Zhengfeng Zhou

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Properties of HERG Channels Stably Expressed in HEK 293 Cells Studied at Physiological Temperature. Biophysical Journal, 1998, 74, 230-241.	0.5	687
2	Most LQT2 Mutations Reduce Kv11.1 (hERG) Current by a Class 2 (Trafficking-Deficient) Mechanism. Circulation, 2006, 113, 365-373.	1.6	363
3	HERG Channel Dysfunction in Human Long QT Syndrome. Journal of Biological Chemistry, 1998, 273, 21061-21066.	3.4	354
4	Nonsense Mutations in hERG Cause a Decrease in Mutant mRNA Transcripts by Nonsense-Mediated mRNA Decay in Human Long-QT Syndrome. Circulation, 2007, 116, 17-24.	1.6	111
5	Degradation of Trafficking-defective Long QT Syndrome Type II Mutant Channels by the Ubiquitin-Proteasome Pathway. Journal of Biological Chemistry, 2005, 280, 19419-19425.	3.4	99
6	Brief Report: Oxidative Stress Mediates Cardiomyocyte Apoptosis in a Human Model of Danon Disease and Heart Failure. Stem Cells, 2015, 33, 2343-2350.	3.2	74
7	Pathogenesis of the Novel Autoimmune-Associated Long-QT Syndrome. Circulation, 2015, 132, 230-240.	1.6	62
8	Recurrent intrauterine fetal loss due to near absence of HERG: Clinical and functional characterization of a homozygous nonsense HERG Q1070X mutation. Heart Rhythm, 2008, 5, 553-561.	0.7	58
9	Defective assembly and trafficking of mutant HERG channels with C-terminal truncations in long QT syndrome. Journal of Molecular and Cellular Cardiology, 2004, 37, 1225-33.	1.9	47
10	Nonsense-mediated mRNA decay caused by a frameshift mutation in a large kindred of type 2 long QT syndrome. Heart Rhythm, 2011, 8, 1200-1206.	0.7	27
11	Inhibition of nonsense-mediated mRNA decay by antisense morpholino oligonucleotides restores functional expression of hERG nonsense and frameshift mutations in long-QT syndrome. Journal of Molecular and Cellular Cardiology, 2011, 50, 223-229.	1.9	25
12	Early LQT2 nonsense mutation generates N-terminally truncated hERG channels with altered gating properties by the reinitiation of translation. Journal of Molecular and Cellular Cardiology, 2012, 53, 725-733.	1.9	24
13	A splice site mutation in hERG leads to cryptic splicing in human long QT syndrome. Journal of Molecular and Cellular Cardiology, 2008, 44, 502-509.	1.9	20
14	Alternative Splicing and Polyadenylation Contribute to the Generation of hERG1 C-terminal Isoforms. Journal of Biological Chemistry, 2010, 285, 32233-32241.	3.4	19
15	LQT2 nonsense mutations generate trafficking defective NH ₂ -terminally truncated channels by the reinitiation of translation. American Journal of Physiology - Heart and Circulatory Physiology, 2013, 305, H1397-H1404.	3.2	18
16	Position of premature termination codons determines susceptibility of hERG mutations to nonsense-mediated mRNA decay in long QT syndrome. Gene, 2014, 539, 190-197.	2.2	15
17	Nonsense-Mediated mRNA Decay of hERG Mutations in Long QT Syndrome. Methods in Molecular Biology, 2018, 1684, 37-49.	0.9	13
18	lsoform-Specific Dominant-Negative Effects Associated with hERG1 G628S Mutation in Long QT Syndrome. PLoS ONE, 2012, 7, e42552.	2.5	9

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#	Article	IF	CITATIONS
19	Upregulation of functional Kv11.1 isoform expression by inhibition of intronic polyadenylation with antisense morpholino oligonucleotides. Journal of Molecular and Cellular Cardiology, 2014, 76, 26-32.	1.9	8
20	Identification of Kv11.1 Isoform Switch as a Novel Pathogenic Mechanism of Long-QT Syndrome. Circulation: Cardiovascular Genetics, 2014, 7, 482-490.	5.1	7
21	Regulation of Isoform Expression by Blocking Polyadenylation Signal Sequences with Morpholinos. Methods in Molecular Biology, 2017, 1565, 141-150.	0.9	4
22	Upregulation of functional Kv11.1a isoform expression by modified U1 small nuclear RNA. Gene, 2018, 641, 220-225.	2.2	4
23	Regulation of Kv11.1 potassium channel C-terminal isoform expression by the RNA-binding proteins HuR and HuD. Journal of Biological Chemistry, 2018, 293, 19624-19632.	3.4	4
24	Regulation of Kv11.1 Isoform Expression by Polyadenylate Binding Protein Nuclear 1. International Journal of Molecular Sciences, 2021, 22, 863.	4.1	3