Geroge Iliakis

List of Publications by Year
in descending order

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120	7,893	70961 41	51492 86
papers	citations	h-index	g-index

120	120	120	8043
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	DNA Damage Clustering after Ionizing Radiation and Consequences in the Processing of Chromatin Breaks. Molecules, 2022, 27, 1540.	1.7	20
2	Shift in G1-Checkpoint from ATM-Alone to a Cooperative ATM Plus ATR Regulation with Increasing Dose of Radiation. Cells, 2022, 11, 63.	1.8	5
3	Depletion of HIF-1Î± by Inducible Cre/loxP Increases the Sensitivity of Cultured Murine Hepatocytes to Ionizing Radiation in Hypoxia. Cells, 2022, 11, 1671.	1.8	3
4	Increased Resection at DSBs in G2-Phase Is a Unique Phenotype Associated with DNA-PKcs Defects That Is Not Shared by Other Factors of c-NHEJ. Cells, 2022, 11, 2099.	1.8	6
5	ATR Contributes More Than ATM in Intra-S-Phase Checkpoint Activation after IR, and DNA-PKcs Facilitates Recovery: Evidence for Modular Integration of ATM/ATR/DNA-PKcs Functions. International Journal of Molecular Sciences, 2022, 23, 7506.	1.8	2
6	Loss of TGFÎ² signaling increases alternative end-joining DNA repair that sensitizes to genotoxic therapies across cancer types. Science Translational Medicine, 2021, 13, .	5.8	33
7	Nucleoside Analogs Radiosensitize G0 Cells by Activating DNA End Resection and Alternative End-Joining. Radiation Research, 2021, 195, 412-426.	0.7	2
8	G2/M Checkpoint Abrogation With Selective Inhibitors Results in Increased Chromatid Breaks and Radiosensitization of 82-6 hTERT and RPE Human Cells. Frontiers in Public Health, 2021, 9, 675095.	1.3	5
9	Proficiency in homologous recombination repair is prerequisite for activation of G2-checkpoint at low radiation doses. DNA Repair, 2021, 101, 103076.	1.3	10
10	Strong Shift to ATR-Dependent Regulation of the G2-Checkpoint after Exposure to High-LET Radiation. Life, 2021, 11, 560.	1.1	8
11	Analysis of chromatid-break-repair detects a homologous recombination to non-homologous end-joining switch with increasing load of DNA double-strand breaks. Mutation Research - Genetic Toxicology and Environmental Mutagenesis, 2021, 867, 503372.	0.9	12
12	Disruption of Chromatin Dynamics by Hypotonic Stress Suppresses HR and Shifts DSB Processing to Error-Prone SSA. International Journal of Molecular Sciences, 2021, 22, 10957.	1.8	1
13	NTRK1/TrkA Activation Overrides the G2/M-Checkpoint upon Irradiation. Cancers, 2021, 13, 6023.	1.7	2
14	Strong suppression of gene conversion with increasing DNA double-strand break load delimited by 53BP1 and RAD52. Nucleic Acids Research, 2020, 48, 1905-1924.	6.5	37
15	Replication protein A: a multifunctional protein with roles in DNA replication, repair and beyond. NAR Cancer, 2020, 2, zcaa022.	1.6	63
16	SCFSKP2 regulates APC/CCDH1-mediated degradation of CTIP to adjust DNA-end resection in G2-phase. Cell Death and Disease, 2020, 11, 548.	2.7	6
17	Complete Genome Sequence of the Deep-Sea Bacterium Moritella marina MP-1 (ATCC 15381). Microbiology Resource Announcements, 2020, 9, .	0.3	1
18	Chromosome breaks generated by low doses of ionizing radiation in G2-phase are processed exclusively by gene conversion. DNA Repair, 2020, 89, 102828.	1.3	19

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19	Biochemical evidence for Ku-independent backup pathways of NHEJ. Nucleic Acids Research, 2020, 48, 5200-5200.	6.5	5
20	Rewiring E2F1 with classical NHEJ via APLF suppression promotes bladder cancer invasiveness. Journal of Experimental and Clinical Cancer Research, 2019, 38, 292.	3.5	15
21	DNA-PKcs and ATM epistatically suppress DNA end resection and hyperactivation of ATR-dependent G2-checkpoint in S-phase irradiated cells. Scientific Reports, 2019, 9, 14597.	1.6	24
22	Necessities in the Processing of DNA Double Strand Breaks and Their Effects on Genomic Instability and Cancer. Cancers, 2019, 11, 1671.	1.7	77
23	Regulation of ABCA1-mediated cholesterol efflux by sphingosine-1-phosphate signaling in macrophages. Journal of Lipid Research, 2019, 60, 506-515.	2.0	32
24	Radiation-dose-dependent functional synergisms between ATM, ATR and DNA-PKcs in checkpoint control and resection in G2-phase. Scientific Reports, 2019, 9, 8255.	1.6	45
25	A method for the cell-cycle-specific analysis of radiation-induced chromosome aberrations and breaks. Mutation Research - Fundamental and Molecular Mechanisms of Mutagenesis, 2019, 815, 10-19.	0.4	7
26	DEFINED BIOLOGICAL MODELS OF HIGH-LET RADIATION LESIONS. Radiation Protection Dosimetry, 2019, 183, 60-68.	0.4	13
27	When a duck is not a duck; a new interdisciplinary synthesis for environmental radiation protection. Environmental Research, 2018, 162, 318-324.	3.7	15
28	Intercellular communication of DNA damage and oxidative status underpin bystander effects. International Journal of Radiation Biology, 2018, 94, 719-726.	1.0	22
29	Relating Linear Energy Transfer to the Formation and Resolution of DNA Repair Foci After Irradiation with Equal Doses of X-ray Photons, Plateau, or Bragg-Peak Protons. International Journal of Molecular Sciences, 2018, 19, 3779.	1.8	29
30	Processing-Challenges Generated by Clusters of DNA Double-Strand Breaks Underpin Increased Effectiveness of High-LET Radiation and Chromothripsis. Advances in Experimental Medicine and Biology, 2018, 1044, 149-168.	0.8	19
31	Restraining Akt1 Phosphorylation Attenuates the Repair of Radiation-Induced DNA Double-Strand Breaks and Reduces the Survival of Irradiated Cancer Cells. International Journal of Molecular Sciences, 2018, 19, 2233.	1.8	12
32	The Biological Foundations of Risks from Ionizing Radiation Exposures: How an Understanding of Associated Effects Will Help Their Quantification and Mitigation. Strategies for Sustainability, 2018, , 149-158.	0.2	0
33	Deregulated BCL-2 family proteins impact on repair of DNA double-strand breaks and are targets to overcome radioresistance in lung cancer. Journal of Cancer Research and Clinical Oncology, 2017, 143, 1733-1744.	1.2	8
34	Novel Biological Approaches for Testing the Contributions of Single DSBs and DSB Clusters to the Biological Effects of High LET Radiation. Frontiers in Oncology, 2016, 6, 163.	1.3	21
35	Measurement of complex DNA damage induction and repair in human cellular systems after exposure to ionizing radiations of varying linear energy transfer (LET). Free Radical Research, 2016, 50, S64-S78.	1.5	95
36	VCP/p97 Extracts Sterically Trapped Ku70/80 Rings from DNA in Double-Strand Break Repair. Molecular Cell, 2016, 64, 189-198.	4.5	91

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37	Non-DSB clustered DNA lesions. Does theory colocalize with the experiment?. Radiation Physics and Chemistry, 2016, 128, 26-35.	1.4	27
38	Chromosome thripsis by DNA double strand break clusters causes enhanced cell lethality, chromosomal translocations and 53BP1-recruitment. Nucleic Acids Research, 2016, 44, 7673-7690.	6.5	46
39	DNA double-strand-break repair in higher eukaryotes and its role in genomic instability and cancer: Cell cycle and proliferation-dependent regulation. Seminars in Cancer Biology, 2016, 37-38, 51-64.	4.3	220
40	<scp>DNA</scp> Ligases I and <scp>III</scp> Support Nucleotide Excision Repair in <scp>DT</scp>40 Cells with Similar Efficiency. Photochemistry and Photobiology, 2015, 91, 1173-1180.	1.3	14
41	Alternative Okazaki Fragment Ligation Pathway by DNA Ligase III. Genes, 2015, 6, 385-398.	1.0	22
42	Role for Artemis nuclease in the repair of radiation-induced DNA double strand breaks by alternative end joining. DNA Repair, 2015, 31, 29-40.	1.3	18
43	Inhibition of Homologous Recombination and Promotion of Mutagenic Repair of DNA Double-Strand Breaks Underpins Arabinosideâ€“Nucleoside Analogue Radiosensitization. Molecular Cancer Therapeutics, 2015, 14, 1424-1433.	1.9	13
44	Alternative end-joining repair pathways are the ultimate backup for abrogated classical non-homologous end-joining and homologous recombination repair: Implications for the formation of chromosome translocations. Mutation Research - Genetic Toxicology and Environmental Mutagenesis, 2015, 793, 166-175.	0.9	159
45	Marked contribution of alternative end-joining to chromosome-translocation-formation by stochastically induced DNA double-strand-breaks in G2-phase human cells. Mutation Research - Genetic Toxicology and Environmental Mutagenesis, 2015, 793, 2-8.	0.9	32
46	The yield of DNA double strand breaks determined after exclusion of those forming from heat-labile lesions predicts tumor cell radiosensitivity to killing. Radiotherapy and Oncology, 2015, 116, 366-373.	0.3	14
47	The contribution of thermally labile sugar lesions to DNA double-strand break formation in cells grown in the presence of BrdU. International Journal of Radiation Biology, 2015, 91, 312-320.	1.0	1
48	Bystander effects as manifestation of intercellular communication of DNA damage and of the cellular oxidative status. Cancer Letters, 2015, 356, 58-71.	3.2	94
49	Requirement for Parp-1 and DNA ligases 1 or 3 but not of Xrcc1 in chromosomal translocation formation by backup end joining. Nucleic Acids Research, 2014, 42, 6380-6392.	6.5	84
50	Break-Induced Replication Repair of Damaged Forks Induces Genomic Duplications in Human Cells. Science, 2014, 343, 88-91.	6.0	387
51	Reduced contribution of thermally labile sugar lesions to DNA double strand break formation after exposure to heavy ions. Radiation Oncology, 2013, 8, 77.	1.2	19
52	Effects of chromatin decondensation on alternative NHEJ. DNA Repair, 2013, 12, 972-981.	1.3	5
53	DNA Double-Strand Break Repair as Determinant of Cellular Radiosensitivity to Killing and Target in Radiation Therapy. Frontiers in Oncology, 2013, 3, 113.	1.3	210
54	DNA double-strand-break complexity levels and their possible contributions to the probability for error-prone processing and repair pathway choice. Nucleic Acids Research, 2013, 41, 7589-7605.	6.5	229

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55	DNA Ligases I and III Cooperate in Alternative Non-Homologous End-Joining in Vertebrates. PLoS ONE, 2013, 8, e59505.	1.1	66
56	Functional redundancy between DNA ligases I and III in DNA replication in vertebrate cells. Nucleic Acids Research, 2012, 40, 2599-2610.	6.5	57
57	Conformational transitions of proteins engaged in DNA double-strand break repair, analysed by tryptophan fluorescence emission and FRET. Biochemical Journal, 2012, 443, 701-709.	1.7	6
58	Dependence of adaptive response and its bystander transmission on the genetic background of tested cells. International Journal of Radiation Biology, 2012, 88, 720-726.	1.0	12
59	Inhibition of B-NHEJ in Plateau-Phase Cells Is Not a Direct Consequence of Suppressed Growth Factor Signaling. International Journal of Radiation Oncology Biology Physics, 2012, 84, e237-e243.	0.4	20
60	Reduced Contribution of Thermally-Labile Sugar Lesions to DNA Double-Strand Break Formation after Exposure to Neutrons. Radiation Research, 2012, 178, 581.	0.7	7
61	Processing of DNA double strand breaks by alternative non-homologous end-joining in hyperacetylated chromatin. Genome Integrity, 2012, 3, 4.	1.0	13
62	The impact of serotonin on the development of bystander damage assessed by Î³-H2AX foci analysis. International Journal of Radiation Biology, 2012, 88, 777-780.	1.0	6
63	In Vitro Rejoining of Double Strand Breaks in Genomic DNA. Methods in Molecular Biology, 2012, 920, 471-484.	0.4	4
64	Analysis of Inhibition of DNA Replication in Irradiated Cells Using the SV40 Based In Vitro Assay of DNA Replication. Methods in Molecular Biology, 2012, 920, 591-602.	0.4	0
65	DNA Ligases I and III Cooperate to Mediate Alternative Non-homologous End-joining in Vertebrates. Qscience Proceedings, 2012, 2012, 61.	0.0	0
66	Induction and repair of DNA double strand breaks: The increasing spectrum of non-homologous end joining pathways. Mutation Research - Fundamental and Molecular Mechanisms of Mutagenesis, 2011, 711, 61-72.	0.4	326
67	Widespread Dependence of Backup NHEJ on Growth State: Ramifications for the Use of DNA-PK Inhibitors. International Journal of Radiation Oncology Biology Physics, 2011, 79, 540-548.	0.4	32
68	Post-irradiation chemical processing of DNA damage generates double-strand breaks in cells already engaged in repair. Nucleic Acids Research, 2011, 39, 8416-8429.	6.5	36
69	New partners for Chk1. Cell Cycle, 2010, 9, 2059-2062.	1.3	1
70	Evidence of an Adaptive Response Targeting DNA Nonhomologous End Joining and Its Transmission to Bystander Cells. Cancer Research, 2010, 70, 8498-8506.	0.4	38
71	Initial Characterization of a Low-Molecular-Weight Factor Enhancing the Checkpoint Response. Radiation Research, 2010, 174, 424.	0.7	0
72	The anatomy and cell cycle evolution of DNA damage signaling and repair foci. Cell Cycle, 2010, 9, 440-449.	1.3	1

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73	Extensive Repair of DNA Double-Strand Breaks in Cells Deficient in the DNA-PK-Dependent Pathway of NHEJ after Exclusion of Heat-Labile Sites. Radiation Research, 2009, 172, 152.	0.7	24
74	Backup pathways of NHEJ in cells of higher eukaryotes: Cell cycle dependence. Radiotherapy and Oncology, 2009, 92, 310-315.	0.3	167
75	Â-H2AX in recognition and signaling of DNA double-strand breaks in the context of chromatin. Nucleic Acids Research, 2008, 36, 5678-5694.	6.5	976
76	Repair of radiation induced DNA double strand breaks by backup NHEJ is enhanced in G2. DNA Repair, 2008, 7, 329-338.	1.3	114
77	DNA double strand break repair inhibition as a cause of heat radiosensitization: Re-evaluation considering backup pathways of NHEJ. International Journal of Hyperthermia, 2008, 24, 17-29.	1.1	48
78	Enhanced Use of Backup Pathways of NHEJ in G₂in Chinese Hamster Mutant Cells with Defects in the Classical Pathway of NHEJ. Radiation Research, 2008, 170, 512-520.	0.7	43
79	Histone H1 functions as a stimulatory factor in backup pathways of NHEJ. Nucleic Acids Research, 2008, 36, 1610-1623.	6.5	70
80	Homology-Directed Repair is Required for the Development of Radioresistance during S Phase: Interplay between Double-Strand Break Repair and Checkpoint Response. Radiation Research, 2007, 167, 1-11.	0.7	73
81	Low levels of DNA ligases III and IV sufficient for effective NHEJ. Journal of Cellular Physiology, 2007, 213, 475-483.	2.0	39
82	Marked Dependence on Growth State of Backup Pathways of NHEJ. International Journal of Radiation Oncology Biology Physics, 2007, 68, 1462-1470.	0.4	49
83	Backup Pathways of Nonhomologous End Joining May Have a Dominant Role in the Formation of Chromosome Aberrations. , 2007, , 67-85.		14
84	In Vitro Rejoining of Double-Strand Breaks in Genomic DNA. Methods in Molecular Biology, 2006, 314, 95-108.	0.4	2
85	PARP-1 and Ku compete for repair of DNA double strand breaks by distinct NHEJ pathways. Nucleic Acids Research, 2006, 34, 6170-6182.	6.5	692
86	Analysis of Inhibition of DNA Replication in Irradiated Cells Using the SV40-Based In Vitro Assay of DNA Replication. Methods in Molecular Biology, 2006, 314, 61-71.	0.4	0
87	Plasmid-Based Assays for DNA End-Joining In Vitro. Methods in Molecular Biology, 2006, 314, 123-131.	0.4	15
88	DNA Ligase III as a Candidate Component of Backup Pathways of Nonhomologous End Joining. Cancer Research, 2005, 65, 4020-4030.	0.4	289
89	Checkpoint Abrogation in G2 Compromises Repair of Chromosomal Breaks in Ataxia Telangiectasia Cells. Cancer Research, 2005, 65, 11292-11296.	0.4	64
90	ATR Affecting Cell Radiosensitivity Is Dependent on Homologous Recombination Repair but Independent of Nonhomologous End Joining. Cancer Research, 2004, 64, 7139-7143.	0.4	123

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91	Caffeine inhibits homology-directed repair of I-SceI-induced DNA double-strand breaks. Oncogene, 2004, 23, 824-834.	2.6	45
92	Backup pathways of NHEJ are suppressed by DNA-PK. Journal of Cellular Biochemistry, 2004, 92, 781-794.	1.2	119
93	DNA damage checkpoint control in cells exposed to ionizing radiation. Oncogene, 2003, 22, 5834-5847.	2.6	433
94	Caffeine Could Not Efficiently Sensitize Homologous Recombination Repair-Deficient Cells to Ionizing Radiation-Induced Killing. Radiation Research, 2003, 159, 420-425.	0.7	46
95	Caffeine-Induced Radiosensitization is Independent of Nonhomologous End Joining of DNA Double-Strand Breaks. Radiation Research, 2003, 159, 426-432.	0.7	43
96	An Overactivated ATR/CHK1 Pathway Is Responsible for the Prolonged G2 Accumulation in Irradiated AT Cells. Journal of Biological Chemistry, 2003, 278, 30869-30874.	1.6	65
97	Biochemical evidence for Ku-independent backup pathways of NHEJ. Nucleic Acids Research, 2003, 31, 5377-5388.	6.5	209
98	Ku affects the CHK1-dependent G(2) checkpoint after ionizing radiation. Cancer Research, 2002, 62, 6031-4.	0.4	19
99	Efficient rejoining of radiation-induced DNA double-strand breaks in vertebrate cells deficient in genes of the RAD52 epistasis group. Oncogene, 2001, 20, 2212-2224.	2.6	149
100	Regulation of DNA Replication after Heat Shock by Replication Protein A-Nucleolin Interactions. Journal of Biological Chemistry, 2001, 276, 20579-20588.	1.6	60
101	Homologous recombination as a potential target for caffeine radiosensitization in mammalian cells: reduced caffeine radiosensitization in XRCC2 and XRCC3 mutants. Oncogene, 2000, 19, 5788-5800.	2.6	89
102	Roles of Replication Protein A and DNA-dependent Protein Kinase in the Regulation of DNA Replication following DNA Damage. Journal of Biological Chemistry, 1999, 274, 22060-22064.	1.6	44
103	Ku70 Is Required for DNA Repair but Not for T Cell Antigen Receptor Gene Recombination In Vivo. Journal of Experimental Medicine, 1997, 186, 921-929.	4.2	249
104	Evidence for Activities Inhibiting In Trans Initiation of DNA Replication in Extract Prepared from Irradiated Cells. Radiation Research, 1996, 145, 408.	0.7	16
105	Regulation of DNA Replication in Irradiated Cells by Trans-Acting Factors. Radiation Research, 1995, 142, 169.	0.7	22
106	Loss of S-phase-dependent radioresistance in irs-1 cells exposed to X-rays. Mutation Research DNA Repair, 1994, 314, 77-85.	3.8	42
107	Evidence That the Product of the xrs Gene Is Predominantly Involved in the Repair of a Subset of Radiation-Induced Interphase Chromosome Breaks Rejoining with Fast Kinetics. Radiation Research, 1994, 138, 34.	0.7	15
108	Ionizing Radiation Induces Two Forms of Interphase Chromosome Breaks in Chinese Hamster Ovary Cells That Rejoin with Different Kinetics and Show Different Sensitivity to Treatment in Hypertonic Medium or Î²-araA. Radiation Research, 1993, 136, 262.	0.7	28

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109	Mechanism of Radiosensitization by Halogenated Pyrimidines: Effect of BrdU on Repair of DNA Breaks, Interphase Chromatin Breaks, and Potentially Lethal Damage in Plateau-Phase CHO Cells. Radiation Research, 1992, 129, 202.	0.7	25
110	Effects of 5â€²-iododeoxyuridine on the repair of radiation induced potentially lethal damage interphase chromatin breaks and DNA double strand breaks in chinese hamster ovary cells. International Journal of Radiation Oncology Biology Physics, 1992, 23, 353-360.	0.4	28
111	Mechanism of Radiosensitization by Halogenated Pyrimidines: Effect of BrdU on Cell Killing and Interphase Chromosome Breakage in Radiation-Sensitive Cells. Radiation Research, 1991, 125, 56.	0.7	21
112	Mechanism of Radiosensitization by Halogenated Pyrimidines: Bromodeoxyuridine and Î²-Arabinofuranosyladenine Affect Similar Subsets of Radiation-Induced Potentially Lethal Lesions in Plateau-Phase Chinese Hamster Ovary Cells. Radiation Research, 1991, 127, 45.	0.7	15
113	Mechanism of Radiosensitization by Halogenated Pyrimidines: Effect of BrdU on Radiation Induction of DNA and Chromosome Damage and Its Correlation with Cell Killing. Radiation Research, 1989, 119, 286.	0.7	55
114	Keynote address: Application of non-hypoxic cell sensitizers in radiobiology and radiotherapy: rationale and future prospects. International Journal of Radiation Oncology Biology Physics, 1989, 16, 1235-1241.	0.4	22
115	Comparative studies on repair inhibition by AraA, AraC and aphidicolin of radiation induced dna and chromosome damage in rodent cells: Comparison with fixation of PLD. International Journal of Radiation Oncology Biology Physics, 1989, 16, 1261-1265.	0.4	16
116	Linear DNA Elution Dose Response Curves Obtained in CHO Cells with Non-unwinding Filter Elution after Appropriate Selection of the Lysis Conditions. International Journal of Radiation Biology, 1989, 55, 569-581.	1.0	87
117	Radiosensitive Xrs-5 and Parental CHO Cells Show Identical DNA Neutral Filter Elution Doseâ€"response: Implications for a Relationship between Cell Radiosensitivity and Induction of DNA Double-strand Breaks. International Journal of Radiation Biology, 1988, 54, 55-62.	1.0	55
118	Variation through the Cell Cycle in the Dose-response of DNA Neutral Filter Elution in X-irradiated Synchronous CHO-cells. International Journal of Radiation Biology, 1988, 53, 729-747.	1.0	66
119	¹²⁵IdUrd-induced Chromosome Fragments, Assayed by Premature Chromosome Condensation, and DNA Double-strand Breaks Have Similar Repair Kinetics in G₁-phase CHO-cells. International Journal of Radiation Biology and Related Studies in Physics, Chemistry, and Medicine, 1987, 52, 705-722.	1.0	23
120	The Pathways of Double-Strand Break Repair. , 0, , .		1