Steve D Wilton

List of Publications by Year
in descending order

113 papers	5,847 citations	136740 32 h-index	79541 73 g-index
118 all docs	118 docs citations	118 times ranked	6800 citing authors

#	Article	IF	Citations
1	Stargardt disease and progress in therapeutic strategies. Ophthalmic Genetics, 2022, 43, 1-26.	0.5	18
2	Investigating the Implications of CFTR Exon Skipping Using a Cftr Exon 9 Deleted Mouse Model. Frontiers in Pharmacology, 2022, 13, 868863.	1.6	1
3	Antisense Oligonucleotide Induction of the hnRNPA1b Isoform Affects Pre-mRNA Splicing of SMN2 in SMA Type I Fibroblasts. International Journal of Molecular Sciences, 2022, 23, 3937.	1.8	0
4	Single Stranded Fully Modified-Phosphorothioate Oligonucleotides can Induce Structured Nuclear Inclusions, Alter Nuclear Protein Localization and Disturb the Transcriptome In Vitro. Frontiers in Genetics, 2022, 13, 791416.	1.1	10
5	NEAT1 polyA-modulating antisense oligonucleotides reveal opposing functions for both long non-coding RNA isoforms in neuroblastoma. Cellular and Molecular Life Sciences, 2021, 78, 2213-2230.	2.4	39
6	Generation of three induced pluripotent stem cell lines from a patient with Usher syndrome caused by biallelic c.949CÂ>ÂA and c.1256GÂ>ÂT mutations in the USH2A gene. Stem Cell Research, 2021, 50, 102129.	0.3	3
7	Proof-of-Concept: Antisense Oligonucleotide Mediated Skipping of Fibrillin-1 Exon 52. International Journal of Molecular Sciences, 2021, 22, 3479.	1.8	6
8	Novel STMN2 Variant Linked to Amyotrophic Lateral Sclerosis Risk and Clinical Phenotype. Frontiers in Aging Neuroscience, 2021, 13, 658226.	1.7	38
9	Targeted SMN Exon Skipping: A Useful Control to Assess In Vitro and In Vivo Splice-Switching Studies. Biomedicines, 2021, 9, 552.	1.4	6
10	Induction of cryptic pre-mRNA splice-switching by antisense oligonucleotides. Scientific Reports, 2021, 11, 15137.	1.6	4
11	Generation of an induced pluripotent stem cell line from a patient with Stargardt disease caused by biallelic c.[5461â€“10T>C;5603A>T];[6077T>C] mutations in the ABCA4 gene. Stem Cell Research, 2021, 54, 102439.	0.3	3
12	Generation of two induced pluripotent stem cell lines from a patient with Stargardt disease caused by compound heterozygous mutations in the ABCA4 gene. Stem Cell Research, 2021, 54, 102448.	0.3	0
13	Antisense oligonucleotide-based drug development for Cystic Fibrosis patients carrying the 3849+10Âkb C-to-T splicing mutation. Journal of Cystic Fibrosis, 2021, 20, 865-875.	0.3	30
14	Exploring microperimetry and autofluorescence endpoints for monitoring disease progression in PRPF31-associated retinopathy. Ophthalmic Genetics, 2021, 42, 1-14.	0.5	8
15	Polyglutamine Ataxias: Our Current Molecular Understanding and What the Future Holds for Antisense Therapies. Biomedicines, 2021, 9, 1499.	1.4	5
16	Splice correction therapies for familial hypercholesterolemic patients with low-density lipoprotein receptor mutations. Current Opinion in Lipidology, 2021, Publish Ahead of Print, 355-362.	1.2	1
17	Analysis of Pathogenic Pseudoexons Reveals Novel Mechanisms Driving Cryptic Splicing. Frontiers in Genetics, 2021, 12, 806946.	1.1	14
18	A Splice Intervention Therapy for Autosomal Recessive Juvenile Parkinsonâ€™s Disease Arising from Parkin Mutations. International Journal of Molecular Sciences, 2020, 21, 7282.	1.8	8

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19	Nonsequential Splicing Events Alter Antisense-Mediated Exon Skipping Outcome in COL7A1. International Journal of Molecular Sciences, 2020, 21, 7705.	1.8	15
20	Phenotypeâ€“genotype correlations in a pseudodominant Stargardt disease pedigree due to a novel ABCA4 deletionâ€“insertion variant causing a splicing defect. Molecular Genetics & Genomic Medicine, 2020, 8, e1259.	0.6	12
21	Morpholino Oligomer-Induced Dystrophin Isoforms to Map the Functional Domains in the Dystrophin Protein. Molecular Therapy - Nucleic Acids, 2020, 22, 263-272.	2.3	9
22	Progress in the molecular pathogenesis and nucleic acid therapeutics for Parkinson's disease in the precision medicine era. Medicinal Research Reviews, 2020, 40, 2650-2681.	5.0	32
23	Single Exon Skipping Can Address a Multi-Exon Duplication in the Dystrophin Gene. International Journal of Molecular Sciences, 2020, 21, 4511.	1.8	3
24	Novel Mutations Found in Individuals with Adult-Onset Pompe Disease. Genes, 2020, 11, 135.	1.0	7
25	Structural Variants May Be a Source of Missing Heritability in sALS. Frontiers in Neuroscience, 2020, 14, 47.	1.4	43
26	Splice modulating antisense oligonucleotides restore some acid-alpha-glucosidase activity in cells derived from patients with late-onset Pompe disease. Scientific Reports, 2020, 10, 6702.	1.6	8
27	In Vitro Validation of Phosphorodiamidate Morpholino Oligomers. Molecules, 2019, 24, 2922.	1.7	16
28	Systematic Approach to Developing Splice Modulating Antisense Oligonucleotides. International Journal of Molecular Sciences, 2019, 20, 5030.	1.8	14
29	Breakpoint junction features of seven DMD deletion mutations. Human Genome Variation, 2019, 6, 39.	0.4	7
30	Reduction of integrin alpha 4 activity through splice modulating antisense oligonucleotides. Scientific Reports, 2019, 9, 12994.	1.6	14
31	Consequences of Making the Inactive Active Through Changes in Antisense Oligonucleotide Chemistries. Frontiers in Genetics, 2019, 10, 1249.	1.1	3
32	Removal of the Polyglutamine Repeat of Ataxin-3 by Redirecting pre-mRNA Processing. International Journal of Molecular Sciences, 2019, 20, 5434.	1.8	9
33	ALS Genetics, Mechanisms, and Therapeutics: Where Are We Now?. Frontiers in Neuroscience, 2019, 13, 1310.	1.4	487
34	<scp>YAP</scp>ping about and not forgetting <scp>TAZ</scp>. FEBS Letters, 2019, 593, 253-276.	1.3	31
35	Antisense Oligonucleotides Targeting Angiogenic Factors as Potential Cancer Therapeutics. Molecular Therapy - Nucleic Acids, 2019, 14, 142-157.	2.3	58
36	A recurrent COL6A1 pseudoexon insertion causes muscular dystrophy and is effectively targeted by splice-correction therapies. JCI Insight, 2019, 4, .	2.3	33

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37	Antisense-mediated splice intervention to treat human disease: the odyssey continues. F1000Research, 2019, 8, 710.	0.8	11
38	Challenges of Interpreting Dystrophin Content by Western Blot. US Neurology, 2019, 15, 40.	0.2	7
39	Precision Medicine through Antisense Oligonucleotide-Mediated Exon Skipping. Trends in Pharmacological Sciences, 2018, 39, 982-994.	4.0	51
40	Skipping of Duplicated Dystrophin Exons: In Vitro Induction and Assessment. Methods in Molecular Biology, 2018, 1828, 219-228.	0.4	1
41	The potential of antisense oligonucleotide therapies for inherited childhood lung diseases. Molecular and Cellular Pediatrics, 2018, 5, 3.	1.0	21
42	Antisense Oligonucleotide-Mediated Terminal Intron Retention of the SMN2 Transcript. Molecular Therapy - Nucleic Acids, 2018, 11, 91-102.	2.3	16
43	Response to â€œRailroading at the FDAâ€• Nature Biotechnology, 2017, 35, 207-209.	9.4	6
44	Efficient Skipping of Single Exon Duplications in DMD Patient-Derived Cell Lines Using an Antisense Oligonucleotide Approach. Journal of Neuromuscular Diseases, 2017, 4, 199-207.	1.1	27
45	Inherited Retinal Disease Therapies Targeting Precursor Messenger Ribonucleic Acid. Vision (Switzerland), 2017, 1, 22.	0.5	5
46	Functional improvement of dystrophic muscle by repression of utrophin: let-7c interaction. PLoS ONE, 2017, 12, e0182676.	1.1	22
47	Polyglutamine ataxias: From Clinical and Molecular Features to Current Therapeutic Strategies. Journal of Genetic Syndromes & Gene Therapy, 2017, 08, .	0.2	7
48	Part 2: Making the â€œunprovenâ€•â€œprovenâ€• Cytotherapy, 2016, 18, 120-123.	0.3	6
49	Correcting the NLRP3 inflammasome deficiency in macrophages from autoimmune NZB mice with exon skipping antisense oligonucleotides. Immunology and Cell Biology, 2016, 94, 520-524.	1.0	7
50	Antisense oligonucleotide development for the treatment of muscular dystrophies. Expert Opinion on Orphan Drugs, 2016, 4, 139-152.	0.5	18
51	Deletion of Dystrophin In-Frame Exon 5 Leads to a Severe Phenotype: Guidance for Exon Skipping Strategies. PLoS ONE, 2016, 11, e0145620.	1.1	17
52	Pseudoexon activation increases phenotype severity in a Becker muscular dystrophy patient. Molecular Genetics & Genomic Medicine, 2015, 3, 320-326.	0.6	23
53	The Role of D4Z4-Encoded Proteins in the Osteogenic Differentiation of Mesenchymal Stromal Cells Isolated from Bone Marrow. Stem Cells and Development, 2015, 24, 2674-2686.	1.1	10
54	Positioning a Scientific Community on Unproven Cellular Therapies: The 2015 International Society for Cellular Therapy Perspective. Cytotherapy, 2015, 17, 1663-1666.	0.3	44

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55	Antisense Oligonucleotide Induction of Progerin in Human Myogenic Cells. PLoS ONE, 2014, 9, e98306.	1.1	10
56	Targeted Exon Skipping to Correct Exon Duplications in the Dystrophin Gene. Molecular Therapy - Nucleic Acids, 2014, 3, e155.	2.3	37
57	Normal and aberrant splicing ofLMNA. Journal of Medical Genetics, 2014, 51, 215-223.	1.5	30
58	Dystrophin as a therapeutic biomarker: Are we ignoring data from the past?. Neuromuscular Disorders, 2014, 24, 463-466.	0.3	15
59	Translation from a DMD exon 5 IRES results in a functional dystrophin isoform that attenuates dystrophinopathy in humans and mice. Nature Medicine, 2014, 20, 992-1000.	15.2	113
60	Primary overâ€expression of AÎ²<scp>PP</scp> in muscle does not lead to the development of inclusion body myositis in a new lineage of the <scp>MCK</scp>â€AÎ²<scp>PP</scp> transgenic mouse. International Journal of Experimental Pathology, 2013, 94, 418-425.	0.6	4
61	A Cell-Based High-Throughput Screening Assay for Posttranscriptional Utrophin Upregulation. Journal of Biomolecular Screening, 2013, 18, 400-406.	2.6	26
62	The influence of non-HLA gene polymorphisms and interactions on disease risk in a Western Australian multiple sclerosis cohort. Journal of Neuroimmunology, 2013, 261, 92-97.	1.1	16
63	Analysis of HLA-DRB3 alleles and supertypical genotypes in the MHC Class II region in sporadic inclusion body myositis. Journal of Neuroimmunology, 2013, 254, 174-177.	1.1	20
64	Complement-mediated muscle cell lysis: A possible mechanism of myonecrosis in anti-SRP associated necrotizing myopathy (ASANM). Journal of Neuroimmunology, 2013, 264, 65-70.	1.1	40
65	Translating the Genomics Revolution: The Need for an International Gene Therapy Consortium for Monogenic Diseases. Molecular Therapy, 2013, 21, 266-268.	3.7	12
66	Improved Antisense Oligonucleotide Design to Suppress Aberrant SMN2 Gene Transcript Processing: Towards a Treatment for Spinal Muscular Atrophy. PLoS ONE, 2013, 8, e62114.	1.1	63
67	Investigation of splicing changes and post-translational processing of LMNA in sporadic inclusion body myositis. International Journal of Clinical and Experimental Pathology, 2013, 6, 1723-33.	0.5	6
68	Targeted Exon Skipping to Address â€œLeakyâ€•Mutations in the Dystrophin Gene. Molecular Therapy - Nucleic Acids, 2012, 1, e48.	2.3	21
69	Multiple exon skipping strategies to by-pass dystrophin mutations. Neuromuscular Disorders, 2012, 22, 297-305.	0.3	17
70	Regulation of eukaryotic gene expression by the untranslated gene regions and other non-coding elements. Cellular and Molecular Life Sciences, 2012, 69, 3613-3634.	2.4	481
71	Optimizing Splice-Switching Oligomer Sequences Using 2â€²-O-Methyl Phosphorothioate Chemistry. Methods in Molecular Biology, 2012, 867, 169-188.	0.4	3
72	Current Status of Pharmaceutical and Genetic Therapeutic Approaches to Treat DMD. Molecular Therapy, 2011, 19, 830-840.	3.7	176

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73	Exon skipping and dystrophin restoration in patients with Duchenne muscular dystrophy after systemic phosphorodiamidate morpholino oligomer treatment: an open-label, phase 2, dose-escalation study. Lancet, The, 2011, 378, 595-605.	6.3	803
74	Novel compounds for the treatment of Duchenne muscular dystrophy: emerging therapeutic agents. The Application of Clinical Genetics, 2011, 4, 29.	1.4	2
75	The FSHD Atrophic Myotube Phenotype Is Caused by DUX4 Expression. PLoS ONE, 2011, 6, e26820.	1.1	146
76	RNA Splicing Manipulation: Strategies to Modify Gene Expression for a Variety of Therapeutic Outcomes. Current Gene Therapy, 2011, 11, 259-275.	0.9	18
77	Gene therapy: therapeutic applications and relevance to pathology. Pathology, 2011, 43, 642-656.	0.3	14
78	Evaluation of exon-skipping strategies for Duchenne muscular dystrophy utilizing dystrophin-deficient zebrafish. Journal of Cellular and Molecular Medicine, 2011, 15, 2643-2651.	1.6	36
79	Mismatched single stranded antisense oligonucleotides can induce efficient dystrophin splice switching. BMC Medical Genetics, 2011, 12, 141.	2.1	6
80	Prophylactic oligonucleotide-mediated enhancement of host acetylcholinesterase protects from organophosphate poisoning. , 2011, , .		2
81	Translational Regulation of Utrophin by miRNAs. PLoS ONE, 2011, 6, e29376.	1.1	44
82	Molecular diagnosis of duchenne muscular dystrophy: past, present and future in relation to implementing therapies. Clinical Biochemist Reviews, 2011, 32, 129-34.	3.3	25
83	Splice Modification to Restore Functional Dystrophin Synthesis in Duchenne Muscular Dystrophy. Current Pharmaceutical Design, 2010, 16, 988-1001.	0.9	13
84	Fibulin-1 Is Increased in Asthma â€“ A Novel Mediator of Airway Remodeling?. PLoS ONE, 2010, 5, e13360.	1.1	55
85	Prevention of Dystrophic Pathology in Severely Affected Dystrophin/Utrophin-deficient Mice by Morpholino-oligomer-mediated Exon-skipping. Molecular Therapy, 2010, 18, 198-205.	3.7	102
86	Dystrophin Isoform Induction In Vivo by Antisense-mediated Alternative Splicing. Molecular Therapy, 2010, 18, 1218-1223.	3.7	23
87	Comparative analysis of antisense oligonucleotide sequences targeting exon 53 of the human DMD gene: Implications for future clinical trials. Neuromuscular Disorders, 2010, 20, 102-110.	0.3	44
88	Personalized exon skipping strategies to address clustered non-deletion dystrophin mutations. Neuromuscular Disorders, 2010, 20, 810-816.	0.3	17
89	Personalised Genetic Intervention for Duchenne Muscular Dystrophy: Antisense Oligomers and Exon Skipping. Current Molecular Pharmacology, 2009, 2, 110-121.	0.7	18
90	Rational Design of Antisense Oligomers to Induce Dystrophin Exon Skipping. Molecular Therapy, 2009, 17, 1418-1426.	3.7	43

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91	Local restoration of dystrophin expression with the morpholino oligomer AVI-4658 in Duchenne muscular dystrophy: a single-blind, placebo-controlled, dose-escalation, proof-of-concept study. Lancet Neurology, The, 2009, 8, 918-928.	4.9	617
92	Characterization of a complex Duchenne muscular dystrophy-causing dystrophin gene inversion and restoration of the reading frame by induced exon skipping. Human Mutation, 2009, 30, 22-28.	1.1	41
93	Do polymorphisms in the familial Parkinsonism genes contribute to risk for sporadic Parkinson's disease?. Movement Disorders, 2009, 24, 833-838.	2.2	56
94	Byâ€passing the nonsense mutation in the 4^{CV} mouse model of muscular dystrophy by induced exon skipping. Journal of Gene Medicine, 2009, 11, 46-56.	1.4	44
95	Proteomic profiling of antisenseâ€induced exon skipping reveals reversal of pathobiochemical abnormalities in dystrophic mdx diaphragm. Proteomics, 2009, 9, 671-685.	1.3	66
96	DMD pseudoexon mutations: splicing efficiency, phenotype, and potential therapy. Annals of Neurology, 2008, 63, 81-89.	2.8	95
97	Exon skipping and Duchenne muscular dystrophy: Hope, hype and how feasible?. Neurology India, 2008, 56, 254.	0.2	24
98	Antisense Oligonucleotide-induced Exon Skipping Across the Human Dystrophin Gene Transcript. Molecular Therapy, 2007, 15, 1288-1296.	3.7	146
99	PTC124, nonsense mutations and Duchenne muscular dystrophy. Neuromuscular Disorders, 2007, 17, 719-720.	0.3	21
100	Antisense oligonucleotide induced exon skipping and the dystrophin gene transcript: cocktails and chemistries. BMC Molecular Biology, 2007, 8, 57.	3.0	66
101	Quantitative linkage genome scan for atopy in a large collection of Caucasian families. Human Genetics, 2007, 121, 83-92.	1.8	14
102	Systemic delivery of morpholino oligonucleotide restores dystrophin expression bodywide and improves dystrophic pathology. Nature Medicine, 2006, 12, 175-177.	15.2	468
103	Dystrophin expression in themdx mouse after localised and systemic administration of a morpholino antisense oligonucleotide. Journal of Gene Medicine, 2006, 8, 207-216.	1.4	169
104	Modification of pre-mRNA processing: application to dystrophin expression. Current Opinion in Molecular Therapeutics, 2006, 8, 130-5.	2.8	13
105	RNA Splicing Manipulation: Strategies to Modify Gene Expression for a Variety of Therapeutic Outcomes. Current Gene Therapy, 2005, 5, 467-483.	0.9	41
106	Antisense oligonucleotides in the treatment of Duchenne muscular dystrophy: Where are we now?. Neuromuscular Disorders, 2005, 15, 399-402.	0.3	17
107	Evaluation of a short interspersed nucleotide element in the 3' untranslated region of the defective dystrophin gene of dogs with muscular dystrophy. American Journal of Veterinary Research, 2001, 62, 1964-1968.	0.3	3
108	Gene therapy and molecular approaches to the treatment of hereditary muscular disorders. Current Opinion in Neurology, 2000, 13, 553-560.	1.8	11

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109	Butyrylcholinesterase K variant and Alzheimer's disease. Journal of Neurology, 1999, 246, 369-370.	1.8	29
110	Snapback SSCP analysis: Engineered conformation changes for the rapid typing of known mutations. , 1998, 11, 252-258.		12
111	Bandstab: A PCR-Based Alternative to Cloning PCR Products. BioTechniques, 1997, 22, 642-645.	0.8	43
112	Three novel mutations and two variants in the gene for Cu/Zn superoxide dismutase in familial amyotrophic lateral sclerosis. Neuromuscular Disorders, 1996, 6, 361-366.	0.3	45
113	Long-range PCR: synthesis of products independent of size. Trends in Genetics, 1996, 12, 458.	2.9	3