# Pavel Zhabyeyev

## List of Publications by Year in Descending Order



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| 39 papers | 702 citations | 13 h-index | 25 g-index |
| 43 ext. papers | 835 ext. citations | 6 avg, IF | 3.78 L-index |

| # | Paper | IF | Citations |
|---|---|---|---|
| 39 | ADAM15 is required for optimal collagen cross-linking and scar formation following myocardial infarction.. *Matrix Biology*, **2022**, 105, 127-127 | 11.4 | 0 |
| 38 | Soluble Epoxide Hydrolase in Aged Female Mice and Human Explanted Hearts Following Ischemic Injury. *International Journal of Molecular Sciences*, **2021**, 22, | 6.3 | 4 |
| 37 | Sickle cell disease, interleukin-18, and arrhythmias. *Blood*, **2021**, 137, 1138-1139 | 2.2 | 1 |
| 36 | Pharmacological and cell-specific genetic PI3K inhibition worsens cardiac remodeling after myocardial infarction. *Journal of Molecular and Cellular Cardiology*, **2021**, 157, 17-30 | 5.8 | 3 |
| 35 | Gelsolin is an important mediator of Angiotensin II-induced activation of cardiac fibroblasts and fibrosis. *FASEB Journal*, **2021**, 35, e21932 | 0.9 | 0 |
| 34 | Cardiovascular toxicity of PI3K inhibitors. *Clinical Science*, **2020**, 134, 2595-2622 | 6.5 | 4 |
| 33 | Inactivation of endothelial cell phosphoinositide 3-kinase inhibits tumor angiogenesis and tumor growth. *Oncogene*, **2020**, 39, 6480-6492 | 9.2 | 5 |
| 32 | Inhibition of PI3Kinase- is pro-arrhythmic and associated with enhanced late Na current, contractility, and Ca release in murine hearts. *Journal of Molecular and Cellular Cardiology*, **2019**, 132, 98-109 | 5.8 | 10 |
| 31 | PI3K Pathway Inhibition With Doxorubicin Treatment Results in Distinct Biventricular Atrophy and Remodeling With Right Ventricular Dysfunction. *Journal of the American Heart Association*, **2019**, 8, e010961 | 6 | 8 |
| 30 | Role of iron metabolism in heart failure: From iron deficiency to iron overload. *Biochimica Et Biophysica Acta - Molecular Basis of Disease*, **2019**, 1865, 1925-1937 | 6.9 | 42 |
| 29 | Investigating the role of endothelial cell-specific p110 isoform of PI3K as a potential target for anti-angiogenic therapy. *FASEB Journal*, **2019**, 33, lb9 | 0.9 | |
| 28 | Testosterone and cardiac remodeling: why are older men susceptible to heart disease?. *American Journal of Physiology - Heart and Circulatory Physiology*, **2019**, 316, H765-H767 | 5.2 | 1 |
| 27 | PI3K in cardioprotection: Cytoskeleton, late Na current, and mechanism of arrhythmias. *Channels*, **2019**, 13, 520-532 | 3 | 7 |
| 26 | Endothelial and cardiomyocyte PI3K divergently regulate cardiac remodelling in response to ischaemic injury. *Cardiovascular Research*, **2019**, 115, 1343-1356 | 9.9 | 13 |
| 25 | TIMP3 deficiency exacerbates iron overload-mediated cardiomyopathy and liver disease. *American Journal of Physiology - Heart and Circulatory Physiology*, **2018**, 314, H978-H990 | 5.2 | 13 |
| 24 | Advanced iron-overload cardiomyopathy in a genetic murine model is rescued by resveratrol therapy. *Bioscience Reports*, **2018**, 38, | 4.1 | 8 |
| 23 | PI3K regulated gelsolin activity is a critical determinant of cardiac cytoskeletal remodeling and heart disease. *Nature Communications*, **2018**, 9, 5390 | 17.4 | 34 |

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| 22 | Unravelling the molecular basis for cardiac iron metabolism and deficiency in heart failure. *European Heart Journal*, **2017**, 38, 373-375 | 9.5 | 11 |
| 21 | PI3Kγ is essential for the recovery from Cre/tamoxifen cardiotoxicity and in myocardial insulin signalling but is not required for normal myocardial contractility in the adult heart. *Cardiovascular Research*, **2015**, 105, 292-303 | 9.9 | 13 |
| 20 | Iron-overload injury and cardiomyopathy in acquired and genetic models is attenuated by resveratrol therapy. *Scientific Reports*, **2015**, 5, 18132 | 4.9 | 63 |
| 19 | Dual loss of PI3Kγ and PI3Kδ signaling leads to an age-dependent cardiomyopathy. *Journal of Molecular and Cellular Cardiology*, **2014**, 77, 155-9 | 5.8 | 5 |
| 18 | Role of sex steroids and sexual dimorphism on cardiac iron metabolism in iron-overload cardiomyopathy. *Translational Research*, **2014**, 163, 141-4 | 11 | 2 |
| 17 | PI3K inhibitors as novel cancer therapies: implications for cardiovascular medicine. *Journal of Cardiac Failure*, **2013**, 19, 268-82 | 3.3 | 22 |
| 16 | Enhanced recovery from ischemia-reperfusion injury in PI3Kγ dominant negative hearts: investigating the role of alternate PI3K isoforms, increased glucose oxidation and MAPK signaling. *Journal of Molecular and Cellular Cardiology*, **2013**, 54, 9-18 | 5.8 | 13 |
| 15 | S4153R is a gain-of-function mutation in the cardiac Ca(2+) release channel ryanodine receptor associated with catecholaminergic polymorphic ventricular tachycardia and paroxysmal atrial fibrillation. *Canadian Journal of Cardiology*, **2013**, 29, 993-6 | 3.8 | 31 |
| 14 | Pressure-overload-induced heart failure induces a selective reduction in glucose oxidation at physiological afterload. *Cardiovascular Research*, **2013**, 97, 676-85 | 9.9 | 85 |
| 13 | Loss of p47phox subunit enhances susceptibility to biomechanical stress and heart failure because of dysregulation of cortactin and actin filaments. *Circulation Research*, **2013**, 112, 1542-56 | 15.7 | 47 |
| 12 | Loss of Apelin exacerbates myocardial infarction adverse remodeling and ischemia-reperfusion injury: therapeutic potential of synthetic Apelin analogues. *Journal of the American Heart Association*, **2013**, 2, e000249 | 6 | 142 |
| 11 | Osmotic modulation of slowly activating IKs in guinea-pig ventricular myocytes. *Cardiovascular Research*, **2011**, 91, 429-36 | 9.9 | 3 |
| 10 | Electroporation-induced inward current in voltage-clamped guinea pig ventricular myocytes. *Journal of Membrane Biology*, **2010**, 238, 69-80 | 2.3 | 16 |
| 9 | Ultraviolet photoalteration of late Na+ current in guinea-pig ventricular myocytes. *Journal of Membrane Biology*, **2006**, 210, 43-50 | 2.3 | 7 |
| 8 | Insensitivity of cardiac delayed-rectifier I(Kr) to tyrosine phosphorylation inhibitors and stimulators. *British Journal of Pharmacology*, **2006**, 148, 724-31 | 8.6 | 3 |
| 7 | Selective block of swelling-activated Cl- channels over cAMP-dependent Cl- channels in ventricular myocytes. *European Journal of Pharmacology*, **2004**, 491, 111-20 | 5.3 | 4 |
| 6 | Inward-rectifier K+ current in guinea-pig ventricular myocytes exposed to hyperosmotic solutions. *Journal of Membrane Biology*, **2004**, 202, 151-60 | 2.3 | 2 |
| 5 | Transient outward current carried by inwardly rectifying K+ channels in guinea pig ventricular myocytes dialyzed with low-K+ solution. *American Journal of Physiology - Cell Physiology*, **2004**, 287, C1396-403 | 5.4 | 7 |

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| 4 | Block of cardiac delayed-rectifier and inward-rectifier K+ currents by nisoldipine. *British Journal of Pharmacology*, **2003**, 140, 863-70 | 8.6 | 14 |
| 3 | Differences in the effects of urinary incontinence agents S-oxybutynin and terodiline on cardiac K(+) currents and action potentials. *British Journal of Pharmacology*, **2000**, 131, 245-54 | 8.6 | 14 |
| 2 | Selective phenylalkylamine block of I(Kr) over other K(+) currents in guinea-pig ventricular myocytes. *British Journal of Pharmacology*, **2000**, 131, 1809-16 | 8.6 | 9 |
| 1 | Low-affinity block of cardiac K(+) currents by nifedipine. *European Journal of Pharmacology*, **2000**, 401, 137-43 | 5.3 | 26 |