David H Edwards

List of Publications by Year
in descending order

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23 papers	843 citations	687220 13 h-index	677027 22 g-index
23 all docs	23 docs citations	23 times ranked	636 citing authors

#	Article	IF	Citations
1	Evidence that cyclic guanosine monophosphate (cGMP) mediates endothelium-dependent relaxation. European Journal of Pharmacology, 1985, 112, 195-202.	1.7	118
2	Gap junctional communication underpins EDHF-type relaxations evoked by ACh in the rat hepatic artery. American Journal of Physiology - Heart and Circulatory Physiology, 2001, 280, H2441-H2450.	1.5	116
3	cAMP facilitates EDHF-type relaxations in conduit arteries by enhancing electrotonic conduction via gap junctions. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 6392-6397.	3.3	105
4	The obligatory link: role of gap junctional communication in endothelium-dependent smooth muscle hyperpolarization. Pharmacological Research, 2004, 49, 551-564.	3.1	79
5	Connexin-mimetic peptides dissociate electrotonic EDHF-type signalling via myoendothelial and smooth muscle gap junctions in the rabbit iliac artery. British Journal of Pharmacology, 2005, 144, 108-114.	2.7	64
6	Hydrogen Peroxide Potentiates the EDHF Phenomenon by Promoting Endothelial Ca2+Mobilization. Arteriosclerosis, Thrombosis, and Vascular Biology, 2008, 28, 1774-1781.	1.1	58
7	Gap Junction-Dependent Increases in Smooth Muscle cAMP Underpin the EDHF Phenomenon in Rabbit Arteries. Biochemical and Biophysical Research Communications, 2001, 283, 583-589.	1.0	54
8	Distinct hyperpolarizing and relaxant roles for gap junctions and endothelium-derived H2O2 in NO-independent relaxations of rabbit arteries. Proceedings of the National Academy of Sciences of the United States of America, 2003, 100, 15212-15217.	3.3	51
9	5-Methyltetrahydrofolate and tetrahydrobiopterin can modulate electrotonically mediated endothelium-dependent vascular relaxation. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 7008-7013.	3.3	42
10	Ascorbic acid and tetrahydrobiopterin potentiate the EDHF phenomenon by generating hydrogen peroxide. Cardiovascular Research, 2009, 84, 218-226.	1.8	39
11	Massive Accumulation of Myofibroblasts inÂthe Critical Isthmus Is Associated WithÂVentricular Tachycardia Inducibility inÂPost-Infarct Swine Heart. JACC: Clinical Electrophysiology, 2017, 3, 703-714.	1.3	23
12	The effect of inorganic arsenic on endothelium-dependent relaxation: Role of NADPH oxidase and hydrogen peroxide. Toxicology, 2013, 306, 50-58.	2.0	19
13	Influence of endothelium on drug-induced relaxation of the rabbit aorta. European Journal of Pharmacology, 1986, 121, 19-23.	1.7	13
14	Modulation of Gap Junction-Dependent Arterial Relaxation by Ascorbic Acid. Journal of Vascular Research, 2007, 44, 410-422.	0.6	13
15	Activities of endothelinâ€1 in the vascular network of the rabbit ear: a microangiographic study. British Journal of Pharmacology, 1990, 101, 781-788.	2.7	11
16	Enhanced inhibition of the EDHF phenomenon by a phenyl methoxyalaninyl phosphoramidate derivative of dideoxyadenosine. British Journal of Pharmacology, 2004, 142, 27-30.	2.7	11
17	Attenuated store-operated Ca2+ entry underpins the dual inhibition of nitric oxide and EDHF-type relaxations by iodinated contrast media. Cardiovascular Research, 2009, 84, 470-478.	1.8	10
18	A Systemized Approach to Investigate Ca2+ Synchronization in Clusters of Human Induced Pluripotent Stem-Cell Derived Cardiomyocytes. Frontiers in Cell and Developmental Biology, 2015, 3, 89.	1.8	5

#	Article	IF	Citations
19	Vasomotion: the case for chaos. Journal of Biorheology, 2009, 23, 11-23.	0.2	4
20	Robust segmentation of brain structures in MRI. , 2009, , .		3
21	Endogenous Nitric Oxide Synthesis Differentially Modulates Pressure-Flow and Pressure-Conductance Relationships in the Internal and External Carotid Artery Circulations of the Rat. Neurologia Medico-Chirurgica, 2002, 42, 527-535.	1.0	2
22	Local, integrated control of blood flow. Autonomic Neuroscience: Basic and Clinical, 2013, 178, 4-8.	1.4	2
23	Decoding Ca2+ Signals as a Non-electrophysiological Method for Assessing Drug Toxicity in Stem Cell-Derived Cardiomyocytes. Methods in Pharmacology and Toxicology, 2017, , 173-190.	0.1	1