Susan Fletcher

List of Publications by Year
in descending order

Source: https://exaly.com/author-pdf/6526645/publications.pdf

Version: 2024-02-01

137 papers	5,557 citations	101384 36 h-index	91712 69 g-index
143 all docs	143 docs citations	143 times ranked	6019 citing authors

#	Article	IF	Citations
1	Stargardt disease and progress in therapeutic strategies. Ophthalmic Genetics, 2022, 43, 1-26.	0.5	18
2	Primary Nasal Epithelial Cells as a Surrogate Cell Culture Model for Type-II Alveolar Cells to Study ABCA-3 Deficiency. Frontiers in Medicine, 2022, 9, 827416.	1.2	0
3	Investigating the Implications of CFTR Exon Skipping Using a Cftr Exon 9 Deleted Mouse Model. Frontiers in Pharmacology, 2022, 13, 868863.	1.6	1
4	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
5	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
6	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
7	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
8	Proof-of-Concept: Antisense Oligonucleotide Mediated Skipping of Fibrillin-1 Exon 52. International Journal of Molecular Sciences, 2021, 22, 3479.	1.8	6
9	Novel STMN2 Variant Linked to Amyotrophic Lateral Sclerosis Risk and Clinical Phenotype. Frontiers in Aging Neuroscience, 2021, 13, 658226.	1.7	38
10	Targeted SMN Exon Skipping: A Useful Control to Assess In Vitro and In Vivo Splice-Switching Studies. Biomedicines, 2021, 9, 552.	1.4	6
11	Induction of cryptic pre-mRNA splice-switching by antisense oligonucleotides. Scientific Reports, 2021, 11, 15137.	1.6	4
12	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
13	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
14	Determinants of Disease Penetrance in PRPF31-Associated Retinopathy. Genes, 2021, 12, 1542.	1.0	14
15	Exploring microperimetry and autofluorescence endpoints for monitoring disease progression in PRPF31-associated retinopathy. Ophthalmic Genetics, 2021, 42, 1-14.	0.5	8
16	Gene replacement therapy restores RCBTB1 expression and cilium length in patientâ€derived retinal pigment epithelium. Journal of Cellular and Molecular Medicine, 2021, 25, 10020-10027.	1.6	3
17	A spotterâ€™s guide to SNPtic exons: The common splice variants underlying some SNPâ€“phenotype correlations. Molecular Genetics & Genomic Medicine, 2021, , e1840.	0.6	2
18	Analysis of Pathogenic Pseudoexons Reveals Novel Mechanisms Driving Cryptic Splicing. Frontiers in Genetics, 2021, 12, 806946.	1.1	14

#	Article	IF	Citations
19	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
20	Nonsequential Splicing Events Alter Antisense-Mediated Exon Skipping Outcome in COL7A1. International Journal of Molecular Sciences, 2020, 21, 7705.	1.8	15
21	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
22	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
23	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
24	Single Exon Skipping Can Address a Multi-Exon Duplication in the Dystrophin Gene. International Journal of Molecular Sciences, 2020, 21, 4511.	1.8	3
25	Novel Mutations Found in Individuals with Adult-Onset Pompe Disease. Genes, 2020, 11, 135.	1.0	7
26	Structural Variants May Be a Source of Missing Heritability in sALS. Frontiers in Neuroscience, 2020, 14, 47.	1.4	43
27	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
28	Systematic Approach to Developing Splice Modulating Antisense Oligonucleotides. , 2020, , .		0
29	Rescue of CFTR function impaired by mutations in exon 15. , 2020, , .		0
30	In Vitro Validation of Phosphorodiamidate Morpholino Oligomers. Molecules, 2019, 24, 2922.	1.7	16
31	Systematic Approach to Developing Splice Modulating Antisense Oligonucleotides. International Journal of Molecular Sciences, 2019, 20, 5030.	1.8	14
32	Breakpoint junction features of seven DMD deletion mutations. Human Genome Variation, 2019, 6, 39.	0.4	7
33	Reduction of integrin alpha 4 activity through splice modulating antisense oligonucleotides. Scientific Reports, 2019, 9, 12994.	1.6	14
34	Link-me: Protocol for a randomised controlled trial of a systematic approach to stepped mental health care in primary care. Contemporary Clinical Trials, 2019, 78, 63-75.	0.8	4
35	Consequences of Making the Inactive Active Through Changes in Antisense Oligonucleotide Chemistries. Frontiers in Genetics, 2019, 10, 1249.	1.1	3
36	Removal of the Polyglutamine Repeat of Ataxin-3 by Redirecting pre-mRNA Processing. International Journal of Molecular Sciences, 2019, 20, 5434.	1.8	9

#	Article	IF	Citations
37	ALS Genetics, Mechanisms, and Therapeutics: Where Are We Now?. Frontiers in Neuroscience, 2019, 13, 1310.	1.4	487
38	<scp>YAP</scp>ping about and not forgetting <scp>TAZ</scp>. FEBS Letters, 2019, 593, 253-276.	1.3	31
39	Generation of two induced pluripotent stem cell lines from a patient with dominant PRPF31 mutation and a related non-penetrant carrier. Stem Cell Research, 2019, 34, 101357.	0.3	7
40	Antisense-mediated splice intervention to treat human disease: the odyssey continues. F1000Research, 2019, 8, 710.	0.8	11
41	Challenges of Interpreting Dystrophin Content by Western Blot. US Neurology, 2019, 15, 40.	0.2	7
42	Design of a framework for the deployment of collaborative independent rare disease-centric registries: Gaucher disease registry model. Blood Cells, Molecules, and Diseases, 2018, 68, 232-238.	0.6	17
43	Precision Medicine through Antisense Oligonucleotide-Mediated Exon Skipping. Trends in Pharmacological Sciences, 2018, 39, 982-994.	4.0	51
44	Skipping of Duplicated Dystrophin Exons: In Vitro Induction and Assessment. Methods in Molecular Biology, 2018, 1828, 219-228.	0.4	1
45	A Morpholino Oligomer Therapy Regime That Restores Mitochondrial Function and Prevents mdx Cardiomyopathy. JACC Basic To Translational Science, 2018, 3, 391-402.	1.9	5
46	The potential of antisense oligonucleotide therapies for inherited childhood lung diseases. Molecular and Cellular Pediatrics, 2018, 5, 3.	1.0	21
47	A platform for discovery of functional cell-penetrating peptides for efficient multi-cargo intracellular delivery. Scientific Reports, 2018, 8, 12538.	1.6	50
48	Antisense Oligonucleotide-Mediated Terminal Intron Retention of the SMN2 Transcript. Molecular Therapy - Nucleic Acids, 2018, 11, 91-102.	2.3	16
49	Response to â€œRailroading at the FDAâ€• Nature Biotechnology, 2017, 35, 207-209.	9.4	6
50	Rational Design of Short Locked Nucleic Acid-Modified 2â€²-O-Methyl Antisense Oligonucleotides for Efficient Exon-Skipping InÂVitro. Molecular Therapy - Nucleic Acids, 2017, 9, 155-161.	2.3	33
51	Translational development of splice-modifying antisense oligomers. Expert Opinion on Biological Therapy, 2017, 17, 15-30.	1.4	19
52	Comprehending the Health Informatics Spectrum: Grappling with System Entropy and Advancing Quality Clinical Research. Frontiers in Public Health, 2017, 5, 224.	1.3	2
53	Inherited Retinal Disease Therapies Targeting Precursor Messenger Ribonucleic Acid. Vision (Switzerland), 2017, 1, 22.	0.5	5
54	Dystrophin expression in the non-DMD population: What is normal?. Neuromuscular Disorders, 2016, 26, S160.	0.3	1

#	Article	IF	Citations
55	Antisense oligonucleotide development for the treatment of muscular dystrophies. Expert Opinion on Orphan Drugs, 2016, 4, 139-152.	0.5	18
56	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
57	Morpholino Oligomer Peptide Therapy Improves Mitochondrial Function in mdx Cardiomyopathy. Biophysical Journal, 2015, 108, 581a-582a.	0.2	0
58	The emperor's new dystrophin: finding sense in the noise. Trends in Molecular Medicine, 2015, 21, 417-426.	3.5	37
59	High-throughput Phenotyping of Wheat Seminal Root Traits in a Breeding Context. Procedia Environmental Sciences, 2015, 29, 102-103.	1.3	7
60	Pseudoexon activation increases phenotype severity in a Becker muscular dystrophy patient. Molecular Genetics & Genomic Medicine, 2015, 3, 320-326.	0.6	23
61	High-throughput phenotyping of seminal root traits in wheat. Plant Methods, 2015, 11, 13.	1.9	150
62	Smart functional nucleic acid chimeras: Enabling tissue specific RNA targeting therapy. RNA Biology, 2015, 12, 412-425.	1.5	32
63	A Registry Framework Enabling Patient-Centred Care. Studies in Health Technology and Informatics, 2015, 214, 8-14.	0.2	11
64	Antisense Oligonucleotide Induction of Progerin in Human Myogenic Cells. PLoS ONE, 2014, 9, e98306.	1.1	10
65	Targeted Exon Skipping to Correct Exon Duplications in the Dystrophin Gene. Molecular Therapy - Nucleic Acids, 2014, 3, e155.	2.3	37
66	Impaired functional communication between the L-type calcium channel and mitochondria contributes to metabolic inhibition in the mdx heart. Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, E2905-14.	3.3	42
67	Dystrophin as a therapeutic biomarker: Are we ignoring data from the past?. Neuromuscular Disorders, 2014, 24, 463-466.	0.3	15
68	Rare Disease Research Roadmap: Navigating the bioinformatics and translational challenges for improved patient health outcomes. Health Policy and Technology, 2014, 3, 325-335.	1.3	9
69	QTL for root angle and number in a population developed from bread wheats (Triticum aestivum) with contrasting adaptation to water-limited environments. Theoretical and Applied Genetics, 2013, 126, 1563-1574.	1.8	160
70	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
71	P.11.5 PMO-mediated dystrophin exon 23 skipping restores mitochondrial function in the mdx mouse heart. Neuromuscular Disorders, 2013, 23, 800.	0.3	1
72	Antisense suppression of donor splice site mutations in the dystrophin gene transcript. Molecular Genetics & Genomic Medicine, 2013, 1, 162-173.	0.6	11

#	Article	IF	Citations
73	Exploration of Delayed-Onset Posttraumatic Stress Disorder After Severe Injury. Psychosomatic Medicine, 2013, 75, 68-75.	1.3	19
74	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
75	Revertant Fibers in the mdx Murine Model of Duchenne Muscular Dystrophy: An Age- and Muscle-Related Reappraisal. PLoS ONE, 2013, 8, e72147.	1.1	27
76	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
77	Investigation of age-related changes in LMNA splicing and expression of progerin in human skeletal muscles. International Journal of Clinical and Experimental Pathology, 2013, 6, 2778-86.	0.5	10
78	Targeted Exon Skipping to Address â€œLeakyâ€•Mutations in the Dystrophin Gene. Molecular Therapy - Nucleic Acids, 2012, 1, e48.	2.3	21
79	Multiple exon skipping strategies to by-pass dystrophin mutations. Neuromuscular Disorders, 2012, 22, 297-305.	0.3	17
80	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
81	Optimizing Splice-Switching Oligomer Sequences Using 2â€²-O-Methyl Phosphorothioate Chemistry. Methods in Molecular Biology, 2012, 867, 169-188.	0.4	3
82	Trauma at the Hands of Another. Journal of Clinical Psychiatry, 2012, 73, 372-376.	1.1	90
83	Novel compounds for the treatment of Duchenne muscular dystrophy: emerging therapeutic agents. The Application of Clinical Genetics, 2011, 4, 29.	1.4	2
84	RNA Splicing Manipulation: Strategies to Modify Gene Expression for a Variety of Therapeutic Outcomes. Current Gene Therapy, 2011, 11, 259-275.	0.9	18
85	Gene therapy: therapeutic applications and relevance to pathology. Pathology, 2011, 43, 642-656.	0.3	14
86	Requiring both avoidance and emotional numbing in DSM-V PTSD: Will it help?. Journal of Affective Disorders, 2011, 130, 483-486.	2.0	52
87	Mismatched single stranded antisense oligonucleotides can induce efficient dystrophin splice switching. BMC Medical Genetics, 2011, 12, 141.	2.1	6
88	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
89	Splice Modification to Restore Functional Dystrophin Synthesis in Duchenne Muscular Dystrophy. Current Pharmaceutical Design, 2010, 16, 988-1001.	0.9	13
90	Splice-switching as a treament for duchenne muscular dystrophy. Pathology, 2010, 42, S31.	0.3	0

#	Article	IF	Citations
91	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
92	Dystrophin Isoform Induction In Vivo by Antisense-mediated Alternative Splicing. Molecular Therapy, 2010, 18, 1218-1223.	3.7	23
93	Personalized exon skipping strategies to address clustered non-deletion dystrophin mutations. Neuromuscular Disorders, 2010, 20, 810-816.	0.3	17
94	Personalised Genetic Intervention for Duchenne Muscular Dystrophy: Antisense Oligomers and Exon Skipping. Current Molecular Pharmacology, 2009, 2, 110-121.	0.7	18
95	Rational Design of Antisense Oligomers to Induce Dystrophin Exon Skipping. Molecular Therapy, 2009, 17, 1418-1426.	3.7	43
96	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
97	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
98	Proteomic profiling of antisenseâ€induced exon skipping reveals reversal of pathobiochemical abnormalities in dystrophic mdx diaphragm. Proteomics, 2009, 9, 671-685.	1.3	66
99	Long-term administration of antisense oligonucleotides into the paraspinal muscles of mdx mice reduces kyphosis. Journal of Applied Physiology, 2008, 105, 662-668.	1.2	13
100	Exon skipping and Duchenne muscular dystrophy: Hope, hype and how feasible?. Neurology India, 2008, 56, 254.	0.2	24
101	The Influence of Antisense Oligonucleotide Length on Dystrophin Exon Skipping. Molecular Therapy, 2007, 15, 157-166.	3.7	74
102	Antisense Oligonucleotide-induced Exon Skipping Across the Human Dystrophin Gene Transcript. Molecular Therapy, 2007, 15, 1288-1296.	3.7	146
103	Morpholino Oligomerâ€"Mediated Exon Skipping Averts the Onset of Dystrophic Pathology in the mdx Mouse. Molecular Therapy, 2007, 15, 1587-1592.	3.7	150
104	Antisense oligonucleotide induced exon skipping and the dystrophin gene transcript: cocktails and chemistries. BMC Molecular Biology, 2007, 8, 57.	3.0	66
105	Induced dystrophin exon skipping in human muscle explants. Neuromuscular Disorders, 2006, 16, 583-590.	0.3	63
106	Induction of revertant fibres in the mdx mouse using antisense oligonucleotides. Genetic Vaccines and Therapy, 2006, 4, 3.	1.5	33
107	Antisense oligonucleotide-induced exon skipping restores dystrophin expression in vitro in a canine model of DMD. Gene Therapy, 2006, 13, 1373-1381.	2.3	193
108	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

#	Article	IF	Citations
109	Redirecting Splicing to Address Dystrophin Mutations: Molecular By-pass Surgery. Progress in Molecular and Subcellular Biology, 2006, 44, 161-197.	0.9	5
110	Modification of pre-mRNA processing: application to dystrophin expression. Current Opinion in Molecular Therapeutics, 2006, 8, 130-5.	2.8	13
111	RNA Splicing Manipulation: Strategies to Modify Gene Expression for a Variety of Therapeutic Outcomes. Current Gene Therapy, 2005, 5, 467-483.	0.9	41
112	Splicing intervention for Duchenne muscular dystrophy. Current Opinion in Pharmacology, 2005, 5, 529-534.	1.7	24
113	Antisense oligonucleotides in the treatment of Duchenne muscular dystrophy: Where are we now?. Neuromuscular Disorders, 2005, 15, 399-402.	0.3	17
114	Terminal antisense oligonucleotide modifications can enhance induced exon skipping. Neuromuscular Disorders, 2005, 15, 622-629.	0.3	17
115	Target selection for antisense oligonucleotide induced exon skipping in the dystrophin gene. Journal of Gene Medicine, 2003, 5, 518-527.	1.4	38
116	Functional amounts of dystrophin produced by skipping the mutated exon in the mdx dystrophic mouse. Nature Medicine, 2003, 9, 1009-1014.	15.2	367
117	Enhanced in vivo delivery of antisense oligonucleotides to restore dystrophin expression in adult mdx mouse muscle. FEBS Letters, 2003, 552, 145-149.	1.3	50
118	Morpholino antisense oligonucleotide induced dystrophin exon 23 skipping in mdx mouse muscle. Human Molecular Genetics, 2003, 12, 1801-1811.	1.4	183
119	Improved antisense oligonucleotide induced exon skipping in themdx mouse model of muscular dystrophy. Journal of Gene Medicine, 2002, 4, 644-654.	1.4	132
120	Cryptic splicing involving the splice site mutation in the canine model of Duchenne muscular dystrophy. Neuromuscular Disorders, 2001, 11, 239-243.	0.3	10
121	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
122	The spread of transgene expression at the site of gene construct injection. Muscle and Nerve, 2001, 24, 488-495.	1.0	28
123	Dystrophin Expression in Muscle Following Gene Transfer with a Fully Deleted ("Gutted") Adenovirus Is Markedly Improved by Trans-Acting Adenoviral Gene Products. Human Gene Therapy, 2001, 12, 1741-1755.	1.4	56
124	Antisense-induced exon skipping and synthesis of dystrophin in the mdx mouse. Proceedings of the National Academy of Sciences of the United States of America, 2001, 98, 42-7.	3.3	192
125	Gene therapy and molecular approaches to the treatment of hereditary muscular disorders. Current Opinion in Neurology, 2000, 13, 553-560.	1.8	11
126	Specific removal of the nonsense mutation from the mdx dystrophin mRNA using antisense oligonucleotides. Neuromuscular Disorders, 1999, 9, 330-338.	0.3	190

#	Article	IF	Citations
127	Direct dystrophin and reporter gene transfer into dog muscle in vivo. , 1998, 21, 159-165.		12
128	Snapback SSCP analysis: Engineered conformation changes for the rapid typing of known mutations. , 1998, 11, 252-258.		12
129	Pax7 includes two polymorphic homeoboxes which contain rearrangements associated with differences in the ability to regenerate damaged skeletal muscle in adult mice. International Journal of Biochemistry and Cell Biology, 1998, 30, 261-269.	1.2	8
130	High-Level Dystrophin Expression after Adenovirus-Mediated Dystrophin Minigene Transfer to Skeletal Muscle of Dystrophic Dogs: Prolongation of Expression with Immunosuppression. Human Gene Therapy, 1998, 9, 629-634.	1.4	72
131	Use of the dog model for Duchenne muscular dystrophy in gene therapy trials. Neuromuscular Disorders, 1997, 7, 325-328.	0.3	49
132	Alternate Pax7 transcripts are expressed specifically in skeletal muscle, brain and other organs of adult mice. International Journal of Biochemistry and Cell Biology, 1997, 29, 1029-1036.	1.2	31
133	Variation in the methylation profile and structure of Pax3 and Pax7 among different mouse strains and during expression. Gene, 1997, 184, 45-53.	1.0	15
134	Calcium phosphate transfection and cell-specific expression of heterologous genes in primary fetal rat hepatocytes. International Journal of Biochemistry and Cell Biology, 1996, 28, 639-650.	1.2	6
135	Specific cloning of DNA fragments unique to the dog Y chromosome. Genetic Analysis, Techniques and Applications, 1993, 10, 77-83.	1.5	4
136	Quantitation of muscle precursor cell activity in skeletal muscle by Northern analysis of MyoD and myogenin expression: Application to dystrophic (mdx) mouse muscle. Molecular and Cellular Neurosciences, 1992, 3, 326-331.	1.0	44
137	The development of rat alpha2-macroglobulin. Studies in vivo and in cultured fetal rat hepatocytes. FEBS Journal, 1988, 171, 703-709.	0.2	32