

Christophe Aj Girard

List of Publications by Year in descending order

Source: <https://exaly.com/author-pdf/4578832/publications.pdf>

Version: 2024-02-01

32
papers

4,670
citations

185998

28
h-index

360668

35
g-index

36
all docs

36
docs citations

36
times ranked

5935
citing authors

#	ARTICLE	IF	CITATIONS
1	Secretion of IL1 by Dedifferentiated Melanoma Cells Inhibits JAK1-STAT3-Driven Actomyosin Contractility of Lymph Node Fibroblastic Reticular Cells. <i>Cancer Research</i> , 2022, 82, 1774-1788.	0.4	12
2	Inhibition of Patched Drug Efflux Increases Vemurafenib Effectiveness against Resistant BrafV600E Melanoma. <i>Cancers</i> , 2020, 12, 1500.	1.7	9
3	A Feed-Forward Mechanosignaling Loop Confers Resistance to Therapies Targeting the MAPK Pathway in BRAF-Mutant Melanoma. <i>Cancer Research</i> , 2020, 80, 1927-1941.	0.4	46
4	Cancer cell-derived long pentraxin 3 (PTX3) promotes melanoma migration through a toll-like receptor 4 (TLR4)/NF- κ B signaling pathway. <i>Oncogene</i> , 2019, 38, 5873-5889.	2.6	71
5	Epitope Spreading of Autoantibody Response to PLA2R Associates with Poor Prognosis in Membranous Nephropathy. <i>Journal of the American Society of Nephrology: JASN</i> , 2016, 27, 1517-1533.	3.0	161
6	PLA2R1 kills cancer cells by inducing mitochondrial stress. <i>Free Radical Biology and Medicine</i> , 2013, 65, 969-977.	1.3	33
7	Group X Secreted Phospholipase A2 Limits the Development of Atherosclerosis in LDL Receptor-Null Mice. <i>Arteriosclerosis, Thrombosis, and Vascular Biology</i> , 2013, 33, 466-473.	1.1	60
8	PLA2R1 Mediates Tumor Suppression by Activating JAK2. <i>Cancer Research</i> , 2013, 73, 6334-6345.	0.4	60
9	Control of Pancreatic β Cell Regeneration by Glucose Metabolism. <i>Cell Metabolism</i> , 2011, 13, 440-449.	7.2	266
10	Overexpression of Fto leads to increased food intake and results in obesity. <i>Nature Genetics</i> , 2010, 42, 1086-1092.	9.4	612
11	Emerging roles of secreted phospholipase A2 enzymes: Lessons from transgenic and knockout mice. <i>Biochimie</i> , 2010, 92, 561-582.	1.3	141
12	Adjacent mutations in the gating loop of Kir6.2 produce neonatal diabetes and hyperinsulinism. <i>EMBO Molecular Medicine</i> , 2009, 1, 166-177.	3.3	36
13	Expression of an activating mutation in the gene encoding the KATP channel subunit Kir6.2 in mouse pancreatic β cells recapitulates neonatal diabetes. <i>Journal of Clinical Investigation</i> , 2009, 119, 80-90.	3.9	95
14	Mosaic Paternal Uniparental Isodisomy and an ABCC8 Gene Mutation in a Patient With Permanent Neonatal Diabetes and Hemihypertrophy. <i>Diabetes</i> , 2008, 57, 255-258.	0.3	15
15	Mechanism of action of a sulphonylurea receptor SUR1 mutation (F132L) that causes DEND syndrome. <i>Human Molecular Genetics</i> , 2007, 16, 2011-2019.	1.4	51
16	Permanent Neonatal Diabetes Caused by Dominant, Recessive, or Compound Heterozygous SUR1 Mutations with Opposite Functional Effects. <i>American Journal of Human Genetics</i> , 2007, 81, 375-382.	2.6	194
17	The Obesity-Associated <i>FTO</i> Gene Encodes a 2-Oxoglutarate-Dependent Nucleic Acid Demethylase. <i>Science</i> , 2007, 318, 1469-1472.	6.0	1,305
18	Sulfonylurea improves CNS function in a case of intermediate DEND syndrome caused by a mutation in KCNJ11. <i>Nature Clinical Practice Neurology</i> , 2007, 3, 640-645.	2.7	102

#	ARTICLE	IF	CITATIONS
19	Functional analysis of two Kir6.2 (KCNJ11) mutations, K170T and E322K, causing neonatal diabetes. <i>Diabetes, Obesity and Metabolism</i> , 2007, 9, 46-55.	2.2	21
20	Functional analysis of six Kir6.2 (KCNJ11) mutations causing neonatal diabetes. <i>Pflugers Archiv European Journal of Physiology</i> , 2006, 453, 323-332.	1.3	53
21	Functional Effects of Mutations at F35 in the NH2-terminus of Kir6.2 (KCNJ11), Causing Neonatal Diabetes, and Response to Sulfonylurea Therapy. <i>Diabetes</i> , 2006, 55, 1731-1737.	0.3	41
22	A heterozygous activating mutation in the sulphonylurea receptor SUR1 (ABCC8) causes neonatal diabetes. <i>Human Molecular Genetics</i> , 2006, 15, 1793-1800.	1.4	196
23	ATP Sensitivity of the ATP-Sensitive K ⁺ Channel in Intact and Permeabilized Pancreatic β^2 -Cells. <i>Diabetes</i> , 2006, 55, 2446-2454.	0.3	68
24	Mutations at the Same Residue (R50) of Kir6.2 (KCNJ11) That Cause Neonatal Diabetes Produce Different Functional Effects. <i>Diabetes</i> , 2006, 55, 1705-1712.	0.3	64
25	Kir6.2 mutations causing neonatal diabetes provide new insights into Kir6.2-SUR1 interactions. <i>EMBO Journal</i> , 2005, 24, 2318-2330.	3.5	63
26	A gating mutation at the internal mouth of the Kir6.2 pore is associated with DEND syndrome. <i>EMBO Reports</i> , 2005, 6, 470-475.	2.0	99
27	Pancreatic two P domain K ⁺ channels TALK-1 and TALK-2 are activated by nitric oxide and reactive oxygen species. <i>Journal of Physiology</i> , 2005, 562, 235-244.	1.3	66
28	Relapsing diabetes can result from moderately activating mutations in KCNJ11. <i>Human Molecular Genetics</i> , 2005, 14, 925-934.	1.4	184
29	Functional effects of KCNJ11 mutations causing neonatal diabetes: enhanced activation by MgATP. <i>Human Molecular Genetics</i> , 2005, 14, 2717-2726.	1.4	74
30	Mechanisms underlying excitatory effects of group I metabotropic glutamate receptors via inhibition of 2P domain K ⁺ channels. <i>EMBO Journal</i> , 2003, 22, 5403-5411.	3.5	171
31	p11, an annexin II subunit, an auxiliary protein associated with the background K ⁺ channel, TASK-1. <i>EMBO Journal</i> , 2002, 21, 4439-4448.	3.5	133
32	Genomic and Functional Characteristics of Novel Human Pancreatic 2P Domain K ⁺ Channels. <i>Biochemical and Biophysical Research Communications</i> , 2001, 282, 249-256.	1.0	157