Sara E Mole

List of Publications by Year
in descending order

140 papers	8,889 citations	66343 42 h-index	45317 90 g-index
154 all docs	154 docs citations	154 times ranked	7789 citing authors

#	Article	IF	Citations
1	Aberrant upregulation of the glycolytic enzyme PFKFB3 in CLN7 neuronal ceroid lipofuscinosis. Nature Communications, 2022, 13, 536.	12.8	14
2	Sex bias and omission exists in Batten disease research: Systematic review of the use of animal disease models. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2022, 1868, 166489.	3.8	3
3	Urine proteomics analysis of patients with neuronal ceroid lipofuscinoses. IScience, 2021, 24, 102020.	4.1	12
4	Global network analysis in Schizosaccharomyces pombe reveals three distinct consequences of the common 1-kb deletion causing juvenile CLN3 disease. Scientific Reports, 2021, 11, 6332.	3.3	9
5	Guidelines on the diagnosis, clinical assessments, treatment and management for CLN2 disease patients. Orphanet Journal of Rare Diseases, 2021, 16, 185.	2.7	17
6	Cerebrospinal fluid neurofilament light levels in CLN2 disease patients treated with enzyme replacement therapy normalise after two years on treatment. F1000Research, 2021, 10, 614.	1.6	4
7	Repurposing of tamoxifen ameliorates CLN3 and CLN7 disease phenotype. EMBO Molecular Medicine, 2021, 13, e13742.	6.9	28
8	Revealing the clinical phenotype of atypical neuronal ceroid lipofuscinosis type 2 disease: Insights from the largest cohort in the world. Journal of Paediatrics and Child Health, 2021, 57, 519-525.	0.8	15
9	Safe and stable generation of induced pluripotent stem cells using doggybone DNA vectors. Molecular Therapy - Methods and Clinical Development, 2021, 23, 348-358.	4.1	5
10	The Genetic Basis of Phenotypic Heterogeneity in the Neuronal Ceroid Lipofuscinoses. Frontiers in Neurology, 2021, 12, 754045.	2.4	24
11	Moving towards a new era of genomics in the neuronal ceroid lipofuscinoses. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2020, 1866, 165571.	3.8	43
12	Future perspectives: What lies ahead for Neuronal Ceroid Lipofuscinosis research?. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2020, 1866, 165681.	3.8	12
13	The neuronal ceroid-lipofuscinoses (Batten disease). , 2020, , 53-71.		3
14	Special edition: The NCLs/Batten disease. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2020, 1866, 165824.	3.8	1
15	Gene Therapy Targeting the Inner Retina Rescues the Retinal Phenotype in a Mouse Model of CLN3 Batten Disease. Human Gene Therapy, 2020, 31, 709-718.	2.7	31
16	Experimental gene therapies for the NCLs. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2020, 1866, 165772.	3.8	11
17	Mutation update: Review of TPP1 gene variants associated with neuronal ceroid lipofuscinosis CLN2 disease. Human Mutation, 2019, 40, 1924-1938.	2.5	46
18	Neonatal brain-directed gene therapy rescues a mouse model of neurodegenerative CLN6 Batten disease. Human Molecular Genetics, 2019, 28, 3867-3879.	2.9	21

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19	Kufs disease due to mutation ofCLN6: clinical, pathological and molecular genetic features. Brain, 2019, 142, 59-69.	7.6	28
20	Clinical challenges and future therapeutic approaches for neuronal ceroid lipofuscinosis. Lancet Neurology, The, 2019, 18, 107-116.	10.2	128
21	Loss of CLN7 results in depletion of soluble lysosomal proteins and impaired mTOR reactivation. Human Molecular Genetics, 2018, 27, 1711-1722.	2.9	47
22	Prevention of Photoreceptor Cell Loss in a Cln6 Mouse Model of Batten Disease Requires CLN6 Gene Transfer to Bipolar Cells. Molecular Therapy, 2018, 26, 1343-1353.	8.2	39
23	Gene Therapy Approaches to Treat the Neurodegeneration and Visual Failure in Neuronal Ceroid Lipofuscinoses. Advances in Experimental Medicine and Biology, 2018, 1074, 91-99.	1.6	14
24	<scp>CLN</scp>8 disease caused by large genomic deletions. Molecular Genetics & Genomic Medicine, 2017, 5, 85-91.	1.2	9
25	NCLs and ER: A stressful relationship. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2017, 1863, 1273-1281.	3.8	20
26	The value of a comprehensive natural history in late infantile <scp>CLN</scp>5 disease. Developmental Medicine and Child Neurology, 2017, 59, 777-778.	2.1	1
27	The Chihuahua dog: A new animal model for neuronal ceroid lipofuscinosis CLN7 disease?. Journal of Neuroscience Research, 2016, 94, 339-347.	2.9	26
28	Mutation of TBCK causes a rare recessive developmental disorder. Neurology: Genetics, 2016, 2, e76.	1.9	19
29	Neuronal ceroid lipofuscinoses. Epileptic Disorders, 2016, 18, 73-88.	1.3	111
30	Diagnosis of neuronal ceroid lipofuscinosis type 2 (CLN2 disease): Expert recommendations for early detection and laboratory diagnosis. Molecular Genetics and Metabolism, 2016, 119, 160-167.	1.1	70
31	Diagnosis and misdiagnosis of adult neuronal ceroid lipofuscinosis (Kufs disease). Neurology, 2016, 87, 579-584.	1.1	28
32	Genetics of the neuronal ceroid lipofuscinoses (Batten disease). Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2015, 1852, 2237-2241.	3.8	253
33	Guidelines for incorporating scientific knowledge and practice on rare diseases into higher education: neuronal ceroid lipofuscinoses as a model disorder. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2015, 1852, 2316-2323.	3.8	13
34	Future perspectives: Moving towards NCL treatments. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2015, 1852, 2336-2338.	3.8	7
35	The Neuronal Ceroid-Lipofuscinoses (Batten Disease). , 2015, , 793-808.		28
36	A central role for TOR signalling in a yeast model for juvenile CLN3 disease. Microbial Cell, 2015, 2, 466-480.	3.2	13

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37	Cathepsin D deficiency causes juvenile-onset ataxia and distinctive muscle pathology. Neurology, 2014, 83, 1873-1875.	1.1	33
38	Development of new treatments for Batten disease. Lancet Neurology, The, 2014, 13, 749-751.	10.2	6
39	Novel CLN3 mutation causing autophagic vacuolar myopathy. Neurology, 2014, 82, 2072-2076.	1.1	37
40	CLN6 disease caused by the same mutation originating in Pakistan has varying pathology. European Journal of Paediatric Neurology, 2013, 17, 657-660.	1.6	17
41	Genetic basis and phenotypic correlations of the neuronal ceroid lipofusinoses. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2013, 1832, 1827-1830.	3.8	109
42	Cathepsin F mutations cause Type B Kufs disease, an adult-onset neuronal ceroid lipofuscinosis. Human Molecular Genetics, 2013, 22, 1417-1423.	2.9	105
43	Mutation of the parkinsonism gene ATP13A2 causes neuronal ceroid-lipofuscinosis. Human Molecular Genetics, 2012, 21, 2646-2650.	2.9	231
44	New nomenclature and classification scheme for the neuronal ceroid lipofuscinoses. Neurology, 2012, 79, 183-191.	1.1	178
45	Strikingly Different Clinicopathological Phenotypes Determined by Progranulin-Mutation Dosage. American Journal of Human Genetics, 2012, 90, 1102-1107.	6.2	414
46	Update of the mutation spectrum and clinical correlations of over 360 mutations in eight genes that underlie the neuronal ceroid lipofuscinoses. Human Mutation, 2012, 33, 42-63.	2.5	273
47	Reply to Comment on â€œDeletion of btn1, an orthologue of CLN3, increases glycolysis and perturbs amino acid metabolism in the fission yeast model of Batten diseaseâ€• Molecular BioSystems, 2011, 7, 1349.	2.9	0
48	Involvement of the mitochondrial compartment in human NCL fibroblasts. Biochemical and Biophysical Research Communications, 2011, 416, 159-164.	2.1	18
49	Analysis of Potential Biomarkers and Modifier Genes Affecting the Clinical Course of CLN3 Disease. Molecular Medicine, 2011, 17, 1253-1261.	4.4	50
50	Kufs Disease, the Major Adult Form of Neuronal Ceroid Lipofuscinosis, Caused by Mutations in CLN6. American Journal of Human Genetics, 2011, 88, 566-573.	6.2	253
51	Mutations in DNAJC5, Encoding Cysteine-String Protein Alpha, Cause Autosomal-Dominant Adult-Onset Neuronal Ceroid Lipofuscinosis. American Journal of Human Genetics, 2011, 89, 241-252.	6.2	236
52	Neuronal Ceroid Lipofuscinosis in Qatar: Report of a Novel Mutation in Ceroid-Lipofuscinosis, Neuronal 5 in the Arab Population. Journal of Child Neurology, 2011, 26, 625-629.	1.4	9
53	Therapeutic Approaches to the Challenge of Neuronal Ceroid Lipofuscinoses. Current Pharmaceutical Biotechnology, 2011, 12, 867-883.	1.6	48
54	Pathogenic mutations cause rapid degradation of lysosomal storage disease-related membrane protein CLN6. Human Mutation, 2010, 31, E1163-E1174.	2.5	18

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55	Deletion of btn1, an orthologue of CLN3, increases glycolysis and perturbs amino acid metabolism in the fission yeast model of Batten disease. Molecular BioSystems, 2010, 6, 1093.	2.9	27
56	The Neuronal Ceroid Lipofuscinoses. , 2010, , 1235-1241.		8
57	The fission yeast model for the lysosomal storage disorder Batten disease predicts disease severity caused by mutations in CLN3. DMM Disease Models and Mechanisms, 2009, 2, 84-92.	2.4	14
58	Mutations in CLN7/MFSD8 are a common cause of variant late-infantile neuronal ceroid lipofuscinosis. Brain, 2009, 132, 810-819.	7.6	116
59	S. pombe btn1, the orthologue of the Batten disease geneCLN3, is required for vacuole protein sorting of Cpy1p and Golgi exit of Vps10p. Journal of Cell Science, 2009, 122, 1163-1173.	2.0	43
60	Mutations in MFSD8/CLN7 are a frequent cause of variant-late infantile neuronal ceroid lipofuscinosis. Human Mutation, 2009, 30, E530-E540.	2.5	59
61	Retention of lysosomal protein CLN5 in the endoplasmic reticulum causes neuronal ceroid lipofuscinosis in Asian Sibship. Human Mutation, 2009, 30, E651-E661.	2.5	48
62	Variant late infantile ceroid lipofuscinoses associated with novel mutations in CLN6. Biochemical and Biophysical Research Communications, 2009, 379, 892-897.	2.1	45
63	A 30-year Follow-Up of a Neuronal Ceroid Lipofuscinosis Patient With Mutations in CLN3 and Protracted Disease Course. Pediatric Neurology, 2009, 40, 134-137.	2.1	15
64	btn1 Affects Endocytosis, Polarization of Sterolâ€Rich Membrane Domains and Polarized Growth in Schizosaccharomyces pombe. Traffic, 2008, 9, 936-950.	2.7	40
65	The transmembrane topology of Batten disease protein CLN3 determined by consensus computational prediction constrained by experimental data. FEBS Letters, 2008, 582, 1019-1024.	2.8	38
66	btn1affects cytokinesis and cell-wall deposition by independent mechanisms, one of which is linked to dysregulation of vacuole pH. Journal of Cell Science, 2008, 121, 2860-2870.	2.0	29
67	Adult neuronal ceroid lipofuscinosis caused by deficiency in palmitoyl protein thioesterase 1. Neurology, 2007, 68, 387-388.	1.1	45
68	A function retained by the common mutant CLN3 protein is responsible for the late onset of juvenile neuronal ceroid lipofuscinosis. Human Molecular Genetics, 2007, 17, 303-312.	2.9	68
69	Topology and endoplasmic reticulum retention signals of the lysosomal storage disease-related membrane protein CLN6. Molecular Membrane Biology, 2007, 24, 74-87.	2.0	44
70	Molecular genetics of the NCLs â€" status and perspectives. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2006, 1762, 857-864.	3.8	69
71	Characterizing pathogenic processes in Batten disease: Use of small eukaryotic model systems. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2006, 1762, 906-919.	3.8	26
72	Homogeneous PCR nucleobase quenching assays to detect four mutations that cause neuronal ceroid lipofuscinosis: T75P and R151X in CLN1, and IVS5-1G>C and R208X in CLN2. Journal of Neuroscience Methods, 2006, 157, 124-131.	2.5	4

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73	Neuronal ceroid lipofuscinoses (NCL). European Journal of Paediatric Neurology, 2006, 10, 255-257.	1.6	17
74	Murine Cathepsin F Deficiency Causes Neuronal Lipofuscinosis and Late-Onset Neurological Disease. Molecular and Cellular Biology, 2006, 26, 2309-2316.	2.3	72
75	Lysosomal storage disease upon disruption of the neuronal chloride transport protein ClC-6. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103, 13854-13859.	7.1	166
76	Identification and characterization ofCaenorhabditis elegans palmitoyl protein thioesterase1. Journal of Neuroscience Research, 2005, 79, 836-848.	2.9	20
77	Correlations between genotype, ultrastructural morphology and clinical phenotype in the neuronal ceroid lipofuscinoses. Neurogenetics, 2005, 6, 107-126.	1.4	261
78	btn1, the Schizosaccharomyces pombe homologue of the human Batten disease gene CLN3, regulates vacuole homeostasis. Journal of Cell Science, 2005, 118, 5525-5536.	2.0	70
79	Pirkko Santavuori (1933-2004). Journal of Child Neurology, 2004, 19, 465-470.	1.4	1
80	The Genetic Spectrum of Human Neuronal Ceroidâ€lipofuscinoses. Brain Pathology, 2004, 14, 70-76.	4.1	90
81	Neuronal ceroid lipofuscinoses (NCL). European Journal of Paediatric Neurology, 2004, 8, 101-103.	1.6	4
82	Variant late infantile neuronal ceroid lipofuscinosis in a subset of Turkish patients is allelic to Northern epilepsy. Human Mutation, 2004, 23, 300-305.	2.5	80
83	CLN6, which is associated with a lysosomal storage disease, is an endoplasmic reticulum protein. Experimental Cell Research, 2004, 298, 399-406.	2.6	101
84	Spectrum ofCLN6mutations in variant late infantile neuronal ceroid lipofuscinosis. Human Mutation, 2003, 22, 35-42.	2.5	73
85	The Gene Mutated in Variant Late-Infantile Neuronal Ceroid Lipofuscinosis (CLN6) and in nclf Mutant Mice Encodes a Novel Predicted Transmembrane Protein. American Journal of Human Genetics, 2002, 70, 537-542.	6.2	171
86	Gene table: Neuronal ceroid lipofuscinoses. European Journal of Paediatric Neurology, 2002, 6, 129-130.	1.6	4
87	Neurodegenerative disease: the neuronal ceroid lipofuscinoses (Batten disease). Current Opinion in Neurology, 2001, 14, 795-803.	3.6	43
88	Identification of a Transactivation Motif in the CLN3 Protein. IUBMB Life, 2001, 51, 295-298.	3.4	2
89	New mutations in the neuronal ceroid lipofuscinosisgenes. European Journal of Paediatric Neurology, 2001, 5, 7-10.	1.6	24
90	Turkish variant late infantile neuronal ceroid lipofuscinosis (CLN7) may be allelic to CLN8. European Journal of Paediatric Neurology, 2001, 5, 21-27.	1.6	38

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91	Analysis of candidate genes in the CLN6 critical regionusing in silico cloning. European Journal of Paediatric Neurology, 2001, 5, 29-31.	1.6	2
92	Analysis of CLN3-protein interactions using the yeasttwo-hybrid system. European Journal of Paediatric Neurology, 2001, 5, 89-93.	1.6	2
93	Genomic structure of three CLN3-like genes in Caenorhabditis elegans. European Journal of Paediatric Neurology, 2001, 5, 121-125.	1.6	6
94	Why and how to assess the aetiological diagnosis of children with intellectual disability/mental retardation and other neurodevelopmental disorders: description of the Finnish approach. European Journal of Paediatric Neurology, 2001, 5, 7-13.	1.6	29
95	Neurodevelopmental risks in twin-to-twin transfusion syndrome: preliminary findings. European Journal of Paediatric Neurology, 2001, 5, 21-27.	1.6	129
96	Neuronal ceroid lipofuscinoses. European Journal of Paediatric Neurology, 2001, 5, 211-212.	1.6	10
97	Full-field ERG in patients with Batten/Spielmeyer-Vogt disease caused by mutations in the CLN3 gene. Ophthalmic Genetics, 2000, 21, 69-77.	1.2	26
98	Full-field ERG in patients with Batten/Spielmeyer-Vogt disease caused by mutations in the CLN3 gene. Ophthalmic Genetics, 2000, 21, 69-77.	1.2	9
99	The neuronal ceroid lipofuscinoses in human EPMR and mnd mutant mice are associated with mutations in CLN8. Nature Genetics, 1999, 23, 233-236.	21.4	277
100	Molecular Genetics of the Neuronal Ceroid Lipofuscinoses. Epilepsia, 1999, 40, 29-32.	5.1	38
101	Neuronal ceroid lipofuscinoses. European Journal of Paediatric Neurology, 1999, 3, 43-44.	1.6	9
102	Molecular basis of the neuronal ceroid lipofuscinoses: Mutations inCLN1,CLN2,CLN3, andCLN5. Human Mutation, 1999, 14, 199-215.	2.5	54
103	Batten's disease: eight genes and still counting?. Lancet, The, 1999, 354, 443-445.	13.7	60
104	A Murine Model for Juvenile NCL: Gene Targeting of MouseCln3. Molecular Genetics and Metabolism, 1999, 66, 309-313.	1.1	31
105	The Molecular Basis of GROD-Storing Neuronal Ceroid Lipofuscinoses in Scotland. Molecular Genetics and Metabolism, 1999, 66, 245-247.	1.1	10
106	Molecular basis of the neuronal ceroid lipofuscinoses: Mutations in CLN1, CLN2, CLN3, and CLN5. Human Mutation, 1999, 14, 199.	2.5	4
107	Elucidation of the exon-intron structure and size of the human protein kinase C beta gene (PRKCB). Human Genetics, 1998, 103, 483-487.	3.8	6
108	Batten Disease: Four Genes and Still Counting. Neurobiology of Disease, 1998, 5, 287-303.	4.4	31

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109	Sharing of PPT mutations between distinct clinical forms of neuronal ceroid lipofuscinoses in patients from Scotland.. Journal of Medical Genetics, 1998, 35, 790-790.	3.2	16
110	Mutations in the palmitoyl-protein thioesterase gene (PPT; CLN1) causing juvenile neuronal ceroid lipofuscinosis with granular osmiophilic deposits [published erratum appears in Hum Mol Genet 1998 Apr;7(4):765]. Human Molecular Genetics, 1998, 7, 291-297.	2.9	122
111	Epitope Mapping of Antibodies Recognising the N-Terminal Domain of Simian Virus Large Tumour Antigen. Intervirology, 1998, 41, 10-16.	2.8	14
112	Genetic Linkage Analysis of a Variant of Juvenile Onset Neuronal Ceroid Lipofuscinosis with Granular Osmiophilic Deposits. Neuropediatrics, 1997, 28, 21-22.	0.6	15
113	Structure of the CLN3 Gene and Predicted Structure, Location and Function of CLN3 Protein. Neuropediatrics, 1997, 28, 12-14.	0.6	18
114	Strategy for Mutation Detection in CLN3: Characterisation of Two Finnish Mutations. Neuropediatrics, 1997, 28, 15-17.	0.6	12
115	Genomic Structure and Complete Nucleotide Sequence of the Batten Disease Gene,CLN3. Genomics, 1997, 40, 346-350.	2.9	47
116	Spectrum of Mutations in the Batten Disease Gene, CLN3. American Journal of Human Genetics, 1997, 61, 310-316.	6.2	181
117	Prenatal diagnosis of Batten's disease. Lancet, The, 1996, 347, 1014-1015.	13.7	40
118	A model for Batten disease protein CLN3: Functional implications from homology and mutations. FEBS Letters, 1996, 399, 75-77.	2.8	71
119	Recent advances in the molecular genetics of the neuronal ceroid lipofuscinoses. Journal of Inherited Metabolic Disease, 1996, 19, 269-274.	3.6	10
120	Rapid diagnostic test for the major mutation underlying Batten disease.. Journal of Medical Genetics, 1996, 33, 1041-1042.	3.2	37
121	Refined localization of the Batten disease gene (CLN3) by haplotype and linkage disequilibrium mapping to D16S288-D16S383 and exclusion from this region of a variant form of Batten disease with granular osmiophilic deposits. American Journal of Medical Genetics Part A, 1995, 57, 312-315.	2.4	16
122	Physical map of the region containing the gene for Batten disease (CLN3). American Journal of Medical Genetics Part A, 1995, 57, 316-319.	2.4	10
123	Analysis of Batten disease candidate genesSTP andSTM. American Journal of Medical Genetics Part A, 1995, 57, 324-326.	2.4	2
124	Phenol sulfotransferases: Candidate genes for Batten disease. American Journal of Medical Genetics Part A, 1995, 57, 327-332.	2.4	4
125	YAC and Cosmid Contigs Spanning the Batten Disease (CLN3) Region at 16p12.1â€“p11.2. Genomics, 1995, 29, 478-489.	2.9	8
126	A multiple interval physical map of the pericentromeric region of human chromosome 10. Human Genetics, 1994, 93, 313-318.	3.8	7

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127	Epitope mapping. Molecular Biotechnology, 1994, 1, 277-287.	2.4	4
128	Mapping of Two Phenol Sulfotransferase Genes, STP and STM, to 16p: Candidate Genes for Batten Disease. Biochemical and Biophysical Research Communications, 1994, 205, 482-489.	2.1	39
129	Genomic Organization and DNA Sequence of the Human Catecholamine-Sulfating Phenol Sulfotransferase Gene (STM). Biochemical and Biophysical Research Communications, 1994, 205, 1325-1332.	2.1	25
130	Genetic Mapping of the Batten Disease Locus (CLN3) to the Interval D16S288-D16S383 by Analysis of Haplotypes and Allelic Association. Genomics, 1994, 22, 465-468.	2.9	33
131	Assignment of Fifty-Four Cosmid Clones to Five Regions of Chromosome 10. Genomics, 1993, 15, 457-458.	2.9	5
132	Assignment of the Human Pulmonary Surfactant Protein D Gene (SFTP4) to 10q22-q23 Close to the Surfactant Protein A Gene Cluster. Genomics, 1993, 17, 294-298.	2.9	60
133	Germ-line mutations of the RET proto-oncogene in multiple endocrine neoplasia type 2A. Nature, 1993, 363, 458-460.	27.8	1,886
134	Genetic linkage studies map the multiple endocrine neoplasia type 2 loci to a small interval on chromosome 10q11.2. Human Molecular Genetics, 1993, 2, 241-246.	2.9	100
135	Localisation of the gene for multiple endocrine neoplasia type 2A to a 480 kb region in chromosome band 10q11.2. Human Molecular Genetics, 1993, 2, 247-252.	2.9	94
136	Characterisation of a boundary between satellite III and aiphoid sequences on human chromosome 10. Nucleic Acids Research, 1992, 20, 4781-4787.	14.5	38
137	Using the polymerase chain reaction to modify expression plasmids for epitope mapping. Nucleic Acids Research, 1989, 17, 3319-3319.	14.5	29
138	pSEMCatR1: procaryotic-eucaryotic shuttle vector compatible with pUR and Î»gt11 expression systems. Nucleic Acids Research, 1987, 15, 9090-9090.	14.5	2
139	Use of simian virus 40 large T-beta-galactosidase fusion proteins in an immunochemical analysis of simian virus 40 large T antigen. Journal of Virology, 1985, 54, 703-710.	3.4	30
140	Cerebrospinal fluid neurofilament light chain levels in CLN2 disease patients treated with enzyme replacement therapy normalise after two years on treatment. F1000Research, 0, 10, 614.	1.6	2