997, 272, 21253-21259.	3.4	140
106	Mechanism of Î²-Adrenergic Receptor Desensitization in Cardiac Hypertrophy Is Increased Î²-Adrenergic Receptor Kinase. Journal of Biological Chemistry, 1997, 272, 17223-17229.	3.4	241
107	Transthoracic Echocardiography in Models of Cardiac Disease in the Mouse. Circulation, 1996, 94, 1109-1117.	1.6	299
108	Physiological effects of inverse agonists in transgenic mice with myocardial overexpression of the Î²2-adrenoceptor. Nature, 1995, 374, 272-276.	27.8	431