

# 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	DNA methylation in Friedreich ataxia silences expression of frataxin isoform E. <i>Scientific Reports</i> , 2022, 12, 5031.	3.3	7
2	Methylated and unmethylated epialleles support variegated epigenetic silencing in Friedreich ataxia. <i>Human Molecular Genetics</i> , 2021, 29, 3818-3829.	2.9	29
3	Epigenetic Heterogeneity in Friedreich Ataxia Underlies Variable FXN Reactivation. <i>Frontiers in Neuroscience</i> , 2021, 15, 752921.	2.8	6
4	Friedreich ataxia- pathogenesis and implications for therapies. <i>Neurobiology of Disease</i> , 2019, 132, 104606.	4.4	84
5	Reversal of epigenetic promoter silencing in Friedreich ataxia by a class I histone deacetylase inhibitor. <i>Nucleic Acids Research</i> , 2016, 44, 5095-5104.	14.5	26
6	FXN Promoter Silencing in the Humanized Mouse Model of Friedreich Ataxia. <i>PLoS ONE</i> , 2015, 10, e0138437.	2.5	15
7	Epigenetic promoter silencing in Friedreich ataxia is dependent on repeat length. <i>Annals of Neurology</i> , 2014, 76, 522-528.	5.3	36
8	Altered Nucleosome Positioning at the Transcription Start Site and Deficient Transcriptional Initiation in Friedreich Ataxia. <i>Journal of Biological Chemistry</i> , 2014, 289, 15194-15202.	3.4	36
9	Funding agencies and disease organizations: Resources and recommendations to facilitate ALS clinical research. <i>Amyotrophic Lateral Sclerosis and Frontotemporal Degeneration</i> , 2013, 14, 62-66.	1.7	5
10	Pms2 Suppresses Large Expansions of the (GAA·TTC) <sub>n</sub> Sequence in Neuronal Tissues. <i>PLoS ONE</i> , 2012, 7, e47085.	2.5	43
11	RNA-mediated transcriptional gene silencing in Friedreich ataxia. <i>Current Opinion in Biotechnology</i> , 2011, 22, S16.	6.6	1
12	Role of transcript and interplay between transcription and replication in triplet-repeat instability in mammalian cells. <i>Nucleic Acids Research</i> , 2011, 39, 526-535.	14.5	11
13	Uptake of genetic testing and long-term tumor surveillance in von Hippel-Lindau disease. <i>BMC Medical Genetics</i> , 2010, 11, 4.	2.1	35
14	SCA10. , 2010, , 80-82.		0
15	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
16	E. coli mismatch repair acts downstream of replication fork stalling to stabilize the expanded (GAA·TTC) <sub>n</sub> sequence. <i>Mutation Research - Fundamental and Molecular Mechanisms of Mutagenesis</i> , 2009, 661, 71-77.	1.0	5
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	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. <i>Nucleic Acids Research</i> , 2007, 35, 6884-6894.	14.5	8

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19	Repair of DNA double-strand breaks within the (GAA*TTC) <sub>n</sub> sequence results in frequent deletion of the triplet-repeat sequence. <i>Nucleic Acids Research</i> , 2007, 36, 489-500.	14.5	17
20	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
21	Progressive gaa expansions in dorsal root ganglia of Friedreich's ataxia patients. <i>Annals of Neurology</i> , 2007, 61, 55-60.	5.3	106
22	Anticipation and intergenerational repeat instability in spinocerebellar ataxia type 17. <i>Annals of Neurology</i> , 2007, 61, 607-610.	5.3	19
23	Distinct distribution of autosomal dominant spinocerebellar ataxia in the Mexican population. <i>Movement Disorders</i> , 2007, 22, 1050-1053.	3.9	35
24	Expansion of GAA trinucleotide repeats in mammals. <i>Genomics</i> , 2006, 87, 57-67.	2.9	42
25	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
26	Replication in mammalian cells recapitulates the locus-specific differences in somatic instability of genomic GAA triplet-repeats. <i>Nucleic Acids Research</i> , 2006, 34, 6352-6361.	14.5	28
27	Clinical heterogeneity of recessive ataxia in the Mexican population. <i>Journal of Neurology, Neurosurgery and Psychiatry</i> , 2006, 77, 1370-1372.	1.9	10
28	Evolution and Instability of the GAA Triplet-Repeat Sequence in Friedreich's Ataxia. , 2006, , 305-319.		4
29	Replication-mediated instability of the GAA triplet repeat mutation in Friedreich ataxia. <i>Nucleic Acids Research</i> , 2004, 32, 5962-5971.	14.5	57
30	Length-dependent structure formation in Friedreich ataxia (GAA) <sub>n</sub> {middle dot}(TTC) <sub>n</sub> repeats at neutral pH. <i>Nucleic Acids Research</i> , 2004, 32, 1224-1231.	14.5	81
31	Friedreich ataxia in carriers of unstable borderline GAA triplet-repeat alleles. <i>Annals of Neurology</i> , 2004, 56, 898-901.	5.3	46
32	Analysis of Unstable Triplet Repeats Using Small-Pool Polymerase Chain Reaction. , 2004, 277, 061-076.		65
33	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
34	Genetic admixture of European FRDA genes is the cause of Friedreich ataxia in the Mexican population. <i>Genomics</i> , 2004, 84, 779-784.	2.9	14
35	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
36	Identification of a positive regulatory element in the myelin-specific promoter of the PMP22 gene. <i>Journal of Neuroscience Research</i> , 2001, 65, 508-519.	2.9	26

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37	Competitive Binding of Triplex-Forming Oligonucleotides in the Two Alternate Promoters of the PMP22 Gene. <i>Oligonucleotides</i> , 2001, 11, 233-246.	4.3	16
38	Very Late-Onset Friedreich Ataxia Despite Large GAA Triplet Repeat Expansions. <i>Archives of Neurology</i> , 2000, 57, 246.	4.5	50
39	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
40	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
41	A mild case of Friedreich ataxia: Lymphocyte and sural nerve analysis for GAA repeat length reveals somatic mosaicism. , 1998, 21, 390-393.		17
42	Reply to Callen. <i>American Journal of Human Genetics</i> , 1998, 63, 270.	6.2	9
43	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
44	Frataxin fracas. <i>Nature Genetics</i> , 1997, 15, 337-338.	21.4	78
45	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