Dror Sharon

List of Publications by Year
in descending order

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83 papers	4,731 citations	94433 37 h-index	110387 64 g-index
83 all docs	83 docs citations	83 times ranked	4556 citing authors

#	Article	IF	Citations
1	Retinitis pigmentosa and allied diseases: numerous diseases, genes, and inheritance patterns. Human Molecular Genetics, 2002, 11, 1219-1227.	2.9	251
2	The olfactory receptor gene superfamily: data mining, classification, and nomenclature. Mammalian Genome, 2000, 11, 1016-1023.	2.2	196
3	Worldwide carrier frequency and genetic prevalence of autosomal recessive inherited retinal diseases. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 2710-2716.	7.1	195
4	RP2 and RPGR Mutations and Clinical Correlations in Patients with X-Linked Retinitis Pigmentosa. American Journal of Human Genetics, 2003, 73, 1131-1146.	6.2	193
5	Shared Mutations in NR2E3 in Enhanced S-cone Syndrome, Goldmann-Favre Syndrome, and Many Cases of Clumped Pigmentary Retinal Degeneration. JAMA Ophthalmology, 2003, 121, 1316.	2.4	160
6	Profile of the genes expressed in the human peripheral retina, macula, and retinal pigment epithelium determined through serial analysis of gene expression (SAGE). Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 315-320.	7.1	155
7	A Nonsense Mutation in PDE6H Causes Autosomal-Recessive Incomplete Achromatopsia. American Journal of Human Genetics, 2012, 91, 527-532.	6.2	148
8	Whole-Exome Sequencing Identifies Mutations in GPR179 Leading to Autosomal-Recessive Complete Congenital Stationary Night Blindness. American Journal of Human Genetics, 2012, 90, 321-330.	6.2	121
9	A Missense Mutation in DHDDS, Encoding Dehydrodolichyl Diphosphate Synthase, Is Associated with Autosomal-Recessive Retinitis Pigmentosa in Ashkenazi Jews. American Journal of Human Genetics, 2011, 88, 207-215.	6.2	120
10	Primate Evolution of an Olfactory Receptor Cluster: Diversification by Gene Conversion and Recent Emergence of Pseudogenes. Genomics, 1999, 61, 24-36.	2.9	119
11	Whole genome sequencing in patients with retinitis pigmentosa reveals pathogenic DNA structural changes and NEK2 as a new disease gene. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, 16139-16144.	7.1	115
12	BBS1 Mutations in a Wide Spectrum of Phenotypes Ranging From Nonsyndromic Retinitis Pigmentosa to Bardet-Biedl Syndrome. JAMA Ophthalmology, 2012, 130, 1425.	2.4	106
13	Mutations in IMPG2, Encoding Interphotoreceptor Matrix Proteoglycan 2, Cause Autosomal-Recessive Retinitis Pigmentosa. American Journal of Human Genetics, 2010, 87, 199-208.	6.2	98
14	Homozygosity Mapping Reveals Null Mutations in FAM161A as a Cause of Autosomal-Recessive Retinitis Pigmentosa. American Journal of Human Genetics, 2010, 87, 382-391.	6.2	98
15	An integrated genetic linkage map of avocado. Theoretical and Applied Genetics, 1997, 95, 911-921.	3.6	95
16	Exome Sequencing and cis-Regulatory Mapping Identify Mutations in MAK, a Gene Encoding a Regulator of Ciliary Length, as a Cause of Retinitis Pigmentosa. American Journal of Human Genetics, 2011, 89, 253-264.	6.2	95
17	Gene Augmentation Therapy Restores Retinal Function and Visual Behavior in a Sheep Model of CNGA3 Achromatopsia. Molecular Therapy, 2015, 23, 1423-1433.	8.2	93
18	Dichotomy of single-nucleotide polymorphism haplotypes in olfactory receptor genes and pseudogenes. Nature Genetics, 2000, 26, 221-224.	21.4	92

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19	The Effect of Cone Opsin Mutations on Retinal Structure and the Integrity of the Photoreceptor Mosaic. , 2012, 53, 8006.		85
20	Mutations in RAB28, Encoding a Farnesylated Small GTPase, Are Associated with Autosomal-Recessive Cone-Rod Dystrophy. American Journal of Human Genetics, 2013, 93, 110-117.	6.2	85
21	Mutations in C8orf37, Encoding a Ciliary Protein, are Associated with Autosomal-Recessive Retinal Dystrophies with Early Macular Involvement. American Journal of Human Genetics, 2012, 90, 102-109.	6.2	82
22	A homozygous nonsense CEP250 mutation combined with a heterozygous nonsense C2orf71 mutation is associated with atypical Usher syndrome. Journal of Medical Genetics, 2014, 51, 460-469.	3.2	78
23	Mutations in PNPLA6 are linked to photoreceptor degeneration and various forms of childhood blindness. Nature Communications, 2015, 6, 5614.	12.8	77
24	Loss of the Metalloprotease ADAM9 Leads to Cone-Rod Dystrophy in Humans and Retinal Degeneration in Mice. American Journal of Human Genetics, 2009, 84, 683-691.	6.2	76
25	A nationwide genetic analysis of inherited retinal diseases in Israel as assessed by the Israeli inherited retinal disease consortium (IIRDC). Human Mutation, 2020, 41, 140-149.	2.5	75
26	Mutations in ARL2BP, Encoding ADP-Ribosylation-Factor-Like 2 Binding Protein, Cause Autosomal-Recessive Retinitis Pigmentosa. American Journal of Human Genetics, 2013, 93, 321-329.	6.2	67
27	Whole Exome Sequencing Reveals Mutations in Known Retinal Disease Genes in 33 out of 68 Israeli Families with Inherited Retinopathies. Scientific Reports, 2015, 5, 13187.	3.3	66
28	Application of DNA fingerprints for identification and genetic analysis of Carica papaya and other Carica species. Euphytica, 1992, 62, 119-126.	1.2	65
29	Molecular Anthropology Meets Genetic Medicine to Treat Blindness in the North African Jewish Population: Human Gene Therapy Initiated in Israel. Human Gene Therapy, 2010, 21, 1749-1757.	2.7	65
30	A Common Founder Mutation ofCERKLUnderlies Autosomal Recessive Retinal Degeneration with Early Macular Involvement among Yemenite Jews. , 2007, 48, 5431.		61
31	Genetics and Disease Expression in the CNGA3 Form of Achromatopsia. Ophthalmology, 2015, 122, 997-1007.	5.2	61
32	Ophthalmic Molecular Genetics. JAMA Ophthalmology, 2004, 122, 70.	2.4	59
33	Inherited retinal diseases: Linking genes, disease-causing variants, and relevant therapeutic modalities. Progress in Retinal and Eye Research, 2022, 89, 101029.	15.5	58
34	Novel Null Mutations in theEYSGene Are a Frequent Cause of Autosomal Recessive Retinitis Pigmentosa in the Israeli Population. , 2010, 51, 4387.		57
35	Frequency, Genotype, and Clinical Spectrum of Best Vitelliform Macular Dystrophy: Data From a National Center in Denmark. American Journal of Ophthalmology, 2012, 154, 403-412.e4.	3.3	56
36	Computer Vision â€“ ECCV 2016. Lecture Notes in Computer Science, 2016, , .	1.3	54

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37	FAM161A, associated with retinitis pigmentosa, is a component of the cilia-basal body complex and interacts with proteins involved in ciliopathies. Human Molecular Genetics, 2012, 21, 5174-5184.	2.9	51
38	Genetic relationships within avocado (Persea americana Mill) cultivars and between Persea species. Theoretical and Applied Genetics, 1997, 94, 279-286.	3.6	47
39	Non-syndromic retinitis pigmentosa due to mutations in the mucopolysaccharidosis type IIIC gene, heparan-alpha-glucosaminide N-acetyltransferase (HGSNAT). Human Molecular Genetics, 2015, 24, 3742-51.	2.9	47
40	Identification of Mutations Causing Inherited Retinal Degenerations in the Israeli and Palestinian Populations Using Homozygosity Mapping. , 2014, 55, 1149.		46
41	A Homozygous Frameshift Mutation inBEST1Causes the Classical Form of Best Disease in an Autosomal Recessive Mode. , 2011, 52, 5332.		44
42	Association Between Missense Mutations in theBBS2Gene and Nonsyndromic Retinitis Pigmentosa. JAMA Ophthalmology, 2015, 133, 312.	2.5	43
43	Variable Retinal Phenotypes Caused by Mutations in the X-Linked Photopigment Gene Array. , 2010, 51, 3884.		42
44	Homozygosity for a NovelABCA4Founder Splicing Mutation Is Associated with Progressive and Severe Stargardt-like Disease. , 2007, 48, 4308.		37
45	Evaluation of Macular Structure and Function by OCT and Electrophysiology in Patients with Vitelliform Macular Dystrophy Due to Mutations in BEST1. Investigative Ophthalmology and Visual Science, 2010, 51, 4754-4765.	3.3	37
46	Whole-Exome Sequencing Identifies Biallelic IDH3A Variants as a Cause of Retinitis Pigmentosa Accompanied by Pseudocoloboma. Ophthalmology, 2017, 124, 992-1003.	5.2	37
47	A non-ancestralRPGR missense mutation in families with either recessive or semi-dominant X-linked retinitis pigmentosa. American Journal of Medical Genetics, Part A, 2007, 143A, 1150-1158.	1.2	36
48	The Spectrum of Retinal Diseases Caused by NR2E3 Mutations in Israeli and Palestinian Patients. JAMA Ophthalmology, 2009, 127, 297.	2.4	36
49	Novel USH2A Mutations in Israeli Patients With Retinitis Pigmentosa and Usher Syndrome Type 2. JAMA Ophthalmology, 2007, 125, 219.	2.4	35
50	Mouseâ€“Human Orthology Relationships in an Olfactory Receptor Gene Cluster. Genomics, 2001, 71, 296-306.	2.9	33
51	Whole Exome Sequencing Reveals GUCY2D as a Major Gene Associated With Cone and Cone-Rod Dystrophy in Israel. Investigative Ophthalmology and Visual Science, 2015, 56, 420-430.	3.3	32
52	Identification and characterization of coding single-nucleotide polymorphisms within a human olfactory receptor gene cluster. Gene, 2000, 260, 87-94.	2.2	30
53	Nonsyndromic Retinitis Pigmentosa in the Ashkenazi Jewish Population. Ophthalmology, 2018, 125, 725-734.	5.2	30
54	Four USH2A Founder Mutations Underlie the Majority of Usher Syndrome Type 2 Cases among Non-Ashkenazi Jews. Genetic Testing and Molecular Biomarkers, 2008, 12, 289-294.	1.7	28

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55	Exome Sequencing Identifies a Founder Frameshift Mutation in an Alternative Exon of USH1C as the Cause of Autosomal Recessive Retinitis Pigmentosa with Late-Onset Hearing Loss. PLoS ONE, 2012, 7, e51566.	2.5	27
56	Mutations inCRB1are a Relatively Common Cause of Autosomal Recessive Early-Onset Retinal Degeneration in the Israeli and Palestinian Populations. , 2013, 54, 2068.		25
57	Cone Dystrophy with Supernormal Rod Response. Ophthalmology, 2013, 120, 2338-2343.	5.2	23
58	Allele frequency analysis of variants reported to cause autosomal dominant inherited retinal diseases question the involvement of 19% of genes and 10% of reported pathogenic variants. Journal of Medical Genetics, 2019, 56, 536-542.	3.2	21
59	Association of Pattern Dystrophy With an HTRA1 Single-Nucleotide Polymorphism. JAMA Ophthalmology, 2012, 130, 987-91.	2.4	19
60	Interactome analysis reveals that FAM161A, deficient in recessive retinitis pigmentosa, is a component of the Golgi-centrosomal network. Human Molecular Genetics, 2015, 24, 3359-3371.	2.9	19
61	Ocular Phenotype Analysis of a Family With Biallelic Mutations in the BEST1 Gene. American Journal of Ophthalmology, 2014, 157, 697-709.e2.	3.3	17
62	Nonsyndromic Early-Onset Cone-Rod Dystrophy and Limb-Girdle Muscular Dystrophy in a Consanguineous Israeli Family are Caused by Two Independent yet Linked Mutations inALMS1andDYSF. Human Mutation, 2015, 36, 836-841.	2.5	17
63	A new mouse model for retinal degeneration due to Fam161a deficiency. Scientific Reports, 2021, 11, 2030.	3.3	17
64	Genome Dynamics, Evolution, and Protein Modeling in the Olfactory Receptor Gene Superfamilya. Annals of the New York Academy of Sciences, 1998, 855, 182-193.	3.8	16
65	Genetic association between DNA fingerprint fragments and loci controlling agriculturally important traits in avocado (Persea americana Mill.). Euphytica, 1995, 84, 81-87.	1.2	15
66	A Complex Expression Pattern ofPax6in the Pigeon Retina. , 2007, 48, 2503.		15
67	Carrier frequency analysis of mutations causing autosomal-recessive-inherited retinal diseases in the Israeli population. European Journal of Human Genetics, 2018, 26, 1159-1166.	2.8	14
68	TRPM1 Mutations are the Most Common Cause of Autosomal Recessive Congenital Stationary Night Blindness (CSNB) in the Palestinian and Israeli Populations. Scientific Reports, 2019, 9, 12047.	3.3	14
69	Unique combination of clinical features in a large cohort of 100 patients with retinitis pigmentosa caused by FAM161A mutations. Scientific Reports, 2020, 10, 15156.	3.3	14
70	Level of Heterozygosity and Mode of Inheritance of Variable Number of Tandem Repeat Loci in Avocado. Journal of the American Society for Horticultural Science, 1996, 121, 768-772.	1.0	13
71	An ancient autosomal haplotype bearing a rare achromatopsia-causing founder mutation is shared among Arab Muslims and Oriental Jews. Human Genetics, 2010, 128, 261-267.	3.8	12
72	KCNV2-Associated Retinopathy: Detailed Retinal Phenotype and Structural Endpointsâ€"KCNV2 Study Group Report 2. American Journal of Ophthalmology, 2021, 230, 1-11.	3.3	11

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73	Reaction of nitrobenzene and azobenzene with transition metal compounds in low oxidation state. Tetrahedron, 1981, 37, 939-942.	1.9	10
74	A Nonsense Mutation inFAM161AIs a Recurrent Founder Allele in Dutch and Belgian Individuals With Autosomal Recessive Retinitis Pigmentosa. , 2015, 56, 7418.		9
75	Combined Occurrence of Autosomal Dominant Aniridia and Autosomal Recessive Albinism in Several Members of a Family. Ophthalmic Genetics, 2015, 36, 175-179.	1.2	7
76	Effect of heat treatment on bend stress relaxation of pure tungsten. Fusion Engineering and Design, 2013, 88, 1735-1738.	1.9	5
77	Enhancer of Zeste Homolog 2 (EZH2) Contributes to Rod Photoreceptor Death Process in Several Forms of Retinal Degeneration and Its Activity Can Serve as a Biomarker for Therapy Efficacy. International Journal of Molecular Sciences, 2021, 22, 9331.	4.1	5
78	Translational Read-Through Drugs (TRIDs) Are Able to Restore Protein Expression and Ciliogenesis in Fibroblasts of Patients with Retinitis Pigmentosa Caused by a Premature Termination Codon in FAM161A. International Journal of Molecular Sciences, 2022, 23, 3541.	4.1	5
79	Identification of autosomal recessive novel genes and retinal phenotypes in members of the solute carrier (SLC) superfamily. Genetics in Medicine, 2022, 24, 1523-1535.	2.4	5
80	Microarray-based gene expression analysis during retinal maturation of albino rats. Graefe's Archive for Clinical and Experimental Ophthalmology, 2008, 246, 693-702.	1.9	4
81	Retinitis pigmentosa and allied diseases: numerous diseases, genes, and inheritance patterns. Human Molecular Genetics, 2003, 12, 583-584.	2.9	2
82	Lack of association between the C2 allele of transferrin and age-related macular degeneration in the Israeli population. Ophthalmic Genetics, 2009, 30, 161-164.	1.2	2
83	Whole-Exome Sequencing Identifies Mutations in GPR179 Leading to Autosomal-Recessive Complete Congenital Stationary Night Blindness. American Journal of Human Genetics, 2012, 91, 209.	6.2	0