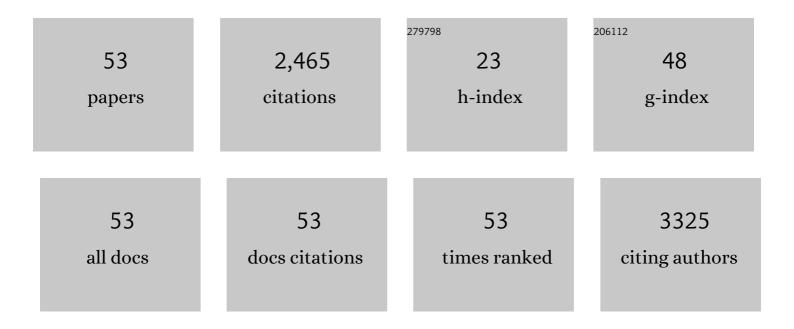


## List of Publications by Year in descending order

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Δτζε Τ Πλς

#	Article	IF	CITATIONS
1	Variations in the Abortive HIV-1 RNA Hairpin Do Not Impede Viral Sensing and Innate Immune Responses. Pathogens, 2021, 10, 897.	2.8	1
2	Transient CRISPR-Cas Treatment Can Prevent Reactivation of HIV-1 Replication in a Latently Infected T-Cell Line. Viruses, 2021, 13, 2461.	3.3	6
3	CRISPR-Cas9 Dual-gRNA Attack Causes Mutation, Excision and Inversion of the HIV-1 Proviral DNA. Viruses, 2020, 12, 330.	3.3	21
4	Extinction of all infectious HIV in cell culture by the CRISPR-Cas12a system with only a single crRNA. Nucleic Acids Research, 2020, 48, 5527-5539.	14.5	56
5	On the generation of the MSD- $ ilde{N}^\circ$ class of defective HIV proviruses. Retrovirology, 2019, 16, 19.	2.0	11
6	Interferon-inducible TRIM22 contributes to maintenance of HIV-1 proviral latency in T cell lines. Virus Research, 2019, 269, 197631.	2.2	10
7	IFI16 Targets the Transcription Factor Sp1 to Suppress HIV-1 Transcription and Latency Reactivation. Cell Host and Microbe, 2019, 25, 858-872.e13.	11.0	83
8	The Impact of HIV-1 Genetic Diversity on CRISPR-Cas9 Antiviral Activity and Viral Escape. Viruses, 2019, 11, 255.	3.3	31
9	How Polypurine Tract Changes in the HIV-1 RNA Genome Can Cause Resistance against the Integrase Inhibitor Dolutegravir. MBio, 2018, 9, .	4.1	13
10	CRISPR-Cas based antiviral strategies against HIV-1. Virus Research, 2018, 244, 321-332.	2.2	69
11	Tackling HIV Persistence: Pharmacological versus CRISPR-Based Shock Strategies. Viruses, 2018, 10, 157.	3.3	23
12	The HIV-1 Tat Protein Enhances Splicing at the Major Splice Donor Site. Journal of Virology, 2018, 92, .	3.4	23
13	Combinatorial CRISPR-Cas9 and RNA Interference Attack on HIV-1 DNA and RNA Can Lead to Cross-Resistance. Antimicrobial Agents and Chemotherapy, 2017, 61, .	3.2	15
14	Establishment of a mouse xenograft model of metastatic adrenocortical carcinoma. Oncotarget, 2017, 8, 51050-51057.	1.8	9
15	A Phylogenetic Survey on the Structure of the HIV-1 Leader RNA Domain That Encodes the Splice Donor Signal. Viruses, 2016, 8, 200.	3.3	7
16	Tet-On Systems For Doxycycline-inducible Gene Expression. Current Gene Therapy, 2016, 16, 156-167.	2.0	253
17	A regulatable AAV vector mediating GDNF biological effects at clinically-approved sub-antimicrobial doxycycline doses. Molecular Therapy - Methods and Clinical Development, 2016, 3, 16027.	4.1	32
18	A Combinatorial CRISPR-Cas9 Attack on HIV-1 DNA Extinguishes All Infectious Provirus in Infected T Cell Cultures. Cell Reports, 2016, 17, 2819-2826.	6.4	112

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#	Article	IF	CITATIONS
19	Construction of Nef-positive doxycycline-dependent HIV-1 variants using bicistronic expression elements. Virology, 2016, 488, 96-107.	2.4	2
20	Selecting the optimal Tetâ€On system for doxycyclineâ€inducible gene expression in transiently transfected and stably transduced mammalian cells. Biotechnology Journal, 2016, 11, 71-79.	3.5	21
21	CRISPR/Cas9: a double-edged sword when used to combat HIV infection. Retrovirology, 2016, 13, 37.	2.0	52
22	CRISPR-Cas9 Can Inhibit HIV-1 Replication but NHEJ Repair Facilitates Virus Escape. Molecular Therapy, 2016, 24, 522-526.	8.2	190
23	Tat-dependent production of an HIV-1 TAR-encoded miRNA-like small RNA. Nucleic Acids Research, 2016, 44, 4340-4353.	14.5	38
24	Conditionally replicating HIV and SIV variants. Virus Research, 2016, 216, 66-75.	2.2	5
25	Role of Occult and Post-acute Phase Replication in Protective Immunity Induced with a Novel Live Attenuated SIV Vaccine. PLoS Pathogens, 2016, 12, e1006083.	4.7	9
26	On the primer binding site mutation that appears and disappears during HIV and SIV replication. Retrovirology, 2015, 12, 75.	2.0	3
27	HIV-1 transcriptional silencing caused by TRIM22 inhibition of Sp1 binding to the viral promoter. Retrovirology, 2015, 12, 104.	2.0	62
28	HIV-1 splicing is controlled by local RNA structure and binding of splicing regulatory proteins at the major 5′ splice site. Journal of General Virology, 2015, 96, 1906-1917.	2.9	18
29	The HIV-1 leader RNA is exquisitely sensitive to structural changes. Virology, 2015, 483, 236-252.	2.4	14
30	Human immunodeficiency virus type 1 splicing at the major splice donor site is controlled by highly conserved RNA sequence and structural elements. Journal of General Virology, 2015, 96, 3389-3395.	2.9	9
31	A Short Sequence Motif in the 5′ Leader of the HIV-1 Genome Modulates Extended RNA Dimer Formation and Virus Replication. Journal of Biological Chemistry, 2014, 289, 35061-35074.	3.4	18
32	Partial Protection by Vaccination Does Not Prevent Chronic Neuro-inflammation in an SIV Model. AIDS Research and Human Retroviruses, 2014, 30, A284-A284.	1.1	1
33	HIV-1 splicing at the major splice donor site is restricted by RNA structure. Virology, 2014, 468-470, 609-620.	2.4	27
34	Targeted sequencing by proximity ligation for comprehensive variant detection and local haplotyping. Nature Biotechnology, 2014, 32, 1019-1025.	17.5	231
35	Retroviral microRNAs. Current Opinion in Virology, 2014, 7, 47-54.	5.4	41
36	The Allosteric HIV-1 Integrase Inhibitor BI-D Affects Virion Maturation but Does Not Influence Packaging of a Functional RNA Genome. PLoS ONE, 2014, 9, e103552.	2.5	22

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37	Tat has a dual role in simian immunodeficiency virus transcription. Journal of General Virology, 2012, 93, 2279-2289.	2.9	6
38	Opening of the TAR hairpin in the HIV-1 genome causes aberrant RNA dimerization and packaging. Retrovirology, 2012, 9, 59.	2.0	26
39	The HIV-1 Tat Protein Has a Versatile Role in Activating Viral Transcription. Journal of Virology, 2011, 85, 9506-9516.	3.4	86
40	HIV-1 evolution: frustrating therapies, but disclosing molecular mechanisms. Philosophical Transactions of the Royal Society B: Biological Sciences, 2010, 365, 1965-1973.	4.0	16
41	Construction of a Doxycycline-Dependent Simian Immunodeficiency Virus Reveals a Nontranscriptional Function of Tat in Viral Replication. Journal of Virology, 2007, 81, 11159-11169.	3.4	25
42	The Genetic Stability of a Conditional Live HIV-1 Variant Can Be Improved by Mutations in the Tet-On Regulatory System That Restrain Evolution. Journal of Biological Chemistry, 2006, 281, 17084-17091.	3.4	25
43	Alternative tRNA Priming of Human Immunodeficiency Virus Type 1 Reverse Transcription Explains Sequence Variation in the Primer-Binding Site That Has Been Attributed to APOBEC3G Activity. Journal of Virology, 2005, 79, 3179-3181.	3.4	17
44	Improving the Safety of a Conditional-Live Human Immunodeficiency Virus Type 1 Vaccine by Controlling both Gene Expression and Cell Entry. Journal of Virology, 2005, 79, 3855-3858.	3.4	21
45	A Conditionally Replicating Virus as a Novel Approach Toward an HIV Vaccine. Methods in Enzymology, 2004, 388, 359-379.	1.0	30
46	Human Immunodeficiency Virus Type 1 Escapes from RNA Interference-Mediated Inhibition. Journal of Virology, 2004, 78, 2601-2605.	3.4	426
47	Viral Evolution as a Tool to Improve the Tetracycline-regulated Gene Expression System. Journal of Biological Chemistry, 2004, 279, 18776-18782.	3.4	111
48	Conditional live virus as a novel approach towards a safe live attenuated HIV vaccine. Expert Review of Vaccines, 2002, 1, 293-301.	4.4	16
49	Conditional Virus Replication as an Approach to a Safe Live Attenuated Human Immunodeficiency Virus Vaccine. Journal of NeuroVirology, 2002, 8, 134-137.	2.1	9
50	HIV-1 RNA Editing, Hypermutation, and Error-Prone Reverse Transcription. Science, 2001, 292, 7a-7.	12.6	35
51	Inhibition of polyadenylation by stable RNA secondary structure. Nucleic Acids Research, 1998, 26, 1870-1876.	14.5	48
52	Regulation of Glutamate Dehydrogenase Expression in the Developing Rat Liver. Control at Different Levels in the Prenatal Period. FEBS Journal, 1996, 235, 677-682.	0.2	11
53	Human immunodeficiency virus uses tRNALys,3as primer for reverse transcription in HeLa-CD4+cells. FEBS Letters, 1994, 341, 49-53.	2.8	19