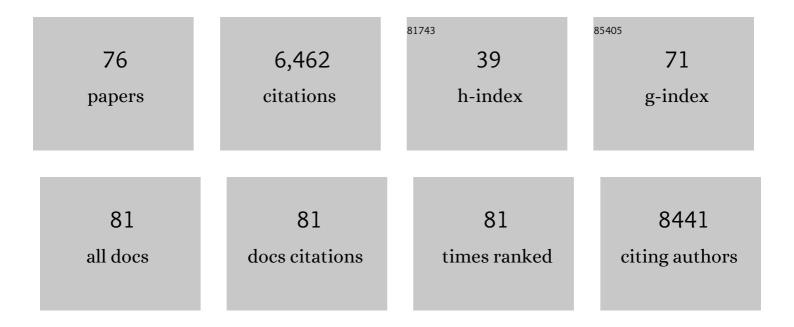
## Christopher D Putnam

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Disease-associated mutations in topoisomerase IlÎ <sup>2</sup> result in defective NK cells. Journal of Allergy and Clinical Immunology, 2022, 149, 2171-2176.e3.	1.5	7
2	Ligation of newly replicated DNA controls the timing of DNA mismatch repair. Current Biology, 2021, 31, 1268-1276.e6.	1.8	19
3	Immunodeficiency and bone marrow failure with mosaic and germline TLR8 gain of function. Blood, 2021, 137, 2450-2462.	0.6	47
4	Rad27 and Exo1 function in different excision pathways for mismatch repair in Saccharomyces cerevisiae. Nature Communications, 2021, 12, 5568.	5.8	9
5	Strand discrimination in DNA mismatch repair. DNA Repair, 2021, 105, 103161.	1.3	31
6	MutS sliding clamps on an uncertain track to DNA mismatch repair. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 20351-20353.	3.3	3
7	FEN1 endonuclease as a therapeutic target for human cancers with defects in homologous recombination. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 19415-19424.	3.3	53
8	Mechanisms underlying genome instability mediated by formation of foldback inversions in Saccharomyces cerevisiae. ELife, 2020, 9, .	2.8	10
9	Essential Saccharomyces cerevisiae genome instability suppressing genes identify potential human tumor suppressors. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 17377-17382.	3.3	8
10	Mutations in topoisomerase IIβ result in a B cell immunodeficiency. Nature Communications, 2019, 10, 3644.	5.8	37
11	Alternative splicing regulates stochastic NLRP3 activity. Nature Communications, 2019, 10, 3238.	5.8	44
12	Guidelines for DNA recombination and repair studies: Cellular assays of DNA repair pathways. Microbial Cell, 2019, 6, 1-64.	1.4	47
13	<i>EPCAM</i> mutation update: Variants associated with congenital tufting enteropathy and Lynch syndrome. Human Mutation, 2019, 40, 142-161.	1.1	51
14	Analyzing Genome Rearrangements in Saccharomyces cerevisiae. Methods in Molecular Biology, 2018, 1672, 43-61.	0.4	9
15	The properties of Msh2–Msh6 ATP binding mutants suggest a signal amplification mechanism in DNA mismatch repair. Journal of Biological Chemistry, 2018, 293, 18055-18070.	1.6	24
16	DNA Mismatch Repair: Mechanisms and Cancer Genetics. , 2018, , .		1
17	The Swr1 chromatin-remodeling complex prevents genome instability induced by replication fork progression defects. Nature Communications, 2018, 9, 3680.	5.8	17
18	Identification of Exo1-Msh2 interaction motifs in DNA mismatch repair and new Msh2-binding partners. Nature Structural and Molecular Biology, 2018, 25, 650-659.	3.6	35

CHRISTOPHER D PUTNAM

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19	Cdc73 suppresses genome instability by mediating telomere homeostasis. PLoS Genetics, 2018, 14, e1007170.	1.5	15
20	SUMO E3 ligase Mms21 prevents spontaneous DNA damage induced genome rearrangements. PLoS Genetics, 2018, 14, e1007250.	1.5	16
21	Pathways and Mechanisms that Prevent Genome Instability in <i>Saccharomyces cerevisiae</i> . Genetics, 2017, 206, 1187-1225.	1.2	49
22	Guinier peak analysis for visual and automated inspection of small-angle X-ray scattering data. Journal of Applied Crystallography, 2016, 49, 1412-1419.	1.9	56
23	A genetic network that suppresses genome rearrangements in Saccharomyces cerevisiae and contains defects in cancers. Nature Communications, 2016, 7, 11256.	5.8	36
24	Evolution of the methyl directed mismatch repair system in Escherichia coli. DNA Repair, 2016, 38, 32-41.	1.3	65
25	Exonuclease 1-dependent and independent mismatch repair. DNA Repair, 2015, 32, 24-32.	1.3	115
26	DNA Repair Pathway Selection Caused by Defects in TEL1, SAE2, and De Novo Telomere Addition Generates Specific Chromosomal Rearrangement Signatures. PLoS Genetics, 2014, 10, e1004277.	1.5	20
27	Mlh2 Is an Accessory Factor for DNA Mismatch Repair in Saccharomyces cerevisiae. PLoS Genetics, 2014, 10, e1004327.	1.5	36
28	A <i>Saccharomyces cerevisiae</i> RNase H2 Interaction Network Functions To Suppress Genome Instability. Molecular and Cellular Biology, 2014, 34, 1521-1534.	1.1	46
29	PCNA and Msh2-Msh6 Activate an Mlh1-Pms1 Endonuclease Pathway Required for Exo1-Independent Mismatch Repair. Molecular Cell, 2014, 55, 291-304.	4.5	89
30	Template homology determines the genetics and mechanisms of gross chromosomal rearrangements in S. cerevisiae (736.11). FASEB Journal, 2014, 28, 736.11.	0.2	0
31	Mutations of Complement Factor I and Potential Mechanisms of Neuroinflammation in Acute Hemorrhagic Leukoencephalitis. Journal of Clinical Immunology, 2013, 33, 162-171.	2.0	34
32	Distinct SUMO Ligases Cooperate with Esc2 and Slx5 to Suppress Duplication-Mediated Genome Rearrangements. PLoS Genetics, 2013, 9, e1003670.	1.5	68
33	Dominant Mutations in S. cerevisiae PMS1 Identify the Mlh1-Pms1 Endonuclease Active Site and an Exonuclease 1-Independent Mismatch Repair Pathway. PLoS Genetics, 2013, 9, e1003869.	1.5	52
34	DNA conformations in mismatch repair probed in solution by X-ray scattering from gold nanocrystals. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, 17308-17313.	3.3	53
35	Engineered Disulfide-forming Amino Acid Substitutions Interfere with a Conformational Change in the Mismatch Recognition Complex Msh2-Msh6 Required for Mismatch Repair. Journal of Biological Chemistry, 2012, 287, 41232-41244.	1.6	13
36	Bioinformatic identification of genes suppressing genome instability. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, E3251-9.	3.3	25

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37	A chemicalâ€genetic screen to unravel the genetic network of CDC28/CDK1 links ubiquitin and Rad6–Bre1 to cell cycle progression. FASEB Journal, 2012, 26, 590.1.	0.2	0
38	Aneuploidy Drives a Mutator Phenotype in Cancer. Science, 2011, 333, 942-943.	6.0	45
39	Mismatch Repair, But Not Heteroduplex Rejection, Is Temporally Coupled to DNA Replication. Science, 2011, 334, 1713-1716.	6.0	109
40	A chemical-genetic screen to unravel the genetic network of <i>CDC28/CDK1</i> links ubiquitin and Rad6–Bre1 to cell cycle progression. Proceedings of the National Academy of Sciences of the United States of America, 2011, 108, 18748-18753.	3.3	31
41	Functional Studies and Homology Modeling of Msh2-Msh3 Predict that Mispair Recognition Involves DNA Bending and Strand Separation. Molecular and Cellular Biology, 2010, 30, 3321-3328.	1.1	33
42	Determination of Gross Chromosomal Rearrangement Rates. Cold Spring Harbor Protocols, 2010, 2010, 2010, pdb.prot5492.	0.2	39
43	Probing DNA- and ATP-mediated Conformational Changes in the MutS Family of Mispair Recognition Proteins Using Deuterium Exchange Mass Spectrometry. Journal of Biological Chemistry, 2010, 285, 13170-13182.	1.6	40
44	Post-Replication Repair Suppresses Duplication-Mediated Genome Instability. PLoS Genetics, 2010, 6, e1000933.	1.5	39
45	A conserved MutS homolog connector domain interface interacts with MutL homologs. Proceedings of the United States of America, 2009, 106, 22223-22228.	3.3	69
46	Perspectives on the DNA damage and replication checkpoint responses in Saccharomyces cerevisiae. DNA Repair, 2009, 8, 974-982.	1.3	68
47	Specific pathways prevent duplication-mediated genome rearrangements. Nature, 2009, 460, 984-989.	13.7	122
48	Inflammasome-Mediated Disease Animal Models Reveal Roles for Innate but Not Adaptive Immunity. Immunity, 2009, 30, 875-887.	6.6	305
49	Escherichia coli MutS Tetramerization Domain Structure Reveals That Stable Dimers but Not Tetramers Are Essential for DNA Mismatch Repair in Vivo. Journal of Biological Chemistry, 2007, 282, 16345-16354.	1.6	55
50	Chimeric Saccharomyces cerevisiae Msh6 protein with an Msh3 mispair-binding domain combines properties of both proteins. Proceedings of the National Academy of Sciences of the United States of America, 2007, 104, 10956-10961.	3.3	35
51	Coupling distant sites in DNA during DNA mismatch repair. Proceedings of the National Academy of Sciences of the United States of America, 2007, 104, 12953-12954.	3.3	41
52	The N Terminus of Saccharomyces cerevisiae Msh6 Is an Unstructured Tether to PCNA. Molecular Cell, 2007, 26, 565-578.	4.5	110
53	The clinical continuum of cryopyrinopathies: Novel CIAS1 mutations in North American patients and a new cryopyrin model. Arthritis and Rheumatism, 2007, 56, 1273-1285.	6.7	362
54	X-ray solution scattering (SAXS) combined with crystallography and computation: defining accurate macromolecular structures, conformations and assemblies in solution. Quarterly Reviews of Biophysics, 2007, 40, 191-285.	2.4	1,026

Christopher D Putnam

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55	Analysis of Grossâ€Chromosomal Rearrangements in Saccharomyces cerevisiae. Methods in Enzymology, 2006, 409, 462-476.	0.4	50
56	Chromosome healing byde novotelomere addition inSaccharomyces cerevisiae. Molecular Microbiology, 2006, 59, 1357-1368.	1.2	85
57	Mutation in Rpa1 results in defective DNA double-strand break repair, chromosomal instability and cancer in mice. Nature Genetics, 2005, 37, 750-755.	9.4	141
58	Saccharomyces cerevisiae as a Model System To Define the Chromosomal Instability Phenotype. Molecular and Cellular Biology, 2005, 25, 7226-7238.	1.1	51
59	Protein mimicry of DNA and pathway regulation. DNA Repair, 2005, 4, 1410-1420.	1.3	63
60	Chromosome healing through terminal deletions generated by de novo telomere additions in Saccharomyces cerevisiae. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 13262-13267.	3.3	51
61	Mechanism and energetics of green fluorescent protein chromophore synthesis revealed by trapped intermediate structures. Proceedings of the National Academy of Sciences of the United States of America, 2003, 100, 12111-12116.	3.3	194
62	Maintenance of Genome Stability in Saccharomyces cerevisiae. Science, 2002, 297, 552-557.	6.0	442
63	Structure and function correlation in histone H2A peptide-mediated gene transfer. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 7467-7471.	3.3	72
64	DNA double-strand break repair from head to tail. Current Opinion in Structural Biology, 2002, 12, 115-122.	2.6	133
65	Structure and mechanism of the RuvB holliday junction branch migration motor. Journal of Molecular Biology, 2001, 311, 297-310.	2.0	157
66	DNA damage recognition and repair pathway coordination revealed by the structural biochemistry of DNA repair enzymes. Progress in Molecular Biology and Translational Science, 2001, 68, 315-347.	1.9	30
67	The food of sweet and bitter fancy. , 2000, 7, 17-18.		13
68	Active and inhibited human catalase structures: ligand and NADPH binding and catalytic mechanism 1 1Edited by R. Huber. Journal of Molecular Biology, 2000, 296, 295-309.	2.0	388
69	Lessons learned from structural results on uracil-DNA glycosylase. Mutation Research DNA Repair, 2000, 460, 183-199.	3.8	117
70	Evolution and mechanism from structures of an ADP-ribosylating toxin and NAD complex. Nature Structural Biology, 1999, 6, 932-936.	9.7	223
71	DNA REPAIR MECHANISMS FOR THE RECOGNITION AND REMOVAL OF DAMAGED DNA BASES. Annual Review of Biophysics and Biomolecular Structure, 1999, 28, 101-128.	18.3	170
72	Mutation of an Active Site Residue inEscherichia coliUracil-DNA Glycosylase:Â Effect on DNA Binding, Uracil Inhibition and Catalysisâ€. Biochemistry, 1999, 38, 4834-4845.	1.2	28

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73	Protein mimicry of DNA from crystal structures of the uracil-DNA glycosylase inhibitor protein and its complex with Escherichia coli uracil-DNA glycosylase 1 1Edited by D. C. Rees. Journal of Molecular Biology, 1999, 287, 331-346.	2.0	120
74	Rational Design of a Functional Metalloenzyme:  Introduction of a Site for Manganese Binding and Oxidation into a Heme Peroxidase. Biochemistry, 1998, 37, 16853-16862.	1.2	63
75	The RNA polymerase I transcription factor UBF is a sequence-tolerant HMG-box protein that can recognize structured nucleic acids. Nucleic Acids Research, 1994, 22, 2651-2657.	6.5	101
76	Rad5 and Its Human Homologs, HLTF and SHPRH, Are Novel Interactors of Mismatch Repair. Frontiers in Cell and Developmental Biology, 0, 10, .	1.8	1