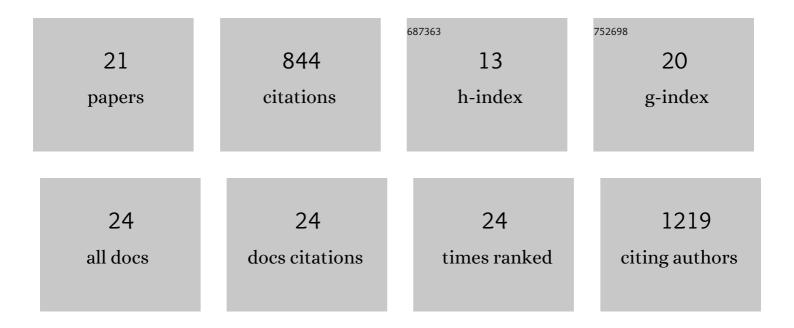
## Ashish Kapoor

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/9085390/publications.pdf Version: 2024-02-01



#	Article	IF	CITATIONS
1	Multiple, independent, common variants at RET, SEMA3 and NRG1 gut enhancers specify Hirschsprung disease risk in European ancestry subjects. Journal of Pediatric Surgery, 2021, 56, 2286-2294.	1.6	3
2	Sequence-based correction of barcode bias in massively parallel reporter assays. Genome Research, 2021, 31, 1638-1645.	5.5	3
3	A multi-enhancer <i>RET</i> regulatory code is disrupted in Hirschsprung disease. Genome Research, 2021, 31, 2199-2208.	5.5	10
4	Combined Genetic Effects of RET and NRG1 Susceptibility Variants on Multifactorial Hirschsprung Disease in Indonesia. Journal of Surgical Research, 2019, 233, 96-99.	1.6	16
5	Multiple SCN5A variant enhancers modulate its cardiac gene expression and the QT interval. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 10636-10645.	7.1	22
6	Molecular Genetic Anatomy and Risk Profile of Hirschsprung's Disease. New England Journal of Medicine, 2019, 380, 1421-1432.	27.0	131
7	Human cardiac <i>cis</i> -regulatory elements, their cognate transcription factors, and regulatory DNA sequence variants. Genome Research, 2018, 28, 1577-1588.	5.5	25
8	Testing the Ret and Sema3d genetic interaction in mouse enteric nervous system development. Human Molecular Genetics, 2017, 26, 1811-1820.	2.9	8
9	Enhancer Variants Synergistically Drive Dysfunction of a Gene Regulatory Network In Hirschsprung Disease. Cell, 2016, 167, 355-368.e10.	28.9	112
10	<i>Trans</i> -ethnic meta-analysis of genome-wide association studies for Hirschsprung disease. Human Molecular Genetics, 2016, 25, ddw333.	2.9	38
11	Rare coding TTN variants are associated with electrocardiographic QT interval in the general population. Scientific Reports, 2016, 6, 28356.	3.3	6
12	Population variation in total genetic risk of Hirschsprung disease from common RET, SEMA3 and NRG1 susceptibility polymorphisms. Human Molecular Genetics, 2015, 24, 2997-3003.	2.9	66
13	Functional Loss of Semaphorin 3C and/or Semaphorin 3D and Their Epistatic Interaction with Ret Are Critical to Hirschsprung Disease Liability. American Journal of Human Genetics, 2015, 96, 581-596.	6.2	118
14	Effects of RET and NRG1 polymorphisms in Indonesian patients with Hirschsprung disease. Journal of Pediatric Surgery, 2014, 49, 1614-1618.	1.6	37
15	Generation of a cre recombinase-conditional Nos1ap over-expression transgenic mouse. Biotechnology Letters, 2014, 36, 1179-1185.	2.2	3
16	An Enhancer Polymorphism at the Cardiomyocyte Intercalated Disc Protein NOS1AP Locus Is a Major Regulator of the QT Interval. American Journal of Human Genetics, 2014, 94, 854-869.	6.2	72
17	Mendelian Puzzles. Science, 2012, 335, 930-931.	12.6	17

18 Genetics and Genomics in Cardiovascular Gene Discovery. , 2012, , 231-259.

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#	Article	IF	CITATIONS
19	An idiopathic epilepsy syndrome linked to 3q13.3â€q21 and missense mutations in the extracellular calcium sensing receptor gene. Annals of Neurology, 2008, 64, 158-167.	5.3	65
20	A Multicenter Study of BRD2 as a Risk Factor for Juvenile Myoclonic Epilepsy. Epilepsia, 2007, 48, 706-712.	5.1	76
21	A novel genetic locus for juvenile myoclonic epilepsy at chromosome 5q12–q14. Human Genetics, 2007, 121, 655-662.	3.8	12