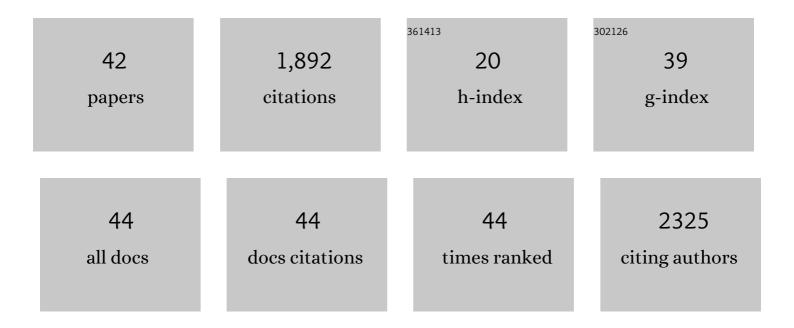
Niels de Wind

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	CNOT6: A Novel Regulator of DNA Mismatch Repair. Cells, 2022, 11, 521.	4.1	4
2	Rev1 deficiency induces replication stress to cause metabolic dysfunction differently in males and females. American Journal of Physiology - Endocrinology and Metabolism, 2022, 322, E319-E329.	3.5	2
3	Predictive functional assayâ€based classification of PMS2 variants in Lynch syndrome. Human Mutation, 2022, , .	2.5	1
4	OUP accepted manuscript. Carcinogenesis, 2021, , .	2.8	3
5	Effect of sequence context on Polζ-dependent error-prone extension past (6-4) photoproducts. DNA Repair, 2020, 87, 102771.	2.8	7
6	DNA mismatch repair-dependent DNA damage responses and cancer. DNA Repair, 2020, 93, 102923.	2.8	43
7	Contribution of mRNA Splicing to Mismatch Repair Gene Sequence Variant Interpretation. Frontiers in Genetics, 2020, 11, 798.	2.3	19
8	Digenic inheritance of <scp> <i>MSH6</i> </scp> and <scp> <i>MUTYH</i> </scp> variants in familial colorectal cancer. Genes Chromosomes and Cancer, 2020, 59, 697-701.	2.8	9
9	Two integrated and highly predictive functional analysis-based procedures for the classification of MSH6 variants in Lynch syndrome. Genetics in Medicine, 2020, 22, 847-856.	2.4	16
10	Mutagenic replication: target for tumor therapy?. Cell Research, 2019, 29, 783-784.	12.0	0
11	A functional assay–based procedure to classify mismatch repair gene variants in Lynch syndrome. Genetics in Medicine, 2019, 21, 1486-1496.	2.4	36
12	Adjuvant Treatment for <i>POLE</i> Proofreading Domain–Mutant Cancers: Sensitivity to Radiotherapy, Chemotherapy, and Nucleoside Analogues. Clinical Cancer Research, 2018, 24, 3197-3203.	7.0	50
13	Rev1 contributes to proper mitochondrial function via the PARP-NAD+-SIRT1-PGC1α axis. Scientific Reports, 2017, 7, 12480.	3.3	17
14	Genomic and functional integrity of the hematopoietic system requires tolerance of oxidative DNA lesions. Blood, 2017, 130, 1523-1534.	1.4	29
15	Comprehensive Mutation Analysis of <i>PMS2</i> in a Large Cohort of Probands Suspected of Lynch Syndrome or Constitutional Mismatch Repair Deficiency Syndrome. Human Mutation, 2016, 37, 1162-1179.	2.5	50
16	DNA mismatch repair: from biophysics to bedside. DNA Repair, 2016, 38, 1-2.	2.8	3
17	De novo mutations in PLXND1 and REV3L cause Möbius syndrome. Nature Communications, 2015, 6, 7199.	12.8	76
18	Roles of mutagenic translesion synthesis in mammalian genome stability, health and disease. DNA Repair, 2015, 29, 56-64.	2.8	33

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19	FANCD2 and REV1 cooperate in the protection of nascent DNA strands in response to replication stress. Nucleic Acids Research, 2015, 43, 8325-8339.	14.5	38
20	Roles of PCNA ubiquitination and TLS polymerases l̂º and l̂∙ in the bypass of methyl methanesulfonate-induced DNA damage. Nucleic Acids Research, 2015, 43, 282-294.	14.5	41
21	Excision of translesion synthesis errors orchestrates responses to helix-distorting DNA lesions. Journal of Cell Biology, 2015, 209, 33-46.	5.2	16
22	When mismatch repair met translesion synthesis. Cell Cycle, 2015, 14, 2377-2378.	2.6	0
23	Consequences of germline variation disrupting the constitutional translational initiation codon start sites of <i>MLH1</i> and <i>BRCA2</i> : Use of potential alternative start sites and implications for predicting variant pathogenicity. Molecular Carcinogenesis, 2015, 54, 513-522.	2.7	14
24	Post-translesion synthesis repair. Oncotarget, 2015, 6, 19342-19343.	1.8	0
25	Redundancy of mammalian Y family DNA polymerases in cellular responses to genomic DNA lesions induced by ultraviolet light. Nucleic Acids Research, 2014, 42, 11071-11082.	14.5	30
26	Maternal Aldehyde Elimination during Pregnancy Preserves the Fetal Genome. Molecular Cell, 2014, 55, 807-817.	9.7	55
27	In memory of John Bruce Hays (1937–2014). DNA Repair, 2014, 16, vi-vii.	2.8	0
28	Inactivation of DNA Mismatch Repair by Variants of Uncertain Significance in the <i>PMS2</i> Gene. Human Mutation, 2013, 34, 1477-1480.	2.5	26
29	Genetic screens to identify pathogenic gene variants in the common cancer predisposition Lynch syndrome. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, 9403-9408.	7.1	21
30	Temporally distinct translesion synthesis pathways for ultraviolet light-induced photoproducts in the mammalian genome. DNA Repair, 2012, 11, 550-558.	2.8	37
31	A rapid and cell-free assay to test the activity of lynch syndrome-associated MSH2 and MSH6 missense variants. Human Mutation, 2012, 33, 488-494.	2.5	46
32	A cell-free assay for the functional analysis of variants of the mismatch repair protein MLH1. Human Mutation, 2010, 31, 247-253.	2.5	56
33	Transcription and replication: Far relatives make uneasy bedfellows. Cell Cycle, 2010, 9, 2300-2304.	2.6	8
34	Transcription-coupled repair and apoptosis provide specific protection against transcription-associated mutagenesis by ultraviolet light. Transcription, 2010, 1, 95-98.	3.1	6
35	Two Distinct Translesion Synthesis Pathways across a Lipid Peroxidation-derived DNA Adduct in Mammalian Cells. Journal of Biological Chemistry, 2009, 284, 191-198.	3.4	26
36	Functional interactions between DNA damage signaling and mutagenic translesion synthesis at post-replicative gaps. Cell Cycle, 2009, 8, 2857-2858.	2.6	4

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#	Article	IF	CITATIONS
37	Gene transcription increases DNA damage-induced mutagenesis in mammalian stem cells. DNA Repair, 2008, 7, 1330-1339.	2.8	26
38	DNA mismatch repair mediates protection from mutagenesis induced by short-wave ultraviolet light. DNA Repair, 2006, 5, 1364-1372.	2.8	19
39	Spontaneous and mutagen-induced loss of DNA mismatch repair in Msh2-heterozygous mammalian cells. Mutation Research - Fundamental and Molecular Mechanisms of Mutagenesis, 2005, 574, 50-57.	1.0	16
40	Biological functions of translesion synthesis proteins in vertebrates. DNA Repair, 2003, 2, 1075-1085.	2.8	27
41	HNPCC-like cancer predisposition in mice through simultaneous loss of Msh3 and Msh6 mismatch-repair protein functions. Nature Genetics, 1999, 23, 359-362.	21.4	199
42	Inactivation of the mouse Msh2 gene results in mismatch repair deficiency, methylation tolerance, hyperrecombination, and predisposition to cancer. Cell, 1995, 82, 321-330.	28.9	777