Nicolas Sluis-Cremer

List of Publications by Year in descending order

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69 papers

2,350 citations

172457 29 h-index 214800 47 g-index

70 all docs

70 docs citations

70 times ranked 2810 citing authors

#	Article	IF	CITATIONS
1	Relative domain orientation of the <scp>L289K HIV</scp> â€1 reverse transcriptase monomer. Protein Science, 2022, 31, e4307.	7.6	1
2	B Lymphocytes, but Not Dendritic Cells, Efficiently HIV-1 <i>Trans</i> Infect Naive CD4 ⁺ T Cells: Implications for the Viral Reservoir. MBio, 2021, 12, .	4.1	5
3	Mutations in the HIV-1 3′-Polypurine Tract and Integrase Strand Transfer Inhibitor Resistance. Antimicrobial Agents and Chemotherapy, 2021, 65, .	3.2	14
4	Retroviral RNase H: Structure, mechanism, and inhibition. The Enzymes, 2021, 50, 227-247.	1.7	4
5	Retroviral reverse transcriptase: Structure, function and inhibition. The Enzymes, 2021, 50, 179-194.	1.7	1
6	Large Multidomain Protein NMR: HIV-1 Reverse Transcriptase Precursor in Solution. International Journal of Molecular Sciences, 2020, 21, 9545.	4.1	1
7	Structural Basis of Reduced Susceptibility to Ceftazidime-Avibactam and Cefiderocol in <i>Enterobacter cloacae</i> Due to AmpC R2 Loop Deletion. Antimicrobial Agents and Chemotherapy, 2020, 64, .	3.2	51
8	Peptides Mimicking the β7/β8 Loop of HIV-1 Reverse Transcriptase p51 as "Hotspot-Targeted―Dimerization Inhibitors. ACS Medicinal Chemistry Letters, 2020, 11, 811-817.	2.8	8
9	Nonnucleoside Reverse Transcriptase Inhibitor Hypersusceptibility and Resistance by Mutation of Residue 181 in HIV-1 Reverse Transcriptase. Antimicrobial Agents and Chemotherapy, 2019, 63, .	3.2	4
10	Type 1-programmed dendritic cells drive antigen-specific latency reversal and immune elimination of persistent HIV-1. EBioMedicine, 2019, 43, 295-306.	6.1	20
11	Naive CD4+ T Cells Harbor a Large Inducible Reservoir of Latent, Replication-competent Human Immunodeficiency Virus Type 1. Clinical Infectious Diseases, 2019, 69, 1919-1925.	5.8	63
12	Inhibitors of Signaling Pathways That Block Reversal of HIV-1 Latency. Antimicrobial Agents and Chemotherapy, 2019, 63, .	3.2	17
13	Small-Molecule Inhibitor of FosA Expands Fosfomycin Activity to Multidrug-Resistant Gram-Negative Pathogens. Antimicrobial Agents and Chemotherapy, 2019, 63, .	3.2	11
14	Origin of the plasmid-mediated fosfomycin resistance gene fosA3. Journal of Antimicrobial Chemotherapy, 2018, 73, 373-376.	3.0	27
15	Future of nonnucleoside reverse transcriptase inhibitors. Proceedings of the National Academy of Sciences of the United States of America, 2018, 115, 637-638.	7.1	14
16	<i>In Vitro</i> Cross-Resistance Profiles of Rilpivirine, Dapivirine, and MIV-150, Nonnucleoside Reverse Transcriptase Inhibitor Microbicides in Clinical Development for the Prevention of HIV-1 Infection. Antimicrobial Agents and Chemotherapy, 2017, 61, .	3.2	13
17	HIV-1 Resistance to the Nonnucleoside Reverse Transcriptase Inhibitors. , 2017, , 521-533.		0
18	Novel assay reveals a large, inducible, replication-competent HIV-1 reservoir in resting CD4+ T cells. Nature Medicine, 2017, 23, 885-889.	30.7	68

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19	Nonnucleoside Reverse Transcriptase Inhibitors Reduce HIV-1 Production from Latently Infected Resting CD4 ⁺ T Cells following Latency Reversal. Antimicrobial Agents and Chemotherapy, 2017, 61, .	3.2	11
20	Inhibition of Fosfomycin Resistance Protein FosA by Phosphonoformate (Foscarnet) in Multidrug-Resistant Gram-Negative Pathogens. Antimicrobial Agents and Chemotherapy, 2017, 61, .	3.2	18
21	Structure and Dynamics of FosA-Mediated Fosfomycin Resistance in Klebsiella pneumoniae and Escherichia coli. Antimicrobial Agents and Chemotherapy, 2017, 61, .	3.2	28
22	Widespread Fosfomycin Resistance in Gram-Negative Bacteria Attributable to the Chromosomal <i>fosA</i> fosA	4.1	138
23	Establishment and Reversal of HIV-1 Latency in Naive and Central Memory CD4 ⁺ T Cells <i>In Vitro</i> . Journal of Virology, 2016, 90, 8059-8073.	3.4	37
24	Glutathione- <i>S</i> -transferase FosA6 of <i>Klebsiella pneumoniae</i> origin conferring fosfomycin resistance in ESBL-producing <i>Escherichia coli</i> Journal of Antimicrobial Chemotherapy, 2016, 71, 2460-2465.	3.0	49
25	Temporal transcriptional response to latency reversing agents identifies specific factors regulating HIV-1 viral transcriptional switch. Retrovirology, 2015, 12, 85.	2.0	14
26	Identification of mechanistically distinct inhibitors of HIV-1 reverse transcriptase through fragment screening. Proceedings of the National Academy of Sciences of the United States of America, 2015, 112, 6979-6984.	7.1	22
27	Silent mutations at codons 65 and 66 in reverse transcriptase alleviate indel formation and restore fitness in subtype B HIV-1 containing D67N and K70R drug resistance mutations. Nucleic Acids Research, 2015, 43, 3256-3271.	14.5	9
28	Resistance to reverse transcriptase inhibitors used in the treatment and prevention of HIV-1 infection. Future Microbiology, 2015, 10, 1773-1782.	2.0	34
29	Therapeutic Approaches to Eradicate Latent HIV-1 in Resting CD4+ T Cells. Current Topics in Medicinal Chemistry, 2015, 16, 1191-1197.	2.1	3
30	Competitive Fitness Assays Indicate that the E138A Substitution in HIV-1 Reverse Transcriptase DecreasesIn VitroSusceptibility to Emtricitabine. Antimicrobial Agents and Chemotherapy, 2014, 58, 2430-2433.	3.2	8
31	The Emerging Profile of Cross-Resistance among the Nonnucleoside HIV-1 Reverse Transcriptase Inhibitors. Viruses, 2014, 6, 2960-2973.	3.3	51
32	Mechanism of allosteric inhibition of HIV-1 reverse transcriptase revealed by single-molecule and ensemble fluorescence. Nucleic Acids Research, 2014, 42, 11687-11696.	14.5	43
33	E138A in HIV-1 reverse transcriptase is more common in subtype C than B: Implications for rilpivirine use in resource-limited settings. Antiviral Research, 2014, 107, 31-34.	4.1	60
34	Molecular mechanism of HIV-1 resistance to 3′-azido-2′,3′-dideoxyguanosine. Antiviral Research, 2014, 162-67.	101,	3
35	Novel high-throughput screen identifies an HIV-1 reverse transcriptase inhibitor with a unique mechanism of action. Biochemical Journal, 2014, 462, 425-432.	3.7	2
36	Discovery of a Small Molecule Agonist of Phosphatidylinositol 3-Kinase p110 \hat{l}_{\pm} That Reactivates Latent HIV-1. PLoS ONE, 2014, 9, e84964.	2.5	21

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37	Biophysical Insights into the Inhibitory Mechanism of Non-Nucleoside HIV-1 Reverse Transcriptase Inhibitors. Biomolecules, 2013, 3, 889-904.	4.0	5
38	Transient kinetic analyses of the ribonuclease H cleavage activity of HIV-1 reverse transcriptase in complex with efavirenz and/or a \hat{I}^2 -thujaplicinol analogue. Biochemical Journal, 2013, 455, 179-184.	3.7	10
39	Replication Fitness of Multiple Nonnucleoside Reverse Transcriptase-Resistant HIV-1 Variants in the Presence of Etravirine Measured by 454 Deep Sequencing. Journal of Virology, 2013, 87, 8805-8807.	3.4	9
40	Frequent Emergence of N348I in HIV-1 Subtype C Reverse Transcriptase with Failure of Initial Therapy Reduces Susceptibility to Reverse-Transcriptase Inhibitors. Clinical Infectious Diseases, 2012, 55, 737-745.	5.8	37
41	Substrate mimicry: HIV-1 reverse transcriptase recognizes 6-modified-3'-azido-2',3'-dideoxyguanosine-5'-triphosphates as adenosine analogs. Nucleic Acids Research, 2012, 40, 381-390.	14.5	4
42	Zidovudine (AZT) Monotherapy Selects for the A360V Mutation in the Connection Domain of HIV-1 Reverse Transcriptase. PLoS ONE, 2012, 7, e31558.	2.5	12
43	Synthesis, antiviral activity, cytotoxicity and cellular pharmacology of l-3′-azido-2′,3′-dideoxypurine nucleosides. European Journal of Medicinal Chemistry, 2011, 46, 3832-3844.	5 . 5	12
44	Inhibitors of Histone Deacetylases. Journal of Biological Chemistry, 2011, 286, 22211-22218.	3.4	129
45	The Base Component of 3′-Azido-2′,3′-Dideoxynucleosides Influences Resistance Mutations Selected in HIV-1 Reverse Transcriptase. Antimicrobial Agents and Chemotherapy, 2011, 55, 3758-3764.	3.2	1
46	N348I in HIV-1 reverse transcriptase decreases susceptibility to tenofovir and etravirine in combination with other resistance mutations. Aids, 2010, 24, 317-319.	2.2	22
47	N348I in reverse transcriptase provides a genetic pathway for HIV-1 to select thymidine analogue mutations and mutations antagonistic to thymidine analogue mutations. Aids, 2010, 24, 659-667.	2.2	21
48	Synthesis and Anti-HIV-1 Activity of a Novel Series of Aminoimidazole Analogs. Letters in Drug Design and Discovery, 2010, 7, 318-323.	0.7	5
49	Anti-Human Immunodeficiency Virus Activity, Cross-Resistance, Cytotoxicity, and Intracellular Pharmacology of the 3′-Azido-2′,3′-Dideoxypurine Nucleosides. Antimicrobial Agents and Chemotherapy, 2009, 53, 3715-3719.	3.2	19
50	Mechanisms of inhibition of HIV replication by non-nucleoside reverse transcriptase inhibitors. Virus Research, 2008, 134, 147-156.	2,2	135
51	Mechanism by which a Glutamine to Leucine Substitution at Residue 509 in the Ribonuclease H Domain of HIV-1 Reverse Transcriptase Confers Zidovudine Resistance. Biochemistry, 2008, 47, 14020-14027.	2.5	30
52	Efavirenz Accelerates HIV-1 Reverse Transcriptase Ribonuclease H Cleavage, Leading to Diminished Zidovudine Excision. Molecular Pharmacology, 2008, 73, 601-606.	2.3	57
53	Molecular mechanisms of bidirectional antagonism between K65R and thymidine analog mutations in HIV-1 reverse transcriptase. Aids, 2007, 21, 1405-1414.	2.2	68
54	Molecular Mechanism by Which the K70E Mutation in Human Immunodeficiency Virus Type 1 Reverse Transcriptase Confers Resistance to Nucleoside Reverse Transcriptase Inhibitors. Antimicrobial Agents and Chemotherapy, 2007, 51, 48-53.	3.2	68

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55	Selection of Mutations in the Connection and RNase H Domains of Human Immunodeficiency Virus Type 1 Reverse Transcriptase That Increase Resistance to 3′-Azido-3′-Dideoxythymidine. Journal of Virology, 2007, 81, 7852-7859.	3.4	79
56	Probing nonnucleoside inhibitorâ€induced activeâ€site distortion in HIVâ€1 reverse transcriptase by transient kinetic analyses. Protein Science, 2007, 16, 1728-1737.	7.6	59
57	N348I in the Connection Domain of HIV-1 Reverse Transcriptase Confers Zidovudine and Nevirapine Resistance. PLoS Medicine, 2007, 4, e335.	8.4	151
58	Radiation Target Analyses of DNA Template/Primer Complexes. Biophysical Journal, 2006, 90, L61-L63.	0.5	1
59	Structureâ^'Activity Relationships of [2 ,5 -Bis-O-(tert-butyldimethylsilyl)-β-d-ribofuranosyl]-3 -spiro-5   -(4   -amino-1   ,2   -oxathiole-2   ,2   -diox Transcriptase Dimerization. Journal of Medicinal Chemistry, 2006, 49, 4834-4841.	kidæ)thymi	n&⊉erivati <mark>v∈</mark>
60	Potent Nonnucleoside Reverse Transcriptase Inhibitors Target HIV-1 Gag-Pol. PLoS Pathogens, 2006, 2, e119.	4.7	95
61	The 3′-Azido Group Is Not the Primary Determinant of 3′-Azido-3′-deoxythymidine (AZT) Responsible for the Excision Phenotype of AZT-resistant HIV-1. Journal of Biological Chemistry, 2005, 280, 29047-29052.	3.4	38
62	Efavirenz enhances the proteolytic processing of an HIV-1 pol polyprotein precursor and reverse transcriptase homodimer formation. FEBS Letters, 2005, 579, 379-384.	2.8	46
63	Conformational Changes in HIV-1 Reverse Transcriptase Induced by Nonnucleoside Reverse Transcriptase Inhibitor Binding. Current HIV Research, 2004, 2, 323-332.	0.5	133
64	Proteolytic processing of an HIV-1 pol polyprotein precursor: insights into the mechanism of reverse transcriptase p66/p51 heterodimer formation. International Journal of Biochemistry and Cell Biology, 2004, 36, 1836-1847.	2.8	52
65	Structure-activity relationships in HIV-1 reverse transcriptase revealed by radiation target analysis. Protein Science, 2003, 12, 2081-2086.	7.6	7
66	Modulation of the oligomeric structures of HIV-1 retroviral enzymes by synthetic peptides and small molecules. FEBS Journal, 2002, 269, 5103-5111.	0.2	45
67	Mutational analysis of Lys65 of HIV-1 reverse transcriptase. Biochemical Journal, 2000, 348, 77-82.	3.7	63
68	Mutational analysis of Lys65 of HIV-1 reverse transcriptase. Biochemical Journal, 2000, 348, 77.	3.7	29
69	Toward a Functional Cure for HIV-1 Infection: The Block and Lock Therapeutic Approach. Frontiers in Virology, 0, 2, .	1.4	3