David M Bedwell

List of Publications by Year
in descending order

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67	5,824	76322	102480
papers	citations	40 h-index	66 g-index

67	67	67	4007
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Aminoglycoside antibiotics restore CFTR function by overcoming premature stop mutations. Nature Medicine, 1996, 2, 467-469.	30.7	464
2	Aminoglycoside antibiotics mediate context-dependent suppression of termination codons in a mammalian translation system. Rna, 2000, 6, 1044-1055.	3.5	335
3	Suppression of a CFTR premature stop mutation in a bronchial epithelial cell line. Nature Medicine, 1997, 3, 1280-1284.	30.7	315
4	The Efficiency of Translation Termination is Determined by a Synergistic Interplay Between Upstream and Downstream Sequences inSaccharomyces cerevisiae. Journal of Molecular Biology, 1995, 251, 334-345.	4.2	289
5	Thespcrtbosomal protein operon ofEschenchia coli:sequence and cotranscriptlon of the rlbosomal protein genes and a protein export gene. Nucleic Acids Research, 1983, 11, 2599-2616.	14.5	286
6	Therapeutics Based on Stop Codon Readthrough. Annual Review of Genomics and Human Genetics, 2014, 15, 371-394.	6.2	247
7	Evidence that Systemic Gentamicin Suppresses Premature Stop Mutations in Patients with Cystic Fibrosis. American Journal of Respiratory and Critical Care Medicine, 2001, 163, 1683-1692.	5.6	238
8	PTC124 is an orally bioavailable compound that promotes suppression of the human CFTR -G542X nonsense allele in a CF mouse model. Proceedings of the National Academy of Sciences of the United States of America, 2008, 105, 2064-2069.	7.1	233
9	Ataluren stimulates ribosomal selection of near-cognate tRNAs to promote nonsense suppression. Proceedings of the National Academy of Sciences of the United States of America, 2016, 113, 12508-12513.	7.1	168
10	Aminoglycoside suppression of a premature stop mutation in a Cftrâ€"/â€" mouse carrying a human CFTR-G542X transgene. Journal of Molecular Medicine, 2002, 80, 595-604.	3.9	160
11	GTP Hydrolysis by eRF3 Facilitates Stop Codon Decoding during Eukaryotic Translation Termination. Molecular and Cellular Biology, 2004, 24, 7769-7778.	2.3	160
12	Leaky termination at premature stop codons antagonizes nonsense-mediated mRNA decay in S. cerevisiae. Rna, 2004, 10, 691-703.	3.5	153
13	Gentamicin-mediated suppression of Hurler syndrome stop mutations restores a low level of alpha-L-iduronidase activity and reduces lysosomal glycosaminoglycan accumulation. Human Molecular Genetics, 2001, 10, 291-299.	2.9	145
14	The vacuolar Ca2+/H+exchanger Vcx1p/Hum1p tightly controls cytosolic Ca2+levels inS. cerevisiae. FEBS Letters, 1999, 451, 132-136.	2.8	139
15	Characterization of Defects in Ion Transport and Tissue Development in Cystic Fibrosis Transmembrane Conductance Regulator (CFTR)-Knockout Rats. PLoS ONE, 2014, 9, e91253.	2.5	133
16	Synthetic Aminoglycosides Efficiently Suppress Cystic Fibrosis Transmembrane Conductance Regulator Nonsense Mutations and Are Enhanced by Ivacaftor. American Journal of Respiratory Cell and Molecular Biology, 2014, 50, 805-816.	2.9	131
17	Clinically relevant aminoglycosides can suppress disease-associated premature stop mutations in the IDUA and P53 cDNAs in a mammalian translation system. Journal of Molecular Medicine, 2002, 80, 367-376.	3.9	124
18	Discrimination Between Defects in Elongation Fidelity and Termination Efficiency Provides Mechanistic Insights into Translational Readthrough. Journal of Molecular Biology, 2005, 348, 801-815.	4.2	102

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19	Nucleotide sequence of the alpha ribosomal protein operon ofEscherichia coli. Nucleic Acids Research, 1985, 13, 3891-3902.	14.5	101
20	Suppression of premature termination codons as a therapeutic approach. Critical Reviews in Biochemistry and Molecular Biology, 2012, 47, 444-463.	5.2	89
21	Attenuation of Nonsense-Mediated mRNA Decay Enhances In Vivo Nonsense Suppression. PLoS ONE, 2013, 8, e60478.	2.5	89
22	Eukaryotic ribosomal RNA determinants of aminoglycoside resistance and their role in translational fidelity. Rna, 2007, 14, 148-157.	3.5	84
23	Suppression of nonsense mutations as a therapeutic approach to treat genetic diseases. Wiley Interdisciplinary Reviews RNA, 2011, 2, 837-852.	6.4	78
24	Discovery of Clinically Approved Agents That Promote Suppression of Cystic Fibrosis Transmembrane Conductance Regulator Nonsense Mutations. American Journal of Respiratory and Critical Care Medicine, 2016, 194, 1092-1103.	5.6	77
25	Identification of the amino acids inserted during suppression of CFTR nonsense mutations and determination of their functional consequences. Human Molecular Genetics, 2017, 26, 3116-3129.	2.9	69
26	Clinical doses of amikacin provide more effective suppression of the human CFTR-G542X stop mutation than gentamicin in a transgenic CF mouse model. Journal of Molecular Medicine, 2006, 84, 573-582.	3.9	68
27	Suppression of CFTR premature termination codons and rescue of CFTR protein and function by the synthetic aminoglycoside NB54. Journal of Molecular Medicine, 2011, 89, 1149-1161.	3.9	67
28	The designer aminoglycoside NB84 significantly reduces glycosaminoglycan accumulation associated with MPS I-H in the Idua-W392X mouse. Molecular Genetics and Metabolism, 2012, 105, 116-125.	1.1	67
29	The Golgi Apparatus Plays a Significant Role in the Maintenance of Ca2+ Homeostasis in the vps33Î”Vacuolar Biogenesis Mutant of Saccharomyces cerevisiae. Journal of Biological Chemistry, 1999, 274, 5939-5947.	3.4	66
30	Aminoglycosides as Potential Pharmacogenetic Agents in the Treatment of Haileyâ€“Hailey Disease. Journal of Investigative Dermatology, 2006, 126, 229-231.	0.7	65
31	Regulation of Î± operon gene expression in Escherichia coli. Journal of Molecular Biology, 1987, 196, 333-345.	4.2	63
32	A small molecule that induces translational readthrough of CFTR nonsense mutations by eRF1 depletion. Nature Communications, 2021, 12, 4358.	12.8	59
33	Extracellular Ca2+ sensing contributes to excess Ca2+ accumulation and vacuolar fragmentation in apmr1Î” mutant ofS. cerevisiae. Journal of Cell Science, 2003, 116, 1637-1646.	2.0	56
34	Distinct eRF3 Requirements Suggest Alternate eRF1 Conformations Mediate Peptide Release during Eukaryotic Translation Termination. Molecular Cell, 2008, 30, 599-609.	9.7	56
35	Characterization of an MPS I-H knock-in mouse that carries a nonsense mutation analogous to the human IDUA-W402X mutation. Molecular Genetics and Metabolism, 2010, 99, 62-71.	1.1	56
36	Loss of the Major Isoform of Phosphoglucomutase Results in Altered Calcium Homeostasis in Saccharomyces cerevisiae. Journal of Biological Chemistry, 2000, 275, 5431-5440.	3.4	55

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37	Tpa1p Is Part of an mRNP Complex That Influences Translation Termination, mRNA Deadenylation, and mRNA Turnover in Saccharomyces cerevisiae. Molecular and Cellular Biology, 2006, 26, 5237-5248.	2.3	53
38	Distinct Paths To Stop Codon Reassignment by the Variant-Code Organisms Tetrahymena and Euplotes. Molecular and Cellular Biology, 2006, 26, 438-447.	2.3	49
39	Long-term nonsense suppression therapy moderates MPS I-H disease progression. Molecular Genetics and Metabolism, 2014, 111, 374-381.	1.1	44
40	Feedback regulation of RNA polymerase subunit synthesis after the conditional overproduction of RNA polymerase in Escherichia coli. Molecular Genetics and Genomics, 1986, 204, 17-23.	2.4	42
41	Intracellular Glucose 1-Phosphate and Glucose 6-Phosphate Levels Modulate Ca2+ Homeostasis in Saccharomyces cerevisiae. Journal of Biological Chemistry, 2002, 277, 45751-45758.	3.4	39
42	Poly-l-aspartic Acid Enhances and Prolongs Gentamicin-mediated Suppression of the CFTR-G542X Mutation in a Cystic Fibrosis Mouse Model. Journal of Biological Chemistry, 2009, 284, 6885-6892.	3.4	38
43	Nonsense suppression activity of PTC124 (ataluren). Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, E64; author reply E65.	7.1	36
44	Hexose phosphorylation and the putative calcium channel component Mid1p are required for the hexose-induced transient elevation of cytosolic calcium response in Saccharomyces cerevisiae. Molecular Microbiology, 2002, 44, 1299-1308.	2.5	34
45	The posttranslational modification of phosphoglucomutase is regulated by galactose induction and glucose repression in Saccharomyces cerevisiae. Journal of Bacteriology, 1995, 177, 3087-3094.	2.2	31
46	Mutations within the first LSGGQ motif of Ste6p cause defects in a-factor transport and mating in Saccharomyces cerevisiae. Journal of Bacteriology, 1996, 178, 1712-1719.	2.2	30
47	Increased expression of ribosomal genes during inhibition of ribosome assembly in Escherichia coli. Journal of Molecular Biology, 1985, 184, 23-30.	4.2	29
48	Identification of eRF1 residues that play critical and complementary roles in stop codon recognition. Rna, 2012, 18, 1210-1221.	3.5	29
49	Both the autophagy and proteasomal pathways facilitate the Ubp3p-dependent depletion of a subset of translation and RNA turnover factors during nitrogen starvation in Saccharomyces cerevisiae. Rna, 2015, 21, 898-910.	3.5	27
50	Inhibition of phosphoglucomutase activity by lithium alters cellular calcium homeostasis and signaling in Saccharomyces cerevisiae. American Journal of Physiology - Cell Physiology, 2005, 289, C58-C67.	4.6	26
51	Mutations in the Yeast Hsp40 Chaperone Protein Ydj1 Cause Defects in Axl1 Biogenesis and Pro-a-factor Processing. Journal of Biological Chemistry, 1999, 274, 34396-34402.	3.4	25
52	Pharmacological Suppression of Premature Stop Mutations that Cause Genetic Diseases. Current Pharmacogenomics and Personalized Medicine: the International Journal for Expert Reviews in Pharmacogenomics, 2005, 3, 259-269.	0.3	25
53	Mice with missense and nonsense NF1 mutations display divergent phenotypes compared to NF1 patients. DMM Disease Models and Mechanisms, 2016, 9, 759-67.	2.4	23
54	The Amino Terminus of the F₁-ATPase Î²-Subunit Precursor Functions as an Intramolecular Chaperone To Facilitate Mitochondrial Protein Import. Molecular and Cellular Biology, 1997, 17, 7169-7177.	2.3	19

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55	Eukaryotic Release Factor 1 Phosphorylation by CK2 Protein Kinase Is Dynamic but Has Little Effect on the Efficiency of Translation Termination in Saccharomyces cerevisiae. Eukaryotic Cell, 2006, 5, 1378-1387.	3.4	19
56	The Ca2+ Homeostasis Defects in a pgm2Î" Strain of Saccharomyces cerevisiae Are Caused by Excessive Vacuolar Ca2+ Uptake Mediated by the Ca2+-ATPase Pmc1p. Journal of Biological Chemistry, 2004, 279, 38495-38502.	3.4	18
57	Characterization of a HindIII-generated DNA fragment carrying the glutamine synthetase gene of Salmonella typhimurium. Gene, 1980, 11, 227-237.	2.2	17
58	Overproduction of PDR3 Suppresses Mitochondrial Import Defects Associated with a TOM70 Null Mutation by Increasing the Expression of TOM72 in Saccharomyces cerevisiae. Molecular and Cellular Biology, 2001, 21, 7576-7586.	2.3	16
59	A Saccharomyces cerevisiae Mutant Unable To Convert Glucose to Glucose-6-Phosphate Accumulates Excessive Glucose in the Endoplasmic Reticulum due to Core Oligosaccharide Trimming. Eukaryotic Cell, 2003, 2, 534-541.	3.4	16
60	Mutation-Directed Therapeutics for Neurofibromatosis Type I. Molecular Therapy - Nucleic Acids, 2020, 20, 739-753.	5.1	16
61	Connection between stop codon reassignment and frequent use of shifty stop frameshifting. Rna, 2009, 15, 889-897.	3.5	14
62	Recoding Therapies for Genetic Diseases. Nucleic Acids and Molecular Biology, 2010, , 123-146.	0.2	7
63	Analysis of patientâ€specific NF1 variants leads to functional insights for Ras signaling that can impact personalized medicine. Human Mutation, 2022, 43, 30-41.	2.5	6
64	Marked repression of CFTR mRNA in the transgenic Cftrtm1kth mouse model. Journal of Cystic Fibrosis, 2014, 13, 351-352.	0.7	4
65	Reply to â€œNonstop treatment of cystic fibrosisâ€• Nature Medicine, 1996, 2, 608-609.	30.7	3
66	Finding sense in the context. ELife, 2020, 9, .	6.0	1
67	Targeted Therapeutics for Rare Disorders. , 2024, , 249-271.		1