Annapaola Franchitto

List of Publications by Year
in descending order

47	2,116	236925	243625
papers	citations	25 h-index	44 g-index

52	52	52	2322
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	R-Loop-Associated Genomic Instability and Implication of WRN and WRNIP1. International Journal of Molecular Sciences, 2022, 23, 1547.	4.1	8
2	Control of replication stress and mitosis in colorectal cancer stem cells through the interplay of PARP1, MRE11 and RAD51. Cell Death and Differentiation, 2021, 28, 2060-2082.	11.2	19
3	Checkpoint Defects Elicit a WRNIP1-Mediated Response to Counteract R-Loop-Associated Genomic Instability. Cancers, 2020, 12, 389.	3.7	11
4	Physiological and Pathological Roles of RAD52 at DNA Replication Forks. Cancers, 2020, 12, 402.	3.7	20
5	ATM pathway activation limits R-loop-associated genomic instability in Werner syndrome cells. Nucleic Acids Research, 2019, 47, 3485-3502.	14.5	43
6	RAD51 and mitotic function of mus81 are essential for recovery from low-dose of camptothecin in the absence of the WRN exonuclease. Nucleic Acids Research, 2019, 47, 6796-6810.	14.5	14
7	Rad52 prevents excessive replication fork reversal and protects from nascent strand degradation. Nature Communications, 2019, 10, 1412.	12.8	60
8	Inducible SMARCAL1 knockdown in iPSC reveals a link between replication stress and altered expression of master differentiation genes. DMM Disease Models and Mechanisms, 2019, 12, .	2.4	9
9	Phosphorylation by CK2 regulates MUS81/EME1 in mitosis and after replication stress. Nucleic Acids Research, 2018, 46, 5109-5124.	14.5	29
10	CSA and CSB play a role in the response to DNA breaks. Oncotarget, 2018, 9, 11581-11591.	1.8	23
11	Way out/way in: How the relationship between WRN and CDK1 may change the fate of collapsed replication forks. Molecular and Cellular Oncology, 2017, 4, e1268243.	0.7	7
12	SLX4 Prevents GEN1-Dependent DSBs During DNA Replication Arrest Under Pathological Conditions in Human Cells. Scientific Reports, 2017, 7, 44464.	3.3	13
13	Crosstalk between mismatch repair and base excision repair in human gastric cancer. Oncotarget, 2017, 8, 84827-84840.	1.8	13
14	WRNIP1: A new guardian of genome integrity at stalled replication forks. Molecular and Cellular Oncology, 2016, 3, e1215777.	0.7	3
15	<scp>WRNIP</scp> 1 protects stalled forks from degradation and promotes fork restart after replication stress. EMBO Journal, 2016, 35, 1437-1451.	7.8	78
16	CDK1 phosphorylates WRN at collapsed replication forks. Nature Communications, 2016, 7, 12880.	12.8	48
17	The WRN exonuclease domain protects nascent strands from pathological MRE11/EXO1-dependent degradation. Nucleic Acids Research, 2015, 43, gkv836.	14.5	67
18	Checkpoint-dependent and independent roles of the Werner syndrome protein in preserving genome integrity in response to mild replication stress. Nucleic Acids Research, 2014, 42, 12628-12639.	14.5	30

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19	Replication fork recovery and regulation of common fragile sites stability. Cellular and Molecular Life Sciences, 2014, 71, 4507-4517.	5.4	25
20	The WRN and MUS81 proteins limit cell death and genome instability following oncogene activation. Oncogene, 2013, 32, 610-620.	5.9	40
21	Genome Instability at Common Fragile Sites: Searching for the Cause of Their Instability. BioMed Research International, 2013, 2013, 1-9.	1.9	22
22	Survival of the Replication Checkpoint Deficient Cells Requires MUS81-RAD52 Function. PLoS Genetics, 2013, 9, e1003910.	3.5	68
23	The RAD9â€“RAD1â€“HUS1 (9.1.1) complex interacts with WRN and is crucial to regulate its response to replication fork stalling. Oncogene, 2012, 31, 2809-2823.	5.9	26
24	Perturbed replication induced genome wide or at common fragile sites is differently managed in the absence of WRN. Carcinogenesis, 2012, 33, 1655-1663.	2.8	47
25	Understanding the molecular basis of common fragile sites instability: Role of the proteins involved in the recovery of stalled replication forks. Cell Cycle, 2011, 10, 4039-4046.	2.6	23
26	The Werner syndrome protein: linking the replication checkpoint response to genome stability. Aging, 2011, 3, 311-318.	3.1	51
27	Che-1 Promotes Tumor Cell Survival by Sustaining Mutant p53 Transcription and Inhibiting DNA Damage Response Activation. Cancer Cell, 2010, 18, 122-134.	16.8	45
28	ATR and ATM differently regulate WRN to prevent DSBs at stalled replication forks and promote replication fork recovery. EMBO Journal, 2010, 29, 3156-3169.	7.8	145
29	Werner syndrome helicase activity is essential in maintaining fragile site stability. Journal of Cell Biology, 2008, 180, 305-314.	5.2	103
30	Replication fork stalling in WRN-deficient cells is overcome by prompt activation of a MUS81-dependent pathway. Journal of Cell Biology, 2008, 183, 241-252.	5.2	100
31	Terminally differentiated muscle cells are defective in base excision DNA repair and hypersensitive to oxygen injury. Proceedings of the National Academy of Sciences of the United States of America, 2007, 104, 17010-17015.	7.1	106
32	RecQ helicases and topoisomerases: implications for genome stability in humans. Italian Journal of Biochemistry, 2007, 56, 115-21.	0.3	2
33	Replication-Dependent DNA Damage Response Triggered by Roscovitine Induces an Uncoupling of DNA Replication Proteins. Cell Cycle, 2006, 5, 2153-2159.	2.6	12
34	Werner Syndrome Protein and the MRE11 Complex are Involved in a Common Pathway of Replication Fork Recovery. Cell Cycle, 2004, 3, 1331-1339.	2.6	38
35	BLM and the FANC proteins collaborate in a common pathway in response to stalled replication forks. EMBO Journal, 2004, 23, 3154-3163.	7.8	115
36	Werner syndrome protein, the MRE11 complex and ATR: menage-Ã-trois in guarding genome stability during DNA replication?. BioEssays, 2004, 26, 306-313.	2.5	32

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37	Werner's syndrome protein is phosphorylated in an ATR/ATM-dependent manner following replication arrest and DNA damage induced during the S phase of the cell cycle. Oncogene, 2003, 22, 1491-1500.	5.9	115
38	The mammalian mismatch repair protein MSH2 is required for correct MRE11 and RAD51 relocalization and for efficient cell cycle arrest induced by ionizing radiation in G2 phase. Oncogene, 2003, 22, 2110-2120.	5.9	93
39	The G2-phase decatenation checkpoint is defective in Werner syndrome cells. Cancer Research, 2003, 63, 3289-95.	0.9	38
40	Protecting genomic integrity during DNA replication: correlation between Werner's and Bloom's syndrome gene products and the MRE11 complex. Human Molecular Genetics, 2002, 11, 2447-2453.	2.9	33
41	Bloom's syndrome protein is required for correct relocalization of RAD50/MRE11/NBS1 complex after replication fork arrest. Journal of Cell Biology, 2002, 157, 19-30.	5.2	115
42	Investigation of G2-phase chromosomal radiosensitivity in hereditary non-polyposis colorectal cancer cells. International Journal of Radiation Biology, 2001, 77, 773-780.	1.8	5
43	Werner's Syndrome Protein Is Required for Correct Recovery after Replication Arrest and DNA Damage Induced in S-Phase of Cell Cycle. Molecular Biology of the Cell, 2001, 12, 2412-2421.	2.1	135
44	Hypersensitivity to camptothecin in MSH2 deficient cells is correlated with a role for MSH2 protein in recombinational repair. Carcinogenesis, 2001, 22, 1781-1787.	2.8	38
45	Evidence that camptothecin-induced aberrations in the G2 phase of cell cycle of Chinese hamster ovary (CHO) cell lines is associated with transcription. Mutation Research - Fundamental and Molecular Mechanisms of Mutagenesis, 2000, 452, 189-195.	1.0	14
46	Werner's syndrome lymphoblastoid cells are hypersensitive to topoisomerase II inhibitors in the G2 phase of the cell cycle. Mutation Research DNA Repair, 2000, 459, 123-133.	3.7	33
47	RAD52 Prevents Excessive Replication Fork Reversal and Protects from Nascent Strand Degradation. SSRN Electronic Journal, 0, , .	0.4	0