Daniel M Jordan

List of Publications by Year
in descending order

26	5,808	471509 17	610901 24
papers	citations	h-index	g-index

35	35	35	16641
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Overcoming constraints on the detection of recessive selection in human genes from population frequency data. American Journal of Human Genetics, 2022, 109, 33-49.	6.2	5
2	Population-Based Penetrance of Deleterious Clinical Variants. JAMA - Journal of the American Medical Association, 2022, 327, 350.	7.4	34
3	Exploiting the GTEx resources to decipher the mechanisms at GWAS loci. Genome Biology, 2021, 22, 49.	8.8	150
4	An integrative multiomic network model links lipid metabolism to glucose regulation in coronary artery disease. Nature Communications, 2021, 12, 547.	12.8	35
5	Probing the aggregated effects of purifying selection per individual on 1,380 medical phenotypes in the UK Biobank. PLoS Genetics, 2021, 17, e1009337.	3.5	2
6	HOPS: a quantitative score reveals pervasive horizontal pleiotropy in human genetic variation is driven by extreme polygenicity of human traits and diseases. Genome Biology, 2019, 20, 222.	8.8	47
7	Quantification of frequency-dependent genetic architectures in 25 UK Biobank traits reveals action of negative selection. Nature Communications, 2019, 10, 790.	12.8	98
8	Reply to â€˜Selective effects of heterozygous protein-truncating variantsâ€™. Nature Genetics, 2019, 51, 3-4.	21.4	6
9	No causal effects of serum urate levels on the risk of chronic kidney disease: A Mendelian randomization study. PLoS Medicine, 2019, 16, e1002725.	8.4	97
10	Using Full Genomic Information to Predict Disease: Breaking Down the Barriers Between Complex and Mendelian Diseases. Annual Review of Genomics and Human Genetics, 2018, 19, 289-301.	6.2	9
11	A literature review at genome scale: improving clinical variant assessment. Genetics in Medicine, 2018, 20, 936-941.	2.4	1
12	Estimating the selective effects of heterozygous protein-truncating variants from human exome data. Nature Genetics, 2017, 49, 806-810.	21.4	157
13	When â€œNof 2â€•is not enough: integrating statistical and functional data in gene discovery. Journal of Physical Education and Sports Management, 2017, 3, a001099.	1.2	2
14	Excess of Deleterious Mutations around HLA Genes Reveals Evolutionary Cost of Balancing Selection. Molecular Biology and Evolution, 2016, 33, 2555-2564.	8.9	55
15	Mitigating False-Positive Associations in Rare Disease Gene Discovery. Human Mutation, 2015, 36, 998-1003.	2.5	18
16	Disproportionate Contributions of Select Genomic Compartments and Cell Types to Genetic Risk for Coronary Artery Disease. PLoS Genetics, 2015, 11, e1005622.	3.5	70
17	Identification of cis-suppression of human disease mutations by comparative genomics. Nature, 2015, 524, 225-229.	27.8	106
18	Widespread Macromolecular Interaction Perturbations in Human Genetic Disorders. Cell, 2015, 161, 647-660.	28.9	482

#	Article	IF	Citations
19	Genome analysis reveals insights into physiology and longevity of the Brandtâ€™s bat Myotis brandtii. Nature Communications, 2013, 4, 2212.	12.8	213
20	Predicting Functional Effect of Human Missense Mutations Using PolyPhenâ€2. Current Protocols in Human Genetics, 2013, 76, Unit7.20.	3.5	2,389
21	Large Numbers of Genetic Variants Considered to be Pathogenic are Common in Asymptomatic Individuals. Human Mutation, 2013, 34, 1216-1220.	2.5	78
22	Evolution and Functional Impact of Rare Coding Variation from Deep Sequencing of Human Exomes. Science, 2012, 337, 64-69.	12.6	1,535
23	Development and Validation of a Computational Method for Assessment of Missense Variants in Hypertrophic Cardiomyopathy. American Journal of Human Genetics, 2011, 88, 183-192.	6.2	73
24	Human allelic variation: perspective from protein function, structure, and evolution. Current Opinion in Structural Biology, 2010, 20, 342-350.	5.7	63
25	Parameterization of Peptide13C Carbonyl Chemical Shielding Anisotropy in Molecular Dynamics Simulations. ChemPhysChem, 2007, 8, 1375-1385.	2.1	6
26	The Landscape of Pervasive Horizontal Pleiotropy in Human Genetic Variation is Driven by Extreme Polygenicity of Human Traits and Diseases. SSRN Electronic Journal, 0, , .	0.4	3