Narendra M Dixit

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Modelling how ribavirin improves interferon response rates in hepatitis C virus infection. Nature, 2004, 432, 922-924.	13.7	344
2	Complex patterns of viral load decay under antiretroviral therapy: influence of pharmacokinetics and intracellular delay. Journal of Theoretical Biology, 2004, 226, 95-109.	0.8	163
3	Targeting TMPRSS2 and Cathepsin B/L together may be synergistic against SARS-CoV-2 infection. PLoS Computational Biology, 2020, 16, e1008461.	1.5	106
4	HIV dynamics with multiple infections of target cells. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 8198-8203.	3.3	94
5	Estimates of Intracellular Delay and Average Drug Efficacy from Viral Load Data of HIV-Infected Individuals under Antiretroviral Therapy. Antiviral Therapy, 2004, 9, 237-246.	0.6	79
6	Recombination increases human immunodeficiency virus fitness, but not necessarily diversity. Journal of General Virology, 2008, 89, 1467-1477.	1.3	57
7	Modeling Viral and Drug Kinetics: Hepatitis C Virus Treatment with Pegylated Interferon Alfa-2b. Seminars in Liver Disease, 2003, 23, 013-018.	1.8	47
8	The two-component signalling networks of <i>Mycobacterium tuberculosis</i> display extensive cross-talk <i>inÂvitro</i> . Biochemical Journal, 2015, 469, 121-134.	1.7	41
9	The SPL7013 dendrimer destabilizes the HIV-1 gp120–CD4 complex. Nanoscale, 2015, 7, 18628-18641.	2.8	41
10	Modeling how antibody responses may determine the efficacy of COVID-19 vaccines. Nature Computational Science, 2022, 2, 123-131.	3.8	39
11	Emergent properties of the interferon-signalling network may underlie the success of hepatitis C treatment. Nature Communications, 2014, 5, 3872.	5.8	37
12	A dynamical motif comprising the interactions between antigens and CD8 T cells may underlie the outcomes of viral infections. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 17393-17398.	3.3	33
13	Preferential Presentation of High-Affinity Immune Complexes in Germinal Centers Can Explain How Passive Immunization Improves the Humoral Response. Cell Reports, 2019, 29, 3946-3957.e5.	2.9	32
14	Estimates of intracellular delay and average drug efficacy from viral load data of HIV-infected individuals under antiretroviral therapy. Antiviral Therapy, 2004, 9, 237-46.	0.6	31
15	Ribavirin-Induced Anemia in Hepatitis C Virus Patients Undergoing Combination Therapy. PLoS Computational Biology, 2011, 7, e1001072.	1.5	30
16	Stochastic Simulations Suggest that HIV-1 Survives Close to Its Error Threshold. PLoS Computational Biology, 2012, 8, e1002684.	1.5	28
17	Timing the Emergence of Resistance to Anti-HIV Drugs with Large Genetic Barriers. PLoS Computational Biology, 2009, 5, e1000305.	1.5	24
18	Increased B Cell Selection Stringency In Germinal Centers Can Explain Improved COVID-19 Vaccine Efficacies With Low Dose Prime or Delayed Boost. Frontiers in Immunology, 2021, 12, 776933.	2.2	24

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19	Mathematical Model of Viral Kinetics In Vitro Estimates the Number of E2-CD81 Complexes Necessary for Hepatitis C Virus Entry. PLoS Computational Biology, 2011, 7, e1002307.	1.5	22
20	Emergence of Recombinant Forms of HIV: Dynamics and Scaling. PLoS Computational Biology, 2007, 3, e205.	1.5	21
21	Estimating Frequencies of Minority Nevirapine-Resistant Strains in Chronically HIV-1-Infected Individuals Nail^ve to Nevirapine by Using Stochastic Simulations and a Mathematical Model. Journal of Virology, 2010, 84, 10230-10240.	1.5	21
22	Simulations reveal that the HIV-1 gp120-CD4 complex dissociates via complex pathways and is a potential target of the polyamidoamine (PAMAM) dendrimer. Journal of Chemical Physics, 2013, 139, 024905.	1.2	21
23	Taking Multiple Infections of Cells and Recombination into Account Leads to Small Within-Host Effective-Population-Size Estimates of HIV-1. PLoS ONE, 2011, 6, e14531.	1.1	21
24	Estimating the Threshold Surface Density of Gp120-CCR5 Complexes Necessary for HIV-1 Envelope-Mediated Cell-Cell Fusion. PLoS ONE, 2011, 6, e19941.	1.1	21
25	A Finite Population Model of Molecular Evolution: Theory and Computation. Journal of Computational Biology, 2012, 19, 1176-1202.	0.8	20
26	Mechanistic insights into the effects of key mutations on SARS-CoV-2 RBD–ACE2 binding. Physical Chemistry Chemical Physics, 2021, 23, 26451-26458.	1.3	19
27	Modeling how reversal of immune exhaustion elicits cure of chronic hepatitis C after the end of treatment with directâ€acting antiviral agents. Immunology and Cell Biology, 2018, 96, 969-980.	1.0	18
28	Influence of recombination on acquisition and reversion of immune escape and compensatory mutations in HIV-1. Epidemics, 2016, 14, 11-25.	1.5	17
29	You Cannot Have Your Synergy and Efficacy Too. Trends in Pharmacological Sciences, 2019, 40, 811-817.	4.0	17
30	Early exposure to broadly neutralizing antibodies may trigger a dynamical switch from progressive disease to lasting control of SHIV infection. PLoS Computational Biology, 2020, 16, e1008064.	1.5	17
31	Towards multiscale modeling of the CD8 ⁺ T cell response to viral infections. Wiley Interdisciplinary Reviews: Systems Biology and Medicine, 2019, 11, e1446.	6.6	16
32	An efficient and scalable top-down method for predicting structures of microbial communities. Nature Computational Science, 2021, 1, 619-628.	3.8	16
33	Strand-specific affinity of host factor hnRNP C1/C2 guides positive to negative-strand ratio in Coxsackievirus B3 infection. RNA Biology, 2019, 16, 1286-1299.	1.5	15
34	Modelling how responsiveness to interferon improves interferon-free treatment of hepatitis C virus infection. PLoS Computational Biology, 2018, 14, e1006335.	1.5	14
35	Modeling recapitulates the heterogeneous outcomes of SARS-CoV-2 infection and quantifies the differences in the innate immune and CD8 T-cell responses between patients experiencing mild and severe symptoms. PLoS Pathogens, 2022, 18, e1010630.	2.1	14
36	Viral Kinetics Suggests a Reconciliation of the Disparate Observations of the Modulation of Claudin-1 Expression on Cells Exposed to Hepatitis C Virus. PLoS ONE, 2012, 7, e36107.	1.1	13

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37	Trade-off between synergy and efficacy in combinations of HIV-1 latency-reversing agents. PLoS Computational Biology, 2018, 14, e1006004.	1.5	13
38	Viral Decay Dynamics and Mathematical Modeling of Treatment Response: Evidence of Lower in vivo Fitness of HIV-1 Subtype C. Journal of Acquired Immune Deficiency Syndromes (1999), 2016, 73, 245-251.	0.9	12
39	Inhibitors of hepatitis C virus entry may be potent ingredients of optimal drug combinations. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, E4524-E4526.	3.3	12
40	Activation of Bacterial Histidine Kinases: Insights into the Kinetics of the <i>cis</i> Autophosphorylation Mechanism. MSphere, 2018, 3, .	1.3	12
41	Interferon at the cellular, individual, and population level in hepatitis C virus infection: Its role in the interferonâ€free treatment era. Immunological Reviews, 2018, 285, 55-71.	2.8	11
42	Pre-existing resistance in the latent reservoir can compromise VRC01 therapy during chronic HIV-1 infection. PLoS Computational Biology, 2020, 16, e1008434.	1.5	11
43	Estimating the fraction of progeny virions that must incorporate APOBEC3G for suppression of productive HIV-1 infection. Virology, 2014, 449, 224-228.	1.1	10
44	Concerted Interactions between Multiple gp41 Trimers and the Target Cell Lipidome May Be Required for HIV-1 Entry. Journal of Chemical Information and Modeling, 2021, 61, 444-454.	2.5	9
45	Mutational pathway maps and founder effects define the within-host spectrum of hepatitis C virus mutants resistant to drugs. PLoS Pathogens, 2019, 15, e1007701.	2.1	8
46	Transmitted HIV-1 is more virulent in heterosexual individuals than men-who-have-sex-with-men. PLoS Pathogens, 2022, 18, e1010319.	2.1	8
47	Scaling law characterizing the dynamics of the transition of HIV-1 to error catastrophe. Physical Biology, 2015, 12, 054001.	0.8	7
48	13 Modelling the in vivo growth rate of HIV: implications for vaccination. Studies in Multidisciplinarity, 2005, , 231-246.	0.0	6
49	A Formula to Estimate the Optimal Dosage of Ribavirin for the Treatment of Chronic Hepatitis C: Influence of Itpa Polymorphisms. Antiviral Therapy, 2012, 17, 1581-1592.	0.6	6
50	Models of Viral Population Dynamics. Current Topics in Microbiology and Immunology, 2015, 392, 277-302.	0.7	6
51	Salmonella escapes antigen presentation through K63 ubiquitination mediated endosomal proteolysis of MHC II via modulation of endosomal acidification in dendritic cells. Pathogens and Disease, 2018, 76, .	0.8	6
52	<i>110th Anniversary:</i> High-Order Interactions Can Eclipse Pairwise Interactions in Shaping the Structure of Microbial Communities. Industrial & Engineering Chemistry Research, 2019, 58, 23508-23518.	1.8	5
53	Physical â€~strength' of the multiâ€protein chain connecting immune cells: Does the weakest link limit antibody affinity maturation?. BioEssays, 2021, 43, 2000159.	1.2	5
54	A Low-Prevalence Single-Nucleotide Polymorphism in the Sensor Kinase PhoR in Mycobacterium tuberculosis Suppresses Its Autophosphatase Activity and Reduces Pathogenic Fitness: Implications in Evolutionary Selection. Frontiers in Microbiology, 2021, 12, 724482.	1.5	5

#	Article	IF	CITATIONS
55	The within-host fitness of HIV-1 increases with age in ART-naÃ⁻ve HIV-1 subtype C infected children. Scientific Reports, 2021, 11, 2990.	1.6	3
56	Bistability in virus–host interaction networks underlies the success of hepatitis C treatments. , 2020, , 131-156.		0