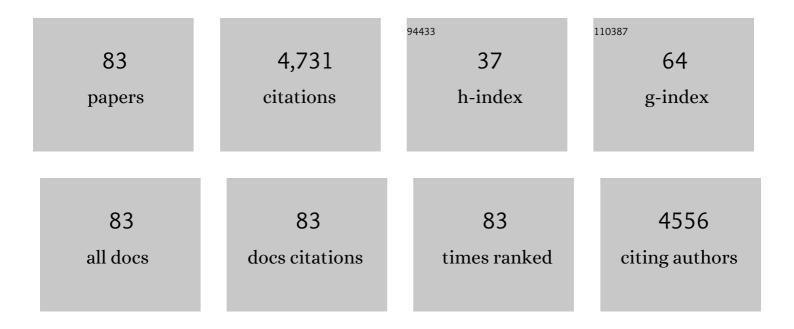
Dror Sharon

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Inherited retinal diseases: Linking genes, disease-causing variants, and relevant therapeutic modalities. Progress in Retinal and Eye Research, 2022, 89, 101029.	15.5	58
2	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
3	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
4	A new mouse model for retinal degeneration due to Fam161a deficiency. Scientific Reports, 2021, 11, 2030.	3.3	17
5	KCNV2-Associated Retinopathy: Detailed Retinal Phenotype and Structural Endpoints—KCNV2 Study Group Report 2. American Journal of Ophthalmology, 2021, 230, 1-11.	3.3	11
6	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
7	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
8	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
9	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
10	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
11	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
12	Nonsyndromic Retinitis Pigmentosa in the Ashkenazi Jewish Population. Ophthalmology, 2018, 125, 725-734.	5.2	30
13	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
14	Whole-Exome Sequencing Identifies Biallelic IDH3A Variants as a Cause of Retinitis Pigmentosa Accompanied by Pseudocoloboma. Ophthalmology, 2017, 124, 992-1003.	5.2	37
15	Computer Vision $\hat{a} \in CCV 2016$. Lecture Notes in Computer Science, 2016, , .	1.3	54
16	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
17	Nonsyndromic Early-Onset Cone-Rod Dystrophy and Limb-Girdle Muscular Dystrophy in a Consanguineous Israeli Family are Caused by Two Independent yet Linked Mutations in <i>ALMS1</i> and <i>DYSF</i> . Human Mutation, 2015, 36, 836-841.	2.5	17
18	A Nonsense Mutation in <i>FAM161A</i> Is a Recurrent Founder Allele in Dutch and Belgian Individuals		9

With Autosomal Recessive Retinitis Pigmentosa. , 2015, 56, 7418.

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19	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
20	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
21	Association Between Missense Mutations in the <i>BBS2</i> Gene and Nonsyndromic Retinitis Pigmentosa. JAMA Ophthalmology, 2015, 133, 312.	2.5	43
22	Genetics and Disease Expression in the CNGA3 Form of Achromatopsia. Ophthalmology, 2015, 122, 997-1007.	5.2	61
23	Mutations in PNPLA6 are linked to photoreceptor degeneration and various forms of childhood blindness. Nature Communications, 2015, 6, 5614.	12.8	77
24	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
25	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
26	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
27	Identification of Mutations Causing Inherited Retinal Degenerations in the Israeli and Palestinian Populations Using Homozygosity Mapping. , 2014, 55, 1149.		46
28	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
29	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
30	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
31	Effect of heat treatment on bend stress relaxation of pure tungsten. Fusion Engineering and Design, 2013, 88, 1735-1738.	1.9	5
32	Cone Dystrophy with Supernormal Rod Response. Ophthalmology, 2013, 120, 2338-2343.	5.2	23
33	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
34	Mutations in <i>CRB1</i> are a Relatively Common Cause of Autosomal Recessive Early-Onset Retinal Degeneration in the Israeli and Palestinian Populations. , 2013, 54, 2068.		25
35	Whole genome sequencing in patients with retinitis pigmentosa reveals pathogenic DNA structural changes and <i>NEK2</i> 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
36	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

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37	The Effect of Cone Opsin Mutations on Retinal Structure and the Integrity of the Photoreceptor Mosaic. , 2012, 53, 8006.		85
38	Association of Pattern Dystrophy With an HTRA1 Single-Nucleotide Polymorphism. JAMA Ophthalmology, 2012, 130, 987-91.	2.4	19
39	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
40	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
41	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
42	A Nonsense Mutation in PDE6H Causes Autosomal-Recessive Incomplete Achromatopsia. American Journal of Human Genetics, 2012, 91, 527-532.	6.2	148
43	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
44	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
45	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
46	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
47	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
48	A Homozygous Frameshift Mutation in <i>BEST1</i> Causes the Classical Form of Best Disease in an Autosomal Recessive Mode. , 2011, 52, 5332.		44
49	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
50	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
51	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
52	Novel Null Mutations in the <i>EYS</i> Gene Are a Frequent Cause of Autosomal Recessive Retinitis Pigmentosa in the Israeli Population. , 2010, 51, 4387.		57
53	Variable Retinal Phenotypes Caused by Mutations in the X-Linked Photopigment Gene Array. , 2010, 51, 3884.		42
54	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

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55	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
56	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
57	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
58	The Spectrum of Retinal Diseases Caused by NR2E3 Mutations in Israeli and Palestinian Patients. JAMA Ophthalmology, 2009, 127, 297.	2.4	36
59	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
60	Four <i>USH2A</i> 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
61	A Common Founder Mutation of <i>CERKL</i> Underlies Autosomal Recessive Retinal Degeneration with Early Macular Involvement among Yemenite Jews. , 2007, 48, 5431.		61
62	Novel USH2A Mutations in Israeli Patients With Retinitis Pigmentosa and Usher Syndrome Type 2. JAMA Ophthalmology, 2007, 125, 219.	2.4	35
63	A Complex Expression Pattern ofPax6in the Pigeon Retina. , 2007, 48, 2503.		15
64	Homozygosity for a Novel <i>ABCA4</i> Founder Splicing Mutation Is Associated with Progressive and Severe Stargardt-like Disease. , 2007, 48, 4308.		37
65	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
66	Ophthalmic Molecular Genetics. JAMA Ophthalmology, 2004, 122, 70.	2.4	59
67	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
68	Retinitis pigmentosa and allied diseases: numerous diseases, genes, and inheritance patterns. Human Molecular Genetics, 2003, 12, 583-584.	2.9	2
69	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
70	Retinitis pigmentosa and allied diseases: numerous diseases, genes, and inheritance patterns. Human Molecular Genetics, 2002, 11, 1219-1227.	2.9	251
71	Profile of the genes expressed in the human peripheral retina, macula, and retinal pigment epithelium determined through serial analysis of gene expression (SACE). Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 315-320.	7.1	155
72	Mouse–Human Orthology Relationships in an Olfactory Receptor Gene Cluster. Genomics, 2001, 71, 296-306.	2.9	33

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73	Dichotomy of single-nucleotide polymorphism haplotypes in olfactory receptor genes and pseudogenes. Nature Genetics, 2000, 26, 221-224.	21.4	92
74	The olfactory receptor gene superfamily: data mining, classification, and nomenclature. Mammalian Genome, 2000, 11, 1016-1023.	2.2	196
75	Identification and characterization of coding single-nucleotide polymorphisms within a human olfactory receptor gene cluster. Gene, 2000, 260, 87-94.	2.2	30
76	Primate Evolution of an Olfactory Receptor Cluster: Diversification by Gene Conversion and Recent Emergence of Pseudogenes. Genomics, 1999, 61, 24-36.	2.9	119
77	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
78	Genetic relationships within avocado (Persea americana Mill) cultivars and between Persea species. Theoretical and Applied Genetics, 1997, 94, 279-286.	3.6	47
79	An integrated genetic linkage map of avocado. Theoretical and Applied Genetics, 1997, 95, 911-921.	3.6	95
80	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
81	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
82	Application of DNA fingerprints for identification and genetic analysis of Carica papaya and other Carica species. Euphytica, 1992, 62, 119-126.	1.2	65
83	Reaction of nitrobenzene and azobenzene with transition metal compounds in low oxidation state. Tetrahedron, 1981, 37, 939-942.	1.9	10