

# Sanjay I Bidichandani

## List of Publications by Year in descending order

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45  
papers

4,567  
citations

236925

25  
h-index

276875

41  
g-index

46  
all docs

46  
docs citations

46  
times ranked

3675  
citing authors

#	ARTICLE	IF	CITATIONS
1	Friedreich's Ataxia: Autosomal Recessive Disease Caused by an Intronic GAA Triplet Repeat Expansion. <i>Science</i> , 1996, 271, 1423-1427.	12.6	2,642
2	The GAA Triplet-Repeat Expansion in Friedreich Ataxia Interferes with Transcription and May Be Associated with an Unusual DNA Structure. <i>American Journal of Human Genetics</i> , 1998, 62, 111-121.	6.2	358
3	Progressive gaa expansions in dorsal root ganglia of Friedreich's ataxia patients. <i>Annals of Neurology</i> , 2007, 61, 55-60.	5.3	106
4	Epigenetic Silencing in Friedreich Ataxia Is Associated with Depletion of CTCF (CCCTC-Binding Factor) and Antisense Transcription. <i>PLoS ONE</i> , 2009, 4, e7914.	2.5	102
5	Friedreich ataxia- pathogenesis and implications for therapies. <i>Neurobiology of Disease</i> , 2019, 132, 104606.	4.4	84
6	Length-dependent structure formation in Friedreich ataxia (GAA) <sub>n</sub> (TTC) <sub>n</sub> repeats at neutral pH. <i>Nucleic Acids Research</i> , 2004, 32, 1224-1231.	14.5	81
7	Frataxin fracas. <i>Nature Genetics</i> , 1997, 15, 337-338.	21.4	78
8	Somatic instability of the expanded GAA triplet-repeat sequence in Friedreich ataxia progresses throughout life. <i>Genomics</i> , 2007, 90, 1-5.	2.9	74
9	Analysis of Unstable Triplet Repeats Using Small-Pool Polymerase Chain Reaction. , 2004, 277, 061-076.		65
10	The GAA triplet-repeat sequence in Friedreich ataxia shows a high level of somatic instability in vivo, with a significant predilection for large contractions. <i>Human Molecular Genetics</i> , 2002, 11, 2175-2187.	2.9	64
11	The GAA triplet-repeat is unstable in the context of the human FXN locus and displays age-dependent expansions in cerebellum and DRG in a transgenic mouse model. <i>Human Genetics</i> , 2006, 120, 633-640.	3.8	59
12	Somatic Sequence Variation at the Friedreich Ataxia Locus Includes Complete Contraction of the Expanded GAA Triplet Repeat, Significant Length Variation in Serially Passaged Lymphoblasts and Enhanced Mutagenesis in the Flanking Sequence. <i>Human Molecular Genetics</i> , 1999, 8, 2425-2436.	2.9	58
13	Replication-mediated instability of the GAA triplet repeat mutation in Friedreich ataxia. <i>Nucleic Acids Research</i> , 2004, 32, 5962-5971.	14.5	57
14	Glucocorticosteroids stimulate the activity of the promoters of peripheral myelin protein-22 and protein zero genes in Schwann cells. <i>Brain Research</i> , 2000, 865, 12-16.	2.2	54
15	Very Late-Onset Friedreich Ataxia Despite Large GAA Triplet Repeat Expansions. <i>Archives of Neurology</i> , 2000, 57, 246.	4.5	50
16	Friedreich ataxia in carriers of unstable borderline GAA triplet-repeat alleles. <i>Annals of Neurology</i> , 2004, 56, 898-901.	5.3	46
17	Acidic Proline-rich Protein Db and Caries in Young Children. <i>Journal of Dental Research</i> , 2007, 86, 1176-1180.	5.2	45
18	Expansion of GAA triplet repeats in the human genome: unique origin of the FRDA mutation at the center of an Alu. <i>Genomics</i> , 2004, 83, 373-383.	2.9	44

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19	Pms2 Suppresses Large Expansions of the (GAA <sup>n</sup> TTC) <sub>n</sub> Sequence in Neuronal Tissues. PLoS ONE, 2012, 7, e47085.	2.5	43
20	Expansion of GAA trinucleotide repeats in mammals. Genomics, 2006, 87, 57-67.	2.9	42
21	Epigenetic promoter silencing in Friedreich ataxia is dependent on repeat length. Annals of Neurology, 2014, 76, 522-528.	5.3	36
22	Altered Nucleosome Positioning at the Transcription Start Site and Deficient Transcriptional Initiation in Friedreich Ataxia. Journal of Biological Chemistry, 2014, 289, 15194-15202.	3.4	36
23	Distinct distribution of autosomal dominant spinocerebellar ataxia in the Mexican population. Movement Disorders, 2007, 22, 1050-1053.	3.9	35
24	Uptake of genetic testing and long-term tumor surveillance in von Hippel-Lindau disease. BMC Medical Genetics, 2010, 11, 4.	2.1	35
25	Methylated and unmethylated alleles support variegated epigenetic silencing in Friedreich ataxia. Human Molecular Genetics, 2021, 29, 3818-3829.	2.9	29
26	Replication in mammalian cells recapitulates the locus-specific differences in somatic instability of genomic GAA triplet-repeats. Nucleic Acids Research, 2006, 34, 6352-6361.	14.5	28
27	Identification of a positive regulatory element in the myelin-specific promoter of the PMP22 gene. Journal of Neuroscience Research, 2001, 65, 508-519.	2.9	26
28	Reversal of epigenetic promoter silencing in Friedreich ataxia by a class I histone deacetylase inhibitor. Nucleic Acids Research, 2016, 44, 5095-5104.	14.5	26
29	Anticipation and intergenerational repeat instability in spinocerebellar ataxia type 17. Annals of Neurology, 2007, 61, 607-610.	5.3	19
30	A mild case of Friedreich ataxia: Lymphocyte and sural nerve analysis for GAA repeat length reveals somatic mosaicism. , 1998, 21, 390-393.		17
31	Repair of DNA double-strand breaks within the (GAA <sup>n</sup> TTC) <sub>n</sub> sequence results in frequent deletion of the triplet-repeat sequence. Nucleic Acids Research, 2007, 36, 489-500.	14.5	17
32	Competitive Binding of Triplex-Forming Oligonucleotides in the Two Alternate Promoters of the PMP22 Gene. Oligonucleotides, 2001, 11, 233-246.	4.3	16
33	FXN Promoter Silencing in the Humanized Mouse Model of Friedreich Ataxia. PLoS ONE, 2015, 10, e0138437.	2.5	15
34	Genetic admixture of European FRDA genes is the cause of Friedreich ataxia in the Mexican population. Genomics, 2004, 84, 779-784.	2.9	14
35	Role of transcript and interplay between transcription and replication in triplet-repeat instability in mammalian cells. Nucleic Acids Research, 2011, 39, 526-535.	14.5	11
36	Clinical heterogeneity of recessive ataxia in the Mexican population. Journal of Neurology, Neurosurgery and Psychiatry, 2006, 77, 1370-1372.	1.9	10

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37	Reply to Callen. American Journal of Human Genetics, 1998, 63, 270.	6.2	9
38	Deficiency of RecA-dependent RecFOR and RecBCD pathways causes increased instability of the (GAA{TTC}) <sub>n</sub> sequence when GAA is the lagging strand template. Nucleic Acids Research, 2007, 35, 6884-6894.	14.5	8
39	DNA methylation in Friedreich ataxia silences expression of frataxin isoform E. Scientific Reports, 2022, 12, 5031.	3.3	7
40	Epigenetic Heterogeneity in Friedreich Ataxia Underlies Variable FXN Reactivation. Frontiers in Neuroscience, 2021, 15, 752921.	2.8	6
41	E. coli mismatch repair acts downstream of replication fork stalling to stabilize the expanded (GAA{TTC}) <sub>n</sub> sequence. Mutation Research - Fundamental and Molecular Mechanisms of Mutagenesis, 2009, 661, 71-77.	1.0	5
42	Funding agencies and disease organizations: Resources and recommendations to facilitate ALS clinical research. Amyotrophic Lateral Sclerosis and Frontotemporal Degeneration, 2013, 14, 62-66.	1.7	5
43	Evolution and Instability of the GAA Triplet-Repeat Sequence in Friedreich's Ataxia. , 2006, , 305-319.		4
44	RNA-mediated transcriptional gene silencing in Friedreich ataxia. Current Opinion in Biotechnology, 2011, 22, S16.	6.6	1
45	SCA10. , 2010, , 80-82.		0