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	Germ-line mutations of the RET proto-oncogene in multiple endocrine neoplasia type 2A. Nature, 1993, 363, 458-460.	27.8	1,886
2	Strikingly Different Clinicopathological Phenotypes Determined by Progranulin-Mutation Dosage. American Journal of Human Genetics, 2012, 90, 1102-1107.	6.2	414
3	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
4	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
5	Correlations between genotype, ultrastructural morphology and clinical phenotype in the neuronal ceroid lipofuscinoses. Neurogenetics, 2005, 6, 107-126.	1.4	261
6	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
7	Genetics of the neuronal ceroid lipofuscinoses (Batten disease). Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2015, 1852, 2237-2241.	3.8	253
8	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
9	Mutation of the parkinsonism gene ATP13A2 causes neuronal ceroid-lipofuscinosis. Human Molecular Genetics, 2012, 21, 2646-2650.	2.9	231
10	Spectrum of Mutations in the Batten Disease Gene, CLN3. American Journal of Human Genetics, 1997, 61, 310-316.	6.2	181
11	New nomenclature and classification scheme for the neuronal ceroid lipofuscinoses. Neurology, 2012, 79, 183-191.	1.1	178
12	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
13	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
14	Neurodevelopmental risks in twin-to-twin transfusion syndrome: preliminary findings. European Journal of Paediatric Neurology, 2001, 5, 21-27.	1.6	129
15	Clinical challenges and future therapeutic approaches for neuronal ceroid lipofuscinosis. Lancet Neurology, The, 2019, 18, 107-116.	10.2	128
16	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
17	Mutations in CLN7/MFSD8 are a common cause of variant late-infantile neuronal ceroid lipofuscinosis. Brain, 2009, 132, 810-819.	7.6	116
18	Neuronal ceroid lipofuscinoses. Epileptic Disorders, 2016, 18, 73-88.	1.3	111

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19	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
20	Cathepsin F mutations cause Type B Kufs disease, an adult-onset neuronal ceroid lipofuscinosis. Human Molecular Genetics, 2013, 22, 1417-1423.	2.9	105
21	CLN6, which is associated with a lysosomal storage disease, is an endoplasmic reticulum protein. Experimental Cell Research, 2004, 298, 399-406.	2.6	101
22	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
23	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
24	The Genetic Spectrum of Human Neuronal Ceroidâ€lipofuscinoses. Brain Pathology, 2004, 14, 70-76.	4.1	90
25	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
26	Spectrum ofCLN6mutations in variant late infantile neuronal ceroid lipofuscinosis. Human Mutation, 2003, 22, 35-42.	2.5	73
27	Murine Cathepsin F Deficiency Causes Neuronal Lipofuscinosis and Late-Onset Neurological Disease. Molecular and Cellular Biology, 2006, 26, 2309-2316.	2.3	72
28	A model for Batten disease protein CLN3: Functional implications from homology and mutations. FEBS Letters, 1996, 399, 75-77.	2.8	71
29	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
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	Molecular genetics of the NCLs â€" status and perspectives. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2006, 1762, 857-864.	3.8	69
32	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
33	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
34	Batten's disease: eight genes and still counting?. Lancet, The, 1999, 354, 443-445.	13.7	60
35	Mutations in MFSD8/CLN7 are a frequent cause of variant-late infantile neuronal ceroid lipofuscinosis. Human Mutation, 2009, 30, E530-E540.	2.5	59
36	Molecular basis of the neuronal ceroid lipofuscinoses: Mutations inCLN1,CLN2,CLN3, andCLN5. Human Mutation, 1999, 14, 199-215.	2.5	54

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37	Analysis of Potential Biomarkers and Modifier Genes Affecting the Clinical Course of CLN3 Disease. Molecular Medicine, 2011, 17, 1253-1261.	4.4	50
38	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
39	Therapeutic Approaches to the Challenge of Neuronal Ceroid Lipofuscinoses. Current Pharmaceutical Biotechnology, 2011, 12, 867-883.	1.6	48
40	Genomic Structure and Complete Nucleotide Sequence of the Batten Disease Gene,CLN3. Genomics, 1997, 40, 346-350.	2.9	47
41	Loss of CLN7 results in depletion of soluble lysosomal proteins and impaired mTOR reactivation. Human Molecular Genetics, 2018, 27, 1711-1722.	2.9	47
42	Mutation update: Review of TPP1 gene variants associated with neuronal ceroid lipofuscinosis CLN2 disease. Human Mutation, 2019, 40, 1924-1938.	2.5	46
43	Adult neuronal ceroid lipofuscinosis caused by deficiency in palmitoyl protein thioesterase 1. Neurology, 2007, 68, 387-388.	1.1	45
44	Variant late infantile ceroid lipofuscinoses associated with novel mutations in CLN6. Biochemical and Biophysical Research Communications, 2009, 379, 892-897.	2.1	45
45	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
46	Neurodegenerative disease: the neuronal ceroid lipofuscinoses (Batten disease). Current Opinion in Neurology, 2001, 14, 795-803.	3.6	43
47	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
48	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
49	Prenatal diagnosis of Batten's disease. Lancet, The, 1996, 347, 1014-1015.	13.7	40
50	btn1 Affects Endocytosis, Polarization of Sterolâ€Rich Membrane Domains and Polarized Growth in Schizosaccharomyces pombe. Traffic, 2008, 9, 936-950.	2.7	40
51	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
52	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
53	Characterisation of a boundary between satellite III and aiphoid sequences on human chromosome 10. Nucleic Acids Research, 1992, 20, 4781-4787.	14.5	38
54	Molecular Genetics of the Neuronal Ceroid Lipofuscinoses. Epilepsia, 1999, 40, 29-32.	5.1	38

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55	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
56	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
57	Rapid diagnostic test for the major mutation underlying Batten disease.. Journal of Medical Genetics, 1996, 33, 1041-1042.	3.2	37
58	Novel CLN3 mutation causing autophagic vacuolar myopathy. Neurology, 2014, 82, 2072-2076.	1.1	37
59	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
60	Cathepsin D deficiency causes juvenile-onset ataxia and distinctive muscle pathology. Neurology, 2014, 83, 1873-1875.	1.1	33
61	Batten Disease: Four Genes and Still Counting. Neurobiology of Disease, 1998, 5, 287-303.	4.4	31
62	A Murine Model for Juvenile NCL: Gene Targeting of MouseCln3. Molecular Genetics and Metabolism, 1999, 66, 309-313.	1.1	31
63	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
64	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
65	Using the polymerase chain reaction to modify expression plasmids for epitope mapping. Nucleic Acids Research, 1989, 17, 3319-3319.	14.5	29
66	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
67	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
68	The Neuronal Ceroid-Lipofuscinoses (Batten Disease). , 2015, , 793-808.		28
69	Diagnosis and misdiagnosis of adult neuronal ceroid lipofuscinosis (Kufs disease). Neurology, 2016, 87, 579-584.	1.1	28
70	Kufs disease due to mutation ofCLN6: clinical, pathological and molecular genetic features. Brain, 2019, 142, 59-69.	7.6	28
71	Repurposing of tamoxifen ameliorates CLN3 and CLN7 disease phenotype. EMBO Molecular Medicine, 2021, 13, e13742.	6.9	28
72	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

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73	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
74	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
75	The Chihuahua dog: A new animal model for neuronal ceroid lipofuscinosis CLN7 disease?. Journal of Neuroscience Research, 2016, 94, 339-347.	2.9	26
76	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
77	New mutations in the neuronal ceroid lipofuscinosisgenes. European Journal of Paediatric Neurology, 2001, 5, 7-10.	1.6	24
78	The Genetic Basis of Phenotypic Heterogeneity in the Neuronal Ceroid Lipofuscinoses. Frontiers in Neurology, 2021, 12, 754045.	2.4	24
79	Neonatal brain-directed gene therapy rescues a mouse model of neurodegenerative CLN6 Batten disease. Human Molecular Genetics, 2019, 28, 3867-3879.	2.9	21
80	Identification and characterization ofCaenorhabditis elegans palmitoyl protein thioesterase1. Journal of Neuroscience Research, 2005, 79, 836-848.	2.9	20
81	NCLs and ER: A stressful relationship. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2017, 1863, 1273-1281.	3.8	20
82	Mutation of TBCK causes a rare recessive developmental disorder. Neurology: Genetics, 2016, 2, e76.	1.9	19
83	Structure of the CLN3 Gene and Predicted Structure, Location and Function of CLN3 Protein. Neuropediatrics, 1997, 28, 12-14.	0.6	18
84	Pathogenic mutations cause rapid degradation of lysosomal storage disease-related membrane protein CLN6. Human Mutation, 2010, 31, E1163-E1174.	2.5	18
85	Involvement of the mitochondrial compartment in human NCL fibroblasts. Biochemical and Biophysical Research Communications, 2011, 416, 159-164.	2.1	18
86	Neuronal ceroid lipofuscinoses (NCL). European Journal of Paediatric Neurology, 2006, 10, 255-257.	1.6	17
87	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
88	Guidelines on the diagnosis, clinical assessments, treatment and management for CLN2 disease patients. Orphanet Journal of Rare Diseases, 2021, 16, 185.	2.7	17
89	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
90	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

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91	Genetic Linkage Analysis of a Variant of Juvenile Onset Neuronal Ceroid Lipofuscinosis with Granular Osmiophilic Deposits. Neuropediatrics, 1997, 28, 21-22.	0.6	15
92	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
93	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
94	Epitope Mapping of Antibodies Recognising the N-Terminal Domain of Simian Virus Large Tumour Antigen. Intervirology, 1998, 41, 10-16.	2.8	14
95	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
96	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
97	Aberrant upregulation of the glycolytic enzyme PFKFB3 in CLN7 neuronal ceroid lipofuscinosis. Nature Communications, 2022, 13, 536.	12.8	14
98	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
99	A central role for TOR signalling in a yeast model for juvenile CLN3 disease. Microbial Cell, 2015, 2, 466-480.	3.2	13
100	Strategy for Mutation Detection in CLN3: Characterisation of Two Finnish Mutations. Neuropediatrics, 1997, 28, 15-17.	0.6	12
101	Future perspectives: What lies ahead for Neuronal Ceroid Lipofuscinosis research?. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2020, 1866, 165681.	3.8	12
102	Urine proteomics analysis of patients with neuronal ceroid lipofuscinoses. IScience, 2021, 24, 102020.	4.1	12
103	Experimental gene therapies for the NCLs. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2020, 1866, 165772.	3.8	11
104	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
105	Recent advances in the molecular genetics of the neuronal ceroid lipofuscinoses. Journal of Inherited Metabolic Disease, 1996, 19, 269-274.	3.6	10
106	The Molecular Basis of GROD-Storing Neuronal Ceroid Lipofuscinoses in Scotland. Molecular Genetics and Metabolism, 1999, 66, 245-247.	1.1	10
107	Neuronal ceroid lipofuscinoses. European Journal of Paediatric Neurology, 2001, 5, 211-212.	1.6	10
108	Neuronal ceroid lipofuscinoses. European Journal of Paediatric Neurology, 1999, 3, 43-44.	1.6	9

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109	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
110	<scp>CLN</scp>8 disease caused by large genomic deletions. Molecular Genetics & Genomic Medicine, 2017, 5, 85-91.	1.2	9
111	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
112	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
113	YAC and Cosmid Contigs Spanning the Batten Disease (CLN3) Region at 16p12.1â€“p11.2. Genomics, 1995, 29, 478-489.	2.9	8
114	The Neuronal Ceroid Lipofuscinoses. , 2010, , 1235-1241.		8
115	A multiple interval physical map of the pericentromeric region of human chromosome 10. Human Genetics, 1994, 93, 313-318.	3.8	7
116	Future perspectives: Moving towards NCL treatments. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2015, 1852, 2336-2338.	3.8	7
117	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
118	Genomic structure of three CLN3-like genes in Caenorhabditis elegans. European Journal of Paediatric Neurology, 2001, 5, 121-125.	1.6	6
119	Development of new treatments for Batten disease. Lancet Neurology, The, 2014, 13, 749-751.	10.2	6
120	Assignment of Fifty-Four Cosmid Clones to Five Regions of Chromosome 10. Genomics, 1993, 15, 457-458.	2.9	5
121	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
122	Epitope mapping. Molecular Biotechnology, 1994, 1, 277-287.	2.4	4
123	Phenol sulfotransferases: Candidate genes for Batten disease. American Journal of Medical Genetics Part A, 1995, 57, 327-332.	2.4	4
124	Gene table: Neuronal ceroid lipofuscinoses. European Journal of Paediatric Neurology, 2002, 6, 129-130.	1.6	4
125	Neuronal ceroid lipofuscinoses (NCL). European Journal of Paediatric Neurology, 2004, 8, 101-103.	1.6	4
126	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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127	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
128	Molecular basis of the neuronal ceroid lipofuscinoses: Mutations in CLN1, CLN2, CLN3, and CLN5. Human Mutation, 1999, 14, 199.	2.5	4
129	The neuronal ceroid-lipofuscinoses (Batten disease). , 2020, , 53-71.		3
130	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
131	pSEMCatR1: procaryotic-eucaryotic shuttle vector compatible with pUR and Î»gt11 expression systems. Nucleic Acids Research, 1987, 15, 9090-9090.	14.5	2
132	Analysis of Batten disease candidate genesSTP andSTM. American Journal of Medical Genetics Part A, 1995, 57, 324-326.	2.4	2
133	Identification of a Transactivation Motif in the CLN3 Protein. IUBMB Life, 2001, 51, 295-298.	3.4	2
134	Analysis of candidate genes in the CLN6 critical regionusing in silico cloning. European Journal of Paediatric Neurology, 2001, 5, 29-31.	1.6	2
135	Analysis of CLN3-protein interactions using the yeasttwo-hybrid system. European Journal of Paediatric Neurology, 2001, 5, 89-93.	1.6	2
136	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
137	Pirkko Santavuori (1933-2004). Journal of Child Neurology, 2004, 19, 465-470.	1.4	1
138	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
139	Special edition: The NCLs/Batten disease. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2020, 1866, 165824.	3.8	1
140	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