

Emmanuelle Masson

List of Publications by Year in descending order

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Version: 2024-02-01

31
papers

1,618
citations

471509

17
h-index

454955

30
g-index

34
all docs

34
docs citations

34
times ranked

1505
citing authors

#	ARTICLE	IF	CITATIONS
1	NGS mismapping confounds the clinical interpretation of the <i>PRSS1</i> p.Ala16Val (c.47C>T) variant in chronic pancreatitis. <i>Gut</i> , 2022, 71, 841-842.	12.1	8
2	Trypsinogen (<i>PRSS1</i> and <i>PRSS2</i>) gene dosage correlates with pancreatitis risk across genetic and transgenic studies: a systematic review and re-analysis. <i>Human Genetics</i> , 2022, 141, 1327-1338.	3.8	8
3	Functionally deficient <i>TRPV6</i> variants contribute to hereditary and familial chronic pancreatitis. <i>Human Mutation</i> , 2022, 43, 228-239.	2.5	7
4	Scale and Scope of Gene-Alcohol Interactions in Chronic Pancreatitis: A Systematic Review. <i>Genes</i> , 2021, 12, 471.	2.4	9
5	The three common polymorphisms p.A986S, p.R990G and p.Q1011E in the calcium sensing receptor (<i>CASR</i>) are not associated with chronic pancreatitis. <i>Pancreatology</i> , 2021, 21, 1299-1304.	1.1	3
6	Splicing Outcomes of 5' Splice Site GT>GC Variants That Generate Wild-Type Transcripts Differ Significantly Between Full-Length and Minigene Splicing Assays. <i>Frontiers in Genetics</i> , 2021, 12, 701652.	2.3	9
7	The corrected breakpoint sequence of the homozygous <i>SPINK1</i> deletion causing severe infantile isolated exocrine pancreatic insufficiency. <i>Human Mutation</i> , 2021, 42, 216-217.	2.5	0
8	Variants That Affect Function of Calcium Channel <i>TRPV6</i> Are Associated With Early-Onset Chronic Pancreatitis. <i>Gastroenterology</i> , 2020, 158, 1626-1641.e8.	1.3	77
9	Analysis of <i>GPRC6A</i> variants in different pancreatitis etiologies. <i>Pancreatology</i> , 2020, 20, 1262-1267.	1.1	1
10	Role of the Common <i>PRSS1-PRSS2</i> Haplotype in Alcoholic and Non-Alcoholic Chronic Pancreatitis: Meta- and Re-Analyses. <i>Genes</i> , 2020, 11, 1349.	2.4	14
11	5' splice site GC>GT and GT>GC variants differ markedly in terms of their functionality and pathogenicity. <i>Human Mutation</i> , 2020, 41, 1358-1364.	2.5	7
12	The Experimentally Obtained Functional Impact Assessments of 5' Splice Site GT>GC Variants Differ Markedly from Those Predicted. <i>Current Genomics</i> , 2020, 21, 56-66.	1.6	16
13	First estimate of the scale of canonical 5' splice site GT>GC variants capable of generating wild-type transcripts. <i>Human Mutation</i> , 2019, 40, 1856-1873.	2.5	25
14	Toward a clinical diagnostic pipeline for <i>SPINK1</i> intronic variants. <i>Human Genomics</i> , 2019, 13, 8.	2.9	8
15	Protease-Sensitive Pancreatic Lipase Variants Are Associated With Early Onset Chronic Pancreatitis. <i>American Journal of Gastroenterology</i> , 2019, 114, 974-983.	0.4	48
16	<i>PRSS1</i> copy number variants and promoter polymorphisms in pancreatitis: common pathogenetic mechanism, different genetic effects. <i>Gut</i> , 2018, 67, 592-593.	12.1	12
17	Genome-wide association study identifies inversion in the <i>CTRB1-CTRB2</i> locus to modify risk for alcoholic and non-alcoholic chronic pancreatitis. <i>Gut</i> , 2018, 67, 1855-1863.	12.1	97
18	<i>SPINK1</i> , <i>PRSS1</i> , <i>CTRC</i> , and <i>CFTR</i> Genotypes Influence Disease Onset and Clinical Outcomes in Chronic Pancreatitis. <i>Clinical and Translational Gastroenterology</i> , 2018, 9, e204.	2.5	76

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19	Severe infantile isolated exocrine pancreatic insufficiency caused by the complete functional loss of the <i>SPINK1</i> gene. <i>Human Mutation</i> , 2017, 38, 1660-1665.	2.5	24
20	Clarifying the clinical relevance of <i>SPINK1</i> intronic variants in chronic pancreatitis. <i>Gut</i> , 2016, 65, 884-886.	12.1	32
21	Overrepresentation of Rare CASR Coding Variants in a Sample of Young French Patients With Idiopathic Chronic Pancreatitis. <i>Pancreas</i> , 2015, 44, 996-998.	1.1	15
22	A recombined allele of the lipase gene <i>CEL</i> and its pseudogene <i>CELP</i> confers susceptibility to chronic pancreatitis. <i>Nature Genetics</i> , 2015, 47, 518-522.	21.4	157
23	Identification of a functional <i>PRSS1</i> promoter variant in linkage disequilibrium with the chronic pancreatitis-protecting rs10273639. <i>Gut</i> , 2015, 64, 1837-1838.	12.1	35
24	Polymorphisms at <i>PRSS1</i> and <i>PRSS2</i> and <i>CLDN2</i> and <i>MORC4</i> loci associate with alcoholic and non-alcoholic chronic pancreatitis in a European replication study. <i>Gut</i> , 2015, 64, 1426-1433.	12.1	105
25	Variants in <i>CPA1</i> are strongly associated with early onset chronic pancreatitis. <i>Nature Genetics</i> , 2013, 45, 1216-1220.	21.4	255
26	A Conservative Assessment of the Major Genetic Causes of Idiopathic Chronic Pancreatitis: Data from a Comprehensive Analysis of <i>PRSS1</i> , <i>SPINK1</i> , <i>CTRC</i> and <i>CFTR</i> Genes in 253 Young French Patients. <i>PLoS ONE</i> , 2013, 8, e73522.	2.5	89
27	Elucidation of the complex structure and origin of the human trypsinogen locus triplication. <i>Human Molecular Genetics</i> , 2009, 18, 3605-3614.	2.9	22
28	Association of rare chymotrypsinogen C (<i>CTRC</i>) gene variations in patients with idiopathic chronic pancreatitis. <i>Human Genetics</i> , 2008, 123, 83-91.	3.8	159
29	Hereditary pancreatitis caused by a double gain-of-function trypsinogen mutation. <i>Human Genetics</i> , 2008, 123, 521-529.	3.8	42
30	Trypsinogen Copy Number Mutations in Patients With Idiopathic Chronic Pancreatitis. <i>Clinical Gastroenterology and Hepatology</i> , 2008, 6, 82-88.	4.4	75
31	Hereditary pancreatitis caused by triplication of the trypsinogen locus. <i>Nature Genetics</i> , 2006, 38, 1372-1374.	21.4	182