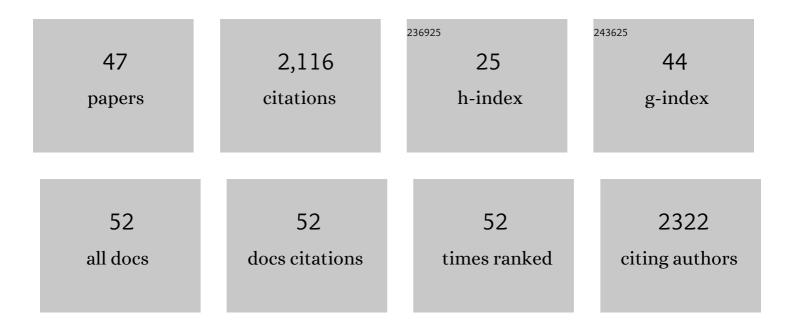
## Annapaola Franchitto

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	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
2	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
3	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
4	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
5	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
6	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
7	Werner syndrome helicase activity is essential in maintaining fragile site stability. Journal of Cell Biology, 2008, 180, 305-314.	5.2	103
8	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
9	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
10	<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
11	Survival of the Replication Checkpoint Deficient Cells Requires MUS81-RAD52 Function. PLoS Genetics, 2013, 9, e1003910.	3.5	68
12	The WRN exonuclease domain protects nascent strands from pathological MRE11/EXO1-dependent degradation. Nucleic Acids Research, 2015, 43, gkv836.	14.5	67
13	Rad52 prevents excessive replication fork reversal and protects from nascent strand degradation. Nature Communications, 2019, 10, 1412.	12.8	60
14	The Werner syndrome protein: linking the replication checkpoint response to genome stability. Aging, 2011, 3, 311-318.	3.1	51
15	CDK1 phosphorylates WRN at collapsed replication forks. Nature Communications, 2016, 7, 12880.	12.8	48
16	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
17	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
18	ATM pathway activation limits R-loop-associated genomic instability in Werner syndrome cells. Nucleic Acids Research. 2019. 47. 3485-3502.	14.5	43

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19	The WRN and MUS81 proteins limit cell death and genome instability following oncogene activation. Oncogene, 2013, 32, 610-620.	5.9	40
20	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
21	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
22	The G2-phase decatenation checkpoint is defective in Werner syndrome cells. Cancer Research, 2003, 63, 3289-95.	0.9	38
23	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
24	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
25	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
26	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
27	Phosphorylation by CK2 regulates MUS81/EME1 in mitosis and after replication stress. Nucleic Acids Research, 2018, 46, 5109-5124.	14.5	29
28	The RAD9–RAD1–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
29	Replication fork recovery and regulation of common fragile sites stability. Cellular and Molecular Life Sciences, 2014, 71, 4507-4517.	5.4	25
30	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
31	CSA and CSB play a role in the response to DNA breaks. Oncotarget, 2018, 9, 11581-11591.	1.8	23
32	Genome Instability at Common Fragile Sites: Searching for the Cause of Their Instability. BioMed Research International, 2013, 2013, 1-9.	1.9	22
33	Physiological and Pathological Roles of RAD52 at DNA Replication Forks. Cancers, 2020, 12, 402.	3.7	20
34	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
35	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
36	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

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37	SLX4 Prevents GEN1-Dependent DSBs During DNA Replication Arrest Under Pathological Conditions in Human Cells. Scientific Reports, 2017, 7, 44464.	3.3	13
38	Crosstalk between mismatch repair and base excision repair in human gastric cancer. Oncotarget, 2017, 8, 84827-84840.	1.8	13
39	Replication-Dependent DNA Damage Response Triggered by Roscovitine Induces an Uncoupling of DNA Replication Proteins. Cell Cycle, 2006, 5, 2153-2159.	2.6	12
40	Checkpoint Defects Elicit a WRNIP1-Mediated Response to Counteract R-Loop-Associated Genomic Instability. Cancers, 2020, 12, 389.	3.7	11
41	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
42	R-Loop-Associated Genomic Instability and Implication of WRN and WRNIP1. International Journal of Molecular Sciences, 2022, 23, 1547.	4.1	8
43	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
44	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
45	WRNIP1: A new guardian of genome integrity at stalled replication forks. Molecular and Cellular Oncology, 2016, 3, e1215777.	0.7	3
46	RecQ helicases and topoisomerases: implications for genome stability in humans. Italian Journal of Biochemistry, 2007, 56, 115-21.	0.3	2
47	RAD52 Prevents Excessive Replication Fork Reversal and Protects from Nascent Strand Degradation. SSRN Electronic Journal, 0, , .	0.4	0