Susan Fletcher

List of Publications by Year
in descending order

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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	ALS Genetics, Mechanisms, and Therapeutics: Where Are We Now?. Frontiers in Neuroscience, 2019, 13, 1310.	1.4	487
2	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
3	Functional amounts of dystrophin produced by skipping the mutated exon in the mdx dystrophic mouse. Nature Medicine, 2003, 9, 1009-1014.	15.2	367
4	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
5	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
6	Specific removal of the nonsense mutation from the mdx dystrophin mRNA using antisense oligonucleotides. Neuromuscular Disorders, 1999, 9, 330-338.	0.3	190
7	Morpholino antisense oligonucleotide induced dystrophin exon 23 skipping in mdx mouse muscle. Human Molecular Genetics, 2003, 12, 1801-1811.	1.4	183
8	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
9	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
10	Morpholino Oligomerâ€“Mediated Exon Skipping Averts the Onset of Dystrophic Pathology in the mdx Mouse. Molecular Therapy, 2007, 15, 1587-1592.	3.7	150
11	High-throughput phenotyping of seminal root traits in wheat. Plant Methods, 2015, 11, 13.	1.9	150
12	Antisense Oligonucleotide-induced Exon Skipping Across the Human Dystrophin Gene Transcript. Molecular Therapy, 2007, 15, 1288-1296.	3.7	146
13	Improved antisense oligonucleotide induced exon skipping in themdx mouse model of muscular dystrophy. Journal of Gene Medicine, 2002, 4, 644-654.	1.4	132
14	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
15	Trauma at the Hands of Another. Journal of Clinical Psychiatry, 2012, 73, 372-376.	1.1	90
16	The Influence of Antisense Oligonucleotide Length on Dystrophin Exon Skipping. Molecular Therapy, 2007, 15, 157-166.	3.7	74
17	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
18	Antisense oligonucleotide induced exon skipping and the dystrophin gene transcript: cocktails and chemistries. BMC Molecular Biology, 2007, 8, 57.	3.0	66

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19	Proteomic profiling of antisenseâ€induced exon skipping reveals reversal of pathobiochemical abnormalities in dystrophic mdx diaphragm. Proteomics, 2009, 9, 671-685.	1.3	66
20	Induced dystrophin exon skipping in human muscle explants. Neuromuscular Disorders, 2006, 16, 583-590.	0.3	63
21	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
22	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
23	Requiring both avoidance and emotional numbing in DSM-V PTSD: Will it help?. Journal of Affective Disorders, 2011, 130, 483-486.	2.0	52
24	Precision Medicine through Antisense Oligonucleotide-Mediated Exon Skipping. Trends in Pharmacological Sciences, 2018, 39, 982-994.	4.0	51
25	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
26	A platform for discovery of functional cell-penetrating peptides for efficient multi-cargo intracellular delivery. Scientific Reports, 2018, 8, 12538.	1.6	50
27	Use of the dog model for Duchenne muscular dystrophy in gene therapy trials. Neuromuscular Disorders, 1997, 7, 325-328.	0.3	49
28	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
29	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
30	Rational Design of Antisense Oligomers to Induce Dystrophin Exon Skipping. Molecular Therapy, 2009, 17, 1418-1426.	3.7	43
31	Structural Variants May Be a Source of Missing Heritability in sALS. Frontiers in Neuroscience, 2020, 14, 47.	1.4	43
32	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
33	RNA Splicing Manipulation: Strategies to Modify Gene Expression for a Variety of Therapeutic Outcomes. Current Gene Therapy, 2005, 5, 467-483.	0.9	41
34	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
35	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
36	Target selection for antisense oligonucleotide induced exon skipping in the dystrophin gene. Journal of Gene Medicine, 2003, 5, 518-527.	1.4	38

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37	Novel STMN2 Variant Linked to Amyotrophic Lateral Sclerosis Risk and Clinical Phenotype. Frontiers in Aging Neuroscience, 2021, 13, 658226.	1.7	38
38	Targeted Exon Skipping to Correct Exon Duplications in the Dystrophin Gene. Molecular Therapy - Nucleic Acids, 2014, 3, e155.	2.3	37
39	The emperor's new dystrophin: finding sense in the noise. Trends in Molecular Medicine, 2015, 21, 417-426.	3.5	37
40	Induction of revertant fibres in the mdx mouse using antisense oligonucleotides. Genetic Vaccines and Therapy, 2006, 4, 3.	1.5	33
41	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
42	The development of rat alpha2-macroglobulin. Studies in vivo and in cultured fetal rat hepatocytes. FEBS Journal, 1988, 171, 703-709.	0.2	32
43	Smart functional nucleic acid chimeras: Enabling tissue specific RNA targeting therapy. RNA Biology, 2015, 12, 412-425.	1.5	32
44	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
45	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
46	<scp>YAP</scp>ping about and not forgetting <scp>TAZ</scp>. FEBS Letters, 2019, 593, 253-276.	1.3	31
47	The spread of transgene expression at the site of gene construct injection. Muscle and Nerve, 2001, 24, 488-495.	1.0	28
48	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
49	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
50	Splicing intervention for Duchenne muscular dystrophy. Current Opinion in Pharmacology, 2005, 5, 529-534.	1.7	24
51	Exon skipping and Duchenne muscular dystrophy: Hope, hype and how feasible?. Neurology India, 2008, 56, 254.	0.2	24
52	Dystrophin Isoform Induction In Vivo by Antisense-mediated Alternative Splicing. Molecular Therapy, 2010, 18, 1218-1223.	3.7	23
53	Pseudoexon activation increases phenotype severity in a Becker muscular dystrophy patient. Molecular Genetics & Genomic Medicine, 2015, 3, 320-326.	0.6	23
54	Targeted Exon Skipping to Address â€œLeakyâ€•Mutations in the Dystrophin Gene. Molecular Therapy - Nucleic Acids, 2012, 1, e48.	2.3	21

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55	The potential of antisense oligonucleotide therapies for inherited childhood lung diseases. Molecular and Cellular Pediatrics, 2018, 5, 3.	1.0	21
56	Exploration of Delayed-Onset Posttraumatic Stress Disorder After Severe Injury. Psychosomatic Medicine, 2013, 75, 68-75.	1.3	19
57	Translational development of splice-modifying antisense oligomers. Expert Opinion on Biological Therapy, 2017, 17, 15-30.	1.4	19
58	Personalised Genetic Intervention for Duchenne Muscular Dystrophy: Antisense Oligomers and Exon Skipping. Current Molecular Pharmacology, 2009, 2, 110-121.	0.7	18
59	RNA Splicing Manipulation: Strategies to Modify Gene Expression for a Variety of Therapeutic Outcomes. Current Gene Therapy, 2011, 11, 259-275.	0.9	18
60	Antisense oligonucleotide development for the treatment of muscular dystrophies. Expert Opinion on Orphan Drugs, 2016, 4, 139-152.	0.5	18
61	Stargardt disease and progress in therapeutic strategies. Ophthalmic Genetics, 2022, 43, 1-26.	0.5	18
62	Antisense oligonucleotides in the treatment of Duchenne muscular dystrophy: Where are we now?. Neuromuscular Disorders, 2005, 15, 399-402.	0.3	17
63	Terminal antisense oligonucleotide modifications can enhance induced exon skipping. Neuromuscular Disorders, 2005, 15, 622-629.	0.3	17
64	Personalized exon skipping strategies to address clustered non-deletion dystrophin mutations. Neuromuscular Disorders, 2010, 20, 810-816.	0.3	17
65	Multiple exon skipping strategies to by-pass dystrophin mutations. Neuromuscular Disorders, 2012, 22, 297-305.	0.3	17
66	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
67	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
68	Antisense Oligonucleotide-Mediated Terminal Intron Retention of the SMN2 Transcript. Molecular Therapy - Nucleic Acids, 2018, 11, 91-102.	2.3	16
69	In Vitro Validation of Phosphorodiamidate Morpholino Oligomers. Molecules, 2019, 24, 2922.	1.7	16
70	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
71	Dystrophin as a therapeutic biomarker: Are we ignoring data from the past?. Neuromuscular Disorders, 2014, 24, 463-466.	0.3	15
72	Nonsequential Splicing Events Alter Antisense-Mediated Exon Skipping Outcome in COL7A1. International Journal of Molecular Sciences, 2020, 21, 7705.	1.8	15

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73	Gene therapy: therapeutic applications and relevance to pathology. Pathology, 2011, 43, 642-656.	0.3	14
74	Systematic Approach to Developing Splice Modulating Antisense Oligonucleotides. International Journal of Molecular Sciences, 2019, 20, 5030.	1.8	14
75	Reduction of integrin alpha 4 activity through splice modulating antisense oligonucleotides. Scientific Reports, 2019, 9, 12994.	1.6	14
76	Determinants of Disease Penetrance in PRPF31-Associated Retinopathy. Genes, 2021, 12, 1542.	1.0	14
77	Analysis of Pathogenic Pseudoexons Reveals Novel Mechanisms Driving Cryptic Splicing. Frontiers in Genetics, 2021, 12, 806946.	1.1	14
78	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
79	Splice Modification to Restore Functional Dystrophin Synthesis in Duchenne Muscular Dystrophy. Current Pharmaceutical Design, 2010, 16, 988-1001.	0.9	13
80	Modification of pre-mRNA processing: application to dystrophin expression. Current Opinion in Molecular Therapeutics, 2006, 8, 130-5.	2.8	13
81	Direct dystrophin and reporter gene transfer into dog muscle in vivo. , 1998, 21, 159-165.		12
82	Snapback SSCP analysis: Engineered conformation changes for the rapid typing of known mutations. , 1998, 11, 252-258.		12
83	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
84	Gene therapy and molecular approaches to the treatment of hereditary muscular disorders. Current Opinion in Neurology, 2000, 13, 553-560.	1.8	11
85	Antisense suppression of donor splice site mutations in the dystrophin gene transcript. Molecular Genetics & Genomic Medicine, 2013, 1, 162-173.	0.6	11
86	Antisense-mediated splice intervention to treat human disease: the odyssey continues. F1000Research, 2019, 8, 710.	0.8	11
87	A Registry Framework Enabling Patient-Centred Care. Studies in Health Technology and Informatics, 2015, 214, 8-14.	0.2	11
88	Cryptic splicing involving the splice site mutation in the canine model of Duchenne muscular dystrophy. Neuromuscular Disorders, 2001, 11, 239-243.	0.3	10
89	Antisense Oligonucleotide Induction of Progerin in Human Myogenic Cells. PLoS ONE, 2014, 9, e98306.	1.1	10
90	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

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91	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
92	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
93	Removal of the Polyglutamine Repeat of Ataxin-3 by Redirecting pre-mRNA Processing. International Journal of Molecular Sciences, 2019, 20, 5434.	1.8	9
94	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
95	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
96	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
97	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
98	Exploring microperimetry and autofluorescence endpoints for monitoring disease progression in PRPF31-associated retinopathy. Ophthalmic Genetics, 2021, 42, 1-14.	0.5	8
99	High-throughput Phenotyping of Wheat Seminal Root Traits in a Breeding Context. Procedia Environmental Sciences, 2015, 29, 102-103.	1.3	7
100	Breakpoint junction features of seven DMD deletion mutations. Human Genome Variation, 2019, 6, 39.	0.4	7
101	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
102	Novel Mutations Found in Individuals with Adult-Onset Pompe Disease. Genes, 2020, 11, 135.	1.0	7
103	Challenges of Interpreting Dystrophin Content by Western Blot. US Neurology, 2019, 15, 40.	0.2	7
104	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
105	Mismatched single stranded antisense oligonucleotides can induce efficient dystrophin splice switching. BMC Medical Genetics, 2011, 12, 141.	2.1	6
106	Response to â€œRailroading at the FDAâ€• Nature Biotechnology, 2017, 35, 207-209.	9.4	6
107	Proof-of-Concept: Antisense Oligonucleotide Mediated Skipping of Fibrillin-1 Exon 52. International Journal of Molecular Sciences, 2021, 22, 3479.	1.8	6
108	Targeted SMN Exon Skipping: A Useful Control to Assess In Vitro and In Vivo Splice-Switching Studies. Biomedicines, 2021, 9, 552.	1.4	6

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109	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
110	Inherited Retinal Disease Therapies Targeting Precursor Messenger Ribonucleic Acid. Vision (Switzerland), 2017, 1, 22.	0.5	5
111	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
112	Redirecting Splicing to Address Dystrophin Mutations: Molecular By-pass Surgery. Progress in Molecular and Subcellular Biology, 2006, 44, 161-197.	0.9	5
113	Specific cloning of DNA fragments unique to the dog Y chromosome. Genetic Analysis, Techniques and Applications, 1993, 10, 77-83.	1.5	4
114	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
115	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
116	Induction of cryptic pre-mRNA splice-switching by antisense oligonucleotides. Scientific Reports, 2021, 11, 15137.	1.6	4
117	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
118	Optimizing Splice-Switching Oligomer Sequences Using 2â€²-O-Methyl Phosphorothioate Chemistry. Methods in Molecular Biology, 2012, 867, 169-188.	0.4	3
119	Consequences of Making the Inactive Active Through Changes in Antisense Oligonucleotide Chemistries. Frontiers in Genetics, 2019, 10, 1249.	1.1	3
120	Single Exon Skipping Can Address a Multi-Exon Duplication in the Dystrophin Gene. International Journal of Molecular Sciences, 2020, 21, 4511.	1.8	3
121	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
122	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
123	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
124	Novel compounds for the treatment of Duchenne muscular dystrophy: emerging therapeutic agents. The Application of Clinical Genetics, 2011, 4, 29.	1.4	2
125	Comprehending the Health Informatics Spectrum: Grappling with System Entropy and Advancing Quality Clinical Research. Frontiers in Public Health, 2017, 5, 224.	1.3	2
126	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

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127	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
128	Dystrophin expression in the non-DMD population: What is normal?. Neuromuscular Disorders, 2016, 26, S160.	0.3	1
129	Skipping of Duplicated Dystrophin Exons: In Vitro Induction and Assessment. Methods in Molecular Biology, 2018, 1828, 219-228.	0.4	1
130	Investigating the Implications of CFTR Exon Skipping Using a Cftr Exon 9 Deleted Mouse Model. Frontiers in Pharmacology, 2022, 13, 868863.	1.6	1
131	Splice-switching as a treament for duchenne muscular dystrophy. Pathology, 2010, 42, S31.	0.3	0
132	Morpholino Oligomer Peptide Therapy Improves Mitochondrial Function in mdx Cardiomyopathy. Biophysical Journal, 2015, 108, 581a-582a.	0.2	0
133	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
134	Systematic Approach to Developing Splice Modulating Antisense Oligonucleotides. , 2020, , .		0
135	Rescue of CFTR function impaired by mutations in exon 15. , 2020, , .		0
136	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
137	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