

Balázs Csaba Németh

List of Publications by Year in descending order

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Version: 2024-02-01

17
papers

415
citations

933447

10
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888059

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17
all docs

17
docs citations

17
times ranked

713
citing authors

#	ARTICLE	IF	CITATIONS
1	Hypertriglyceridemia-induced acute pancreatitis: A prospective, multicenter, international cohort analysis of 716 acute pancreatitis cases. <i>Pancreatology</i> , 2020, 20, 608-616.	1.1	73
2	Antibiotic therapy in acute pancreatitis: From global overuse to evidence based recommendations. <i>Pancreatology</i> , 2019, 19, 488-499.	1.1	70
3	Human cationic trypsinogen (<i>PRSS1</i>) variants and chronic pancreatitis. <i>American Journal of Physiology - Renal Physiology</i> , 2014, 306, G466-G473.	3.4	68
4	EASYAPP: An artificial intelligence model and application for early and easy prediction of severity in acute pancreatitis. <i>Clinical and Translational Medicine</i> , 2022, 12, .	4.0	37
5	Relevance of Î±-defensins (HNP1-3) and defensin Î²-1 in diabetes. <i>World Journal of Gastroenterology</i> , 2014, 20, 9128-37.	3.3	29
6	Autoactivation of Mouse Trypsinogens Is Regulated by Chymotrypsin C via Cleavage of the Autolysis Loop. <i>Journal of Biological Chemistry</i> , 2013, 288, 24049-24062.	3.4	28
7	Chronic pancreatitis: Multicentre prospective data collection and analysis by the Hungarian Pancreatic Study Group. <i>PLoS ONE</i> , 2017, 12, e0171420.	2.5	23
8	Misfolding cationic trypsinogen variant p.L104P causes hereditary pancreatitis. <i>Gut</i> , 2017, 66, 1727-1728.	12.1	22
9	Genetic Analysis of Human Chymotrypsin-Like Elastases 3A and 3B (CELA3A and CELA3B) to Assess the Role of Complex Formation between Proelastases and Procarboxypeptidases in Chronic Pancreatitis. <i>International Journal of Molecular Sciences</i> , 2016, 17, 2148.	4.1	13
10	Natural single-nucleotide deletion in chymotrypsinogen C gene increases severity of secretagogue-induced pancreatitis in C57BL/6 mice. <i>JCI Insight</i> , 2019, 4, e129717.	5.0	13
11	Novel p.K374E variant of CPA1 causes misfolding-induced hereditary pancreatitis with autosomal dominant inheritance. <i>Gut</i> , 2020, 69, 790-792.	12.1	11
12	SPINK1 Promoter Variants in Chronic Pancreatitis. <i>Pancreas</i> , 2016, 45, 148-153.	1.1	10
13	Novel PRSS1 Mutation p.P17T Validates Pathogenic Relevance of CTRC-Mediated Processing of the Trypsinogen Activation Peptide in Chronic Pancreatitis. <i>American Journal of Gastroenterology</i> , 2017, 112, 1896-1898.	0.4	7
14	Recurrent acute pancreatitis induced by 5-ASA and azathioprine in ulcerative colitis. <i>Pancreatology</i> , 2020, 20, 1656-1660.	1.1	5
15	Risk of chronic pancreatitis in carriers of loss-of-function CTRC variants: A meta-analysis. <i>PLoS ONE</i> , 2022, 17, e0268859.	2.5	3
16	Evolutionary expansion of polyaspartate motif in the activation peptide of mouse cationic trypsinogen limits autoactivation and protects against pancreatitis. <i>American Journal of Physiology - Renal Physiology</i> , 2021, 321, G719-G734.	3.4	2
17	Evaluation of the Pathogenic Significance of the Novel p.T58M Chymotrypsin C Variant in Recurrent Acute Pancreatitis. <i>Pancreas</i> , 2019, 48, e12-e14.	1.1	1