

Richard D Kolodner

List of Publications by Year in descending order

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134
papers

20,723
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15504

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129
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all docs

138
docs citations

138
times ranked

13549
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#	ARTICLE	IF	CITATIONS
1	The human mutator gene homolog MSH2 and its association with hereditary nonpolyposis colon cancer. <i>Cell</i> , 1993, 75, 1027-1038.	28.9	2,706
2	Mutation in the DNA mismatch repair gene homologue hMLH 1 is associated with hereditary non-polyposis colon cancer. <i>Nature</i> , 1994, 368, 258-261.	27.8	2,001
3	Mutation of a new gene encoding a putative pyrin-like protein causes familial cold autoinflammatory syndrome and Muckle-Wells syndrome. <i>Nature Genetics</i> , 2001, 29, 301-305.	21.4	1,488
4	Eukaryotic DNA mismatch repair. <i>Current Opinion in Genetics and Development</i> , 1999, 9, 89-96.	3.3	781
5	<i>Saccharomyces</i> Ku70, Mre11/Rad50, and RPA Proteins Regulate Adaptation to G2/M Arrest after DNA Damage. <i>Cell</i> , 1998, 94, 399-409.	28.9	729
6	Meiotic Pachytene Arrest in MLH1-Deficient Mice. <i>Cell</i> , 1996, 85, 1125-1134.	28.9	528
7	A Novel Mutation Avoidance Mechanism Dependent on <i>S. cerevisiae</i> RAD27 Is Distinct from DNA Mismatch Repair. <i>Cell</i> , 1997, 88, 253-263.	28.9	452
8	Maintenance of Genome Stability in <i>Saccharomyces cerevisiae</i> . <i>Science</i> , 2002, 297, 552-557.	12.6	442
9	Rewiring of Genetic Networks in Response to DNA Damage. <i>Science</i> , 2010, 330, 1385-1389.	12.6	408
10	The clinical continuum of cryopyrinopathies: Novel CIAS1 mutations in North American patients and a new cryopyrin model. <i>Arthritis and Rheumatism</i> , 2007, 56, 1273-1285.	6.7	362
11	Gross chromosomal rearrangements in <i>Saccharomyces cerevisiae</i> replication and recombination defective mutants. <i>Nature Genetics</i> , 1999, 23, 81-85.	21.4	360
12	An overview of Cdk1-controlled targets and processes. <i>Cell Division</i> , 2010, 5, 11.	2.4	338
13	Multiple pathways cooperate in the suppression of genome instability in <i>Saccharomyces cerevisiae</i> . <i>Nature</i> , 2001, 411, 1073-1076.	27.8	336
14	Mutation in the Mismatch Repair Gene Msh6 Causes Cancer Susceptibility. <i>Cell</i> , 1997, 91, 467-477.	28.9	326
15	SGS1, the <i>Saccharomyces cerevisiae</i> homologue of BLM and WRN, suppresses genome instability and homeologous recombination. <i>Nature Genetics</i> , 2001, 27, 113-116.	21.4	309
16	Suppression of Spontaneous Chromosomal Rearrangements by S Phase Checkpoint Functions in <i>Saccharomyces cerevisiae</i> . <i>Cell</i> , 2001, 104, 397-408.	28.9	301
17	Inactivation of Exonuclease 1 in mice results in DNA mismatch repair defects, increased cancer susceptibility, and male and female sterility. <i>Genes and Development</i> , 2003, 17, 603-614.	5.9	282
18	Structure of the Human MSH2 Locus and Analysis of Two Muir-Torre Kindreds for msh2 Mutations. <i>Genomics</i> , 1994, 24, 516-526.	2.9	276

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19	A genomewide screen in <i>Saccharomyces cerevisiae</i> for genes that suppress the accumulation of mutations. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2003, 100, 11529-11534.	7.1	248
20	Germ-line msh6 mutations in colorectal cancer families. <i>Cancer Research</i> , 1999, 59, 5068-74.	0.9	241
21	Haploinsufficiency of Flap endonuclease (Fen1) leads to rapid tumor progression. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2002, 99, 9924-9929.	7.1	227
22	Proliferating cell nuclear antigen and Msh2p-Msh6p interact to form an active mispair recognition complex. <i>Nature Genetics</i> , 2000, 26, 375-378.	21.4	215
23	Lynch Syndrome (Hereditary Nonpolyposis Colorectal Cancer) Diagnostics. <i>Journal of the National Cancer Institute</i> , 2007, 99, 291-299.	6.3	201
24	Genetic Analysis of Yeast RPA1 Reveals Its Multiple Functions in DNA Metabolism. <i>Genetics</i> , 1998, 148, 989-1005.	2.9	185
25	Visualization of Eukaryotic DNA Mismatch Repair Reveals Distinct Recognition and Repair Intermediates. <i>Cell</i> , 2011, 147, 1040-1053.	28.9	183
26	Characterization of DNA-binding and strand-exchange stimulation properties of γ -RPA, a yeast single-strand-DNA-binding protein. <i>Journal of Molecular Biology</i> , 1992, 227, 54-71.	4.2	180
27	<i>exo1</i> -Dependent Mutator Mutations: Model System for Studying Functional Interactions in Mismatch Repair. <i>Molecular and Cellular Biology</i> , 2001, 21, 5142-5155.	2.3	177
28	A hereditary nonpolyposis colorectal carcinoma case associated with hypermethylation of the MLH1 gene in normal tissue and loss of heterozygosity of the unmethylated allele in the resulting microsatellite instability-high tumor. <i>Cancer Research</i> , 2002, 62, 3925-8.	0.9	174
29	<i>recA</i> -independent general genetic recombination of plasmids. <i>Nature</i> , 1981, 294, 184-186.	27.8	168
30	Chromosomal Rearrangements Occur in <i>S. cerevisiae</i> <i>rfa1</i> Mutator Mutants Due to Mutagenic Lesions Processed by Double-Strand-Break Repair. <i>Molecular Cell</i> , 1998, 2, 9-22.	9.7	153
31	Mutation in <i>Rpa1</i> results in defective DNA double-strand break repair, chromosomal instability and cancer in mice. <i>Nature Genetics</i> , 2005, 37, 750-755.	21.4	141
32	Dominant effects of an Msh6 missense mutation on DNA repair and cancer susceptibility. <i>Cancer Cell</i> , 2004, 6, 139-150.	16.8	140
33	Functional analysis of human MLH1 mutations in <i>Saccharomyces cerevisiae</i> . <i>Nature Genetics</i> , 1998, 19, 384-389.	21.4	136
34	<i>Saccharomyces cerevisiae</i> chromatin-assembly factors that act during DNA replication function in the maintenance of genome stability. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2003, 100, 6640-6645.	7.1	136
35	Suppression of genome instability by redundant S-phase checkpoint pathways in <i>Saccharomyces cerevisiae</i> . <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2002, 99, 4500-4507.	7.1	135
36	The Evolutionarily Conserved Zinc Finger Motif in the Largest Subunit of Human Replication Protein A Is Required for DNA Replication and Mismatch Repair but Not for Nucleotide Excision Repair. <i>Journal of Biological Chemistry</i> , 1998, 273, 1453-1461.	3.4	130

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37	Specific pathways prevent duplication-mediated genome rearrangements. <i>Nature</i> , 2009, 460, 984-989.	27.8	122
38	Biochemical Characterization of the Interaction between the <i>Saccharomyces cerevisiae</i> MSH2-MSH6 Complex and Mismatched Bases in DNA. <i>Journal of Biological Chemistry</i> , 1999, 274, 26668-26682.	3.4	121
39	Interpretation of Genetic Test Results for Hereditary Nonpolyposis Colorectal Cancer. <i>JAMA - Journal of the American Medical Association</i> , 1999, 282, 247.	7.4	118
40	Analysis of the Interaction between the <i>Saccharomyces cerevisiae</i> MSH2-MSH6 and MLH1-PMS1 Complexes with DNA Using a Reversible DNA End-blocking System. <i>Journal of Biological Chemistry</i> , 2005, 280, 22245-22257.	3.4	116
41	Exonuclease 1-dependent and independent mismatch repair. <i>DNA Repair</i> , 2015, 32, 24-32.	2.8	115
42	Links between replication, recombination and genome instability in eukaryotes. <i>Trends in Biochemical Sciences</i> , 2000, 25, 196-200.	7.5	111
43	The N Terminus of <i>Saccharomyces cerevisiae</i> Msh6 Is an Unstructured Tether to PCNA. <i>Molecular Cell</i> , 2007, 26, 565-578.	9.7	110
44	Mismatch Repair, But Not Heteroduplex Rejection, Is Temporally Coupled to DNA Replication. <i>Science</i> , 2011, 334, 1713-1716.	12.6	109
45	The role of heteroduplex correction in gene conversion in <i>Saccharomyces cerevisiae</i> . <i>Nature</i> , 1987, 328, 362-364.	27.8	108
46	A Biological Network in <i>Saccharomyces cerevisiae</i> Prevents the Deleterious Effects of Endogenous Oxidative DNA Damage. <i>Molecular Cell</i> , 2005, 17, 709-720.	9.7	104
47	New insights into the mechanism of DNA mismatch repair. <i>Chromosoma</i> , 2015, 124, 443-462.	2.2	103
48	Inhibition of Nuclear PTEN Tyrosine Phosphorylation Enhances Glioma Radiation Sensitivity through Attenuated DNA Repair. <i>Cancer Cell</i> , 2019, 35, 504-518.e7.	16.8	102
49	<i>Saccharomyces cerevisiae</i> pol30 (Proliferating Cell Nuclear Antigen) Mutations Impair Replication Fidelity and Mismatch Repair. <i>Molecular and Cellular Biology</i> , 1999, 19, 7801-7815.	2.3	100
50	Tumour predisposition and cancer syndromes as models to study gene-environment interactions. <i>Nature Reviews Cancer</i> , 2020, 20, 533-549.	28.4	93
51	Homologous pairing proteins encoded by the <i>Escherichia coli</i> recE and recT genes. <i>Molecular Microbiology</i> , 1994, 11, 23-30.	2.5	92
52	<i>Saccharomyces cerevisiae</i> Msh2-Msh3 Acts in Repair of Base-Base Mismatches. <i>Molecular and Cellular Biology</i> , 2007, 27, 6546-6554.	2.3	89
53	PCNA and Msh2-Msh6 Activate an Mlh1-Pms1 Endonuclease Pathway Required for Exo1-Independent Mismatch Repair. <i>Molecular Cell</i> , 2014, 55, 291-304.	9.7	89
54	Transfer of the MSH2-MSH6 Complex from Proliferating Cell Nuclear Antigen to Mismatched Bases in DNA. <i>Journal of Biological Chemistry</i> , 2003, 278, 14-17.	3.4	87

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55	Inhibition of Msh6 ATPase Activity by Mispai red DNA Induces a Msh2(ATP)-Msh6(ATP) State Capable of Hydrolysis-Independent Movement along DNA. <i>Molecular Cell</i> , 2006, 22, 39-49.	9.7	87
56	The Recombination-deficient Mutant RPA (rfa1-t11) Is Displaced Slowly from Single-stranded DNA by Rad51 Protein. <i>Journal of Biological Chemistry</i> , 2003, 278, 23410-23417.	3.4	85
57	Chromosome healing by de novo telomere addition in <i>Saccharomyces cerevisiae</i> . <i>Molecular Microbiology</i> , 2006, 59, 1357-1368.	2.5	85
58	An FHA domain-mediated protein interaction network of Rad53 reveals its role in polarized cell growth. <i>Journal of Cell Biology</i> , 2006, 175, 743-753.	5.2	85
59	Oxygen metabolism and reactive oxygen species cause chromosomal rearrangements and cell death. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2007, 104, 9747-9752.	7.1	84
60	Characterization of Hereditary Nonpolyposis Colorectal Cancer Families From a Population-Based Series of Cases. <i>Journal of the National Cancer Institute</i> , 2000, 92, 1517-1522.	6.3	80
61	Checkpoint proteins control morphogenetic events during DNA replication stress in <i>Saccharomyces cerevisiae</i> . <i>Journal of Cell Biology</i> , 2006, 175, 729-741.	5.2	79
62	Induction of genome instability by DNA damage in <i>Saccharomyces cerevisiae</i> . <i>DNA Repair</i> , 2003, 2, 243-258.	2.8	74
63	The C-terminal domain of yeast PCNA is required for physical and functional interactions with Cdc9 DNA ligase. <i>Nucleic Acids Research</i> , 2007, 35, 1624-1637.	14.5	70
64	A conserved MutS homolog connector domain interface interacts with MutL homologs. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2009, 106, 22223-22228.	7.1	69
65	Perspectives on the DNA damage and replication checkpoint responses in <i>Saccharomyces cerevisiae</i> . <i>DNA Repair</i> , 2009, 8, 974-982.	2.8	68
66	Distinct SUMO Ligases Cooperate with Esc2 and Slx5 to Suppress Duplication-Mediated Genome Rearrangements. <i>PLoS Genetics</i> , 2013, 9, e1003670.	3.5	68
67	A Genetic and Structural Study of Genome Rearrangements Mediated by High Copy Repeat Ty1 Elements. <i>PLoS Genetics</i> , 2011, 7, e1002089.	3.5	65
68	Control of Translocations between Highly Diverged Genes by Sgs1, the <i>Saccharomyces cerevisiae</i> Homolog of the Bloom's Syndrome Protein. <i>Molecular and Cellular Biology</i> , 2006, 26, 5406-5420.	2.3	62
69	Isolation and Characterization of New Proliferating Cell Nuclear Antigen (PCNA) Mutator Mutants That Are Defective in DNA Mismatch Repair. <i>Molecular and Cellular Biology</i> , 2002, 22, 6669-6680.	2.3	61
70	Recombination and the Tel1 and Mec1 checkpoints differentially effect genome rearrangements driven by telomere dysfunction in yeast. <i>Nature Genetics</i> , 2004, 36, 612-617.	21.4	61
71	Checkpoint Kinases Regulate a Global Network of Transcription Factors in Response to DNA Damage. <i>Cell Reports</i> , 2013, 4, 174-188.	6.4	61
72	Dominant <i>Saccharomyces cerevisiae</i> msh6 Mutations Cause Increased Mismatch Binding and Decreased Dissociation from Mismatches by Msh2-Msh6 in the Presence of ATP. <i>Journal of Biological Chemistry</i> , 2002, 277, 25545-25553.	3.4	59

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73	Novel dominant mutations in <i>Saccharomyces cerevisiae</i> MSH6. <i>Nature Genetics</i> , 2000, 24, 53-56.	21.4	58
74	Replication Protein A Is Required for Meiotic Recombination in <i>Saccharomyces cerevisiae</i> . <i>Genetics</i> , 2002, 161, 535-547.	2.9	57
75	<i>Escherichia coli</i> MutS Tetramerization Domain Structure Reveals That Stable Dimers but Not Tetramers Are Essential for DNA Mismatch Repair in Vivo. <i>Journal of Biological Chemistry</i> , 2007, 282, 16345-16354.	3.4	55
76	The MER3 Helicase Involved in Meiotic Crossing Over Is Stimulated by Single-stranded DNA-binding Proteins and Unwinds DNA in the 3' to 5' Direction. <i>Journal of Biological Chemistry</i> , 2001, 276, 31487-31493.	3.4	54
77	Reconstitution of long and short patch mismatch repair reactions using <i>Saccharomyces cerevisiae</i> proteins. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2013, 110, 18472-18477.	7.1	53
78	DNA conformations in mismatch repair probed in solution by X-ray scattering from gold nanocrystals. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2013, 110, 17308-17313.	7.1	53
79	FEN1 endonuclease as a therapeutic target for human cancers with defects in homologous recombination. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2020, 117, 19415-19424.	7.1	53
80	Dominant Mutations in <i>S. cerevisiae</i> PMS1 Identify the Mlh1-Pms1 Endonuclease Active Site and an Exonuclease 1-Independent Mismatch Repair Pathway. <i>PLoS Genetics</i> , 2013, 9, e1003869.	3.5	52
81	Chromosome healing through terminal deletions generated by de novo telomere additions in <i>Saccharomyces cerevisiae</i> . <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2004, 101, 13262-13267.	7.1	51
82	<i>Saccharomyces cerevisiae</i> as a Model System To Define the Chromosomal Instability Phenotype. <i>Molecular and Cellular Biology</i> , 2005, 25, 7226-7238.	2.3	51
83	Analysis of Gross Chromosomal Rearrangements in <i>Saccharomyces cerevisiae</i> . <i>Methods in Enzymology</i> , 2006, 409, 462-476.	1.0	50
84	Interaction between the Msh2 and Msh6 Nucleotide-binding Sites in the <i>Saccharomyces cerevisiae</i> Msh2-Msh6 Complex. <i>Journal of Biological Chemistry</i> , 2010, 285, 9301-9310.	3.4	50
85	Mitotic checkpoint function in the formation of gross chromosomal rearrangements in <i>Saccharomyces cerevisiae</i> . <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2004, 101, 15980-15985.	7.1	49
86	Pathways and Mechanisms that Prevent Genome Instability in <i>Saccharomyces cerevisiae</i> . <i>Genetics</i> , 2017, 206, 1187-1225.	2.9	49
87	Guidelines for DNA recombination and repair studies: Cellular assays of DNA repair pathways. <i>Microbial Cell</i> , 2019, 6, 1-64.	3.2	47
88	A <i>Saccharomyces cerevisiae</i> RNase H2 Interaction Network Functions To Suppress Genome Instability. <i>Molecular and Cellular Biology</i> , 2014, 34, 1521-1534.	2.3	46
89	EXO1 variants occur commonly in normal population: evidence against a role in hereditary nonpolyposis colorectal cancer. <i>Cancer Research</i> , 2003, 63, 154-8.	0.9	46
90	Aneuploidy Drives a Mutator Phenotype in Cancer. <i>Science</i> , 2011, 333, 942-943.	12.6	45

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91	A personal historical view of DNA mismatch repair with an emphasis on eukaryotic DNA mismatch repair. <i>DNA Repair</i> , 2016, 38, 3-13.	2.8	44
92	Alternative splicing regulates stochastic NLRP3 activity. <i>Nature Communications</i> , 2019, 10, 3238.	12.8	44
93	Suppression of spontaneous genome rearrangements in yeast DNA helicase mutants. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2006, 103, 18196-18201.	7.1	42
94	Mispair-specific Recruitment of the Mlh1-Pms1 Complex Identifies Repair Substrates of the <i>Saccharomyces cerevisiae</i> Msh2-Msh3 Complex. <i>Journal of Biological Chemistry</i> , 2014, 289, 9352-9364.	3.4	42
95	Coupling distant sites in DNA during DNA mismatch repair. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2007, 104, 12953-12954.	7.1	41
96	Determination of Gross Chromosomal Rearrangement Rates. <i>Cold Spring Harbor Protocols</i> , 2010, 2010, pdb.prot5492.	0.3	39
97	Post-Replication Repair Suppresses Duplication-Mediated Genome Instability. <i>PLoS Genetics</i> , 2010, 6, e1000933.	3.5	39
98	Cdc28/Cdk1 positively and negatively affects genome stability in <i>S. cerevisiae</i> . <i>Journal of Cell Biology</i> , 2009, 185, 423-437.	5.2	37
99	Mlh2 Is an Accessory Factor for DNA Mismatch Repair in <i>Saccharomyces cerevisiae</i> . <i>PLoS Genetics</i> , 2014, 10, e1004327.	3.5	36
100	A genetic network that suppresses genome rearrangements in <i>Saccharomyces cerevisiae</i> and contains defects in cancers. <i>Nature Communications</i> , 2016, 7, 11256.	12.8	36
101	Chimeric <i>Saccharomyces cerevisiae</i> Msh6 protein with an Msh3 mispair-binding domain combines properties of both proteins. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2007, 104, 10956-10961.	7.1	35
102	Identification of Exo1-Msh2 interaction motifs in DNA mismatch repair and new Msh2-binding partners. <i>Nature Structural and Molecular Biology</i> , 2018, 25, 650-659.	8.2	35
103	Suppression of gross chromosomal rearrangements by the multiple functions of the Mre11-Rad50-Xrs2 complex in <i>Saccharomyces cerevisiae</i> . <i>DNA Repair</i> , 2005, 4, 606-617.	2.8	34
104	Stabilization of Dicentric Translocations through Secondary Rearrangements Mediated by Multiple Mechanisms in <i>S. cerevisiae</i> . <i>PLoS ONE</i> , 2009, 4, e6389.	2.5	34
105	Functional Studies and Homology Modeling of Msh2-Msh3 Predict that Mispair Recognition Involves DNA Bending and Strand Separation. <i>Molecular and Cellular Biology</i> , 2010, 30, 3321-3328.	2.3	33
106	Isolation of genetic elements that increase frequencies of plasmid recombinants. <i>Nature</i> , 1983, 303, 256-259.	27.8	29
107	Checkpoint functions are required for normal S-phase progression in <i>Saccharomyces cerevisiae</i> RCAF- and CAF-I-defective mutants. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2006, 103, 3710-3715.	7.1	29
108	Activation of <i>Saccharomyces cerevisiae</i> Mlh1-Pms1 Endonuclease in a Reconstituted Mismatch Repair System. <i>Journal of Biological Chemistry</i> , 2015, 290, 21580-21590.	3.4	29

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109	Biochemical basis for dominant mutations in the <i>Saccharomyces cerevisiae</i> MSH6 gene. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103, 558-563.	7.1	27
110	Bioinformatic identification of genes suppressing genome instability. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, E3251-9.	7.1	25
111	Reconstitution of <i>Saccharomyces cerevisiae</i> DNA polymerase $\hat{\mu}$ -dependent mismatch repair with purified proteins. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, 3607-3612.	7.1	24
112	The properties of Msh2â€‘Msh6 ATP binding mutants suggest a signal amplification mechanism in DNA mismatch repair. Journal of Biological Chemistry, 2018, 293, 18055-18070.	3.4	24
113	The <i>Saccharomyces cerevisiae</i> Rad6 Postreplication Repair and Siz1/Srs2 Homologous Recombination-Inhibiting Pathways Process DNA Damage That Arises in <i>asf1</i> Mutants. Molecular and Cellular Biology, 2009, 29, 5226-5237.	2.3	23
114	Genetic analysis of ionizing radiation-induced mutagenesis in <i>Saccharomyces cerevisiae</i> reveals TransLesion Synthesis (TLS) independent of PCNA K164 SUMOylation and ubiquitination. DNA Repair, 2006, 5, 1475-1488.	2.8	21
115	Low frequency of hMSH2 mutations in Swedish HNPCC families. , 1997, 74, 134-137.		20
116	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.	3.5	20
117	Ligation of newly replicated DNA controls the timing of DNA mismatch repair. Current Biology, 2021, 31, 1268-1276.e6.	3.9	19
118	Guarding against mutation. Nature, 2000, 407, 687-689.	27.8	18
119	The Swr1 chromatin-remodeling complex prevents genome instability induced by replication fork progression defects. Nature Communications, 2018, 9, 3680.	12.8	17
120	SUMO E3 ligase Mms21 prevents spontaneous DNA damage induced genome rearrangements. PLoS Genetics, 2018, 14, e1007250.	3.5	16
121	Rapid Analysis of <i>Saccharomyces cerevisiae</i> Genome Rearrangements by Multiplex Ligationâ€‘Dependent Probe Amplification. PLoS Genetics, 2012, 8, e1002539.	3.5	15
122	Cdc3 suppresses genome instability by mediating telomere homeostasis. PLoS Genetics, 2018, 14, e1007170.	3.5	15
123	Loss of the Thioredoxin Reductase Trr1 Suppresses the Genomic Instability of Peroxiredoxin tsa1 Mutants. PLoS ONE, 2014, 9, e108123.	2.5	14
124	Uner Tan syndrome caused by a homozygous TUBB2B mutation affecting microtubule stability. Human Molecular Genetics, 2016, 26, ddw383.	2.9	11
125	Mechanisms underlying genome instability mediated by formation of foldback inversions in <i>Saccharomyces cerevisiae</i> . ELife, 2020, 9, .	6.0	10
126	Analyzing Genome Rearrangements in <i>Saccharomyces cerevisiae</i> . Methods in Molecular Biology, 2018, 1672, 43-61.	0.9	9

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127	Rad27 and Exo1 function in different excision pathways for mismatch repair in <i>Saccharomyces cerevisiae</i> . <i>Nature Communications</i> , 2021, 12, 5568.	12.8	9
128	S-Score: A Scoring System for the Identification and Prioritization of Predicted Cancer Genes. <i>PLoS ONE</i> , 2014, 9, e94147.	2.5	8
129	Essential <i>Saccharomyces cerevisiae</i> genome instability suppressing genes identify potential human tumor suppressors. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2019, 116, 17377-17382.	7.1	8
130	The Yeast HSM3 Gene Is Not Involved in DNA Mismatch Repair in Rapidly Dividing Cells. <i>Genetics</i> , 2000, 154, 491-493.	2.9	4
131	DNA Mismatch Repair: Mechanisms and Cancer Genetics. , 2018, , .		1
132	Rad5 and Its Human Homologs, HLTf and SHPRH, Are Novel Interactors of Mismatch Repair. <i>Frontiers in Cell and Developmental Biology</i> , 0, 10, .	3.7	1
133	A chemicalâ€genetic screen to unravel the genetic network of CDC28/CDK1 links ubiquitin and Rad6â€Bre1 to cell cycle progression. <i>FASEB Journal</i> , 2012, 26, 590.1.	0.5	0
134	Template homology determines the genetics and mechanisms of gross chromosomal rearrangements in <i>S. cerevisiae</i> (736.11). <i>FASEB Journal</i> , 2014, 28, 736.11.	0.5	0