Benjamin E Gewurz

List of Publications by Year in descending order

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RENIAMIN F CEMILOZ

#	Article	IF	CITATIONS
1	Epigenetic control of the Epstein-Barr lifecycle. Current Opinion in Virology, 2022, 52, 78-88.	5.4	21
2	Epstein–Barr virus oncoprotein–driven B cell metabolism remodeling. PLoS Pathogens, 2022, 18, e1010254.	4.7	11
3	Epstein–Barr virus latency programs dynamically sensitize B cells to ferroptosis. Proceedings of the National Academy of Sciences of the United States of America, 2022, 119, e2118300119.	7.1	19
4	Epstein-Barr virus BNRF1 destabilizes SMC5/6 cohesin complexes to evade its restriction of replication compartments. Cell Reports, 2022, 38, 110411.	6.4	31
5	The nuclear lamina binds the EBV genome during latency and regulates viral gene expression. PLoS Pathogens, 2022, 18, e1010400.	4.7	6
6	Therapeutically Increasing MHC-I Expression Potentiates Immune Checkpoint Blockade. Cancer Discovery, 2021, 11, 1524-1541.	9.4	103
7	N(6)â€methyladenosineâ€binding protein YTHDF1 suppresses EBV replication and promotes EBV RNA decay. EMBO Reports, 2021, 22, e50128.	4.5	59
8	SARS-CoV-2 hijacks folate and one-carbon metabolism for viral replication. Nature Communications, 2021, 12, 1676.	12.8	102
9	Innate Immune Modulation Induced by EBV Lytic Infection Promotes Endothelial Cell Inflammation and Vascular Injury in Scleroderma. Frontiers in Immunology, 2021, 12, 651013.	4.8	11
10	EBNA1 inhibitors have potent and selective antitumor activity in xenograft models of Epstein–Barr virus-associated gastric cancer. Gastric Cancer, 2021, 24, 1076-1088.	5.3	19
11	CYB561A3 is the key lysosomal iron reductase required for Burkitt B-cell growth and survival. Blood, 2021, 138, 2216-2230.	1.4	20
12	Abstract 65: Therapeutically increasing MHC-I expression potentiates immune checkpoint blockade. , 2021, , .		6
13	Epstein-Barr Virus Induced Cytidine Metabolism Roles in Transformed B-Cell Growth and Survival. MBio, 2021, 12, e0153021.	4.1	16
14	700â€Increasing MHC-I expression to potentiate immune checkpoint blockade therapy. , 2021, 9, A728-A728.		0
15	Histone Loaders CAF1 and HIRA Restrict Epstein-Barr Virus B-Cell Lytic Reactivation. MBio, 2020, 11, .	4.1	17
16	Single-cell transcriptomic analysis defines the interplay between tumor cells, viral infection, and the microenvironment in nasopharyngeal carcinoma. Cell Research, 2020, 30, 950-965.	12.0	111
17	DNA methylation enzymes and PRC1 restrict B-cell Epstein–Barr virus oncoprotein expression. Nature Microbiology, 2020, 5, 1051-1063.	13.3	32
18	Epigenetic reprogramming sensitizes immunologically silent EBV+ lymphomas to virus-directed immunotherapy. Blood, 2020, 135, 1870-1881.	1.4	39

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19	CD21 (Complement Receptor 2) Is the Receptor for Epstein-Barr Virus Entry into T Cells. Journal of Virology, 2020, 94, .	3.4	33
20	MYC Controls the Epstein-Barr Virus Lytic Switch. Molecular Cell, 2020, 78, 653-669.e8.	9.7	67
21	CRISPR/Cas9 Screens Reveal Multiple Layers of B cell CD40 Regulation. Cell Reports, 2019, 28, 1307-1322.e8.	6.4	18
22	Epstein-Barr-Virus-Induced One-Carbon Metabolism Drives B Cell Transformation. Cell Metabolism, 2019, 30, 539-555.e11.	16.2	119
23	Epstein-Barr virus subverts mevalonate and fatty acid pathways to promote infected B-cell proliferation and survival. PLoS Pathogens, 2019, 15, e1008030.	4.7	57
24	TAF Family Proteins and MEF2C Are Essential for Epstein-Barr Virus Super-Enhancer Activity. Journal of Virology, 2019, 93, .	3.4	10
25	Genome-wide CRISPR-based gene knockout screens reveal cellular factors and pathways essential for nasopharyngeal carcinoma. Journal of Biological Chemistry, 2019, 294, 9734-9745.	3.4	12
26	RNA Sequencing Analyses of Gene Expression during Epstein-Barr Virus Infection of Primary B Lymphocytes. Journal of Virology, 2019, 93, .	3.4	71
27	Epstein-Barr Virus Nuclear Antigen Leader Protein Coactivates EP300. Journal of Virology, 2018, 92, .	3.4	15
28	CRISPR/Cas9â€Mediated Genome Editing in Epsteinâ€Barr Virusâ€Transformed Lymphoblastoid Bâ€Cell Lines. Current Protocols in Molecular Biology, 2018, 121, 31.12.1-31.12.23.	2.9	27
29	Modulating Gene Expression in Epsteinâ€Barr Virus (EBV)â€Positive B Cell Lines with CRISPRa and CRISPRi. Current Protocols in Molecular Biology, 2018, 121, 31.13.1-31.13.18.	2.9	4
30	Ephrin receptor A2 is an epithelial cell receptor for Epstein–Barr virus entry. Nature Microbiology, 2018, 3, 1-8.	13.3	151
31	Epigenetic crossroads of the Epstein-Barr virus B-cell relationship. Current Opinion in Virology, 2018, 32, 15-23.	5.4	17
32	CRISPR–Cas9 Genetic Analysis of Virus–Host Interactions. Viruses, 2018, 10, 55.	3.3	20
33	CRISPR/Cas9 Screens Reveal Epstein-Barr Virus-Transformed B Cell Host Dependency Factors. Cell Host and Microbe, 2017, 21, 580-591.e7.	11.0	113
34	A Temporal Proteomic Map of Epstein-Barr Virus Lytic Replication in B Cells. Cell Reports, 2017, 19, 1479-1493.	6.4	83
35	Mouse model of Epstein–Barr virus LMP1- and LMP2A-driven germinal center B-cell lymphoproliferative disease. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, 4751-4756.	7.1	44
36	The Epstein-Barr Virus Regulome in Lymphoblastoid Cells. Cell Host and Microbe, 2017, 22, 561-573.e4.	11.0	89

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37	Epstein-Barr Virus LMP1-Mediated Oncogenicity. Journal of Virology, 2017, 91, .	3.4	103
38	CRISPR/Cas9 Screens Reveal Requirements for Host Cell Sulfation and Fucosylation in Bacterial Type III Secretion System-Mediated Cytotoxicity. Cell Host and Microbe, 2016, 20, 226-237.	11.0	64
39	Epstein-Barr Virus Oncoprotein Super-enhancers Control B Cell Growth. Cell Host and Microbe, 2015, 17, 205-216.	11.0	146
40	TRAF1 Coordinates Polyubiquitin Signaling to Enhance Epstein-Barr Virus LMP1-Mediated Growth and Survival Pathway Activation. PLoS Pathogens, 2015, 11, e1004890.	4.7	67
41	Evasion of affinity-based selection in germinal centers by Epstein–Barr virus LMP2A. Proceedings of the United States of America, 2015, 112, 11612-11617.	7.1	43
42	Regulation of p53 and Rb Links the Alternative NF-κB Pathway to EZH2 Expression and Cell Senescence. PLoS Genetics, 2014, 10, e1004642.	3.5	83
43	The NF-κB Genomic Landscape in Lymphoblastoid B Cells. Cell Reports, 2014, 8, 1595-1606.	6.4	147
44	Genome-wide siRNA screen for mediators of NF-κB activation. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, 2467-2472.	7.1	100
45	Canonical NF-κB Activation Is Essential for Epstein-Barr Virus Latent Membrane Protein 1 TES2/CTAR2 Gene Regulation. Journal of Virology, 2011, 85, 6764-6773.	3.4	43
46	Epstein-Barr latent membrane protein 1 transformation site 2 activates NF-κB in the absence of NF-κB essential modifier residues 133–224 or 373–419. Proceedings of the National Academy of Sciences of the United States of America, 2010, 107, 18103-18108.	7.1	12
47	Human herpesvirus 6 encephalitis. Current Infectious Disease Reports, 2008, 10, 292-9.	3.0	38
48	IRF7 activation by Epstein-Barr virus latent membrane protein 1 requires localization at activation sites and TRAF6, but not TRAF2 or TRAF3. Proceedings of the National Academy of Sciences of the United States of America, 2008, 105, 18448-18453.	7.1	42
49	Herpesvirus evasion of T-cell immunity. , 2007, , 1117-1136.		4
50	DNA-Damage Control: Claspin Destruction Turns off the Checkpoint. Current Biology, 2006, 16, R932-R934.	3.9	11
51	HIV and the breast. Aids Reader, 2005, 15, 392-6, 399-402.	0.3	9
52	US2, a Human Cytomegalovirus-encoded Type I Membrane Protein, Contains a Non-cleavable Amino-terminal Signal Peptide. Journal of Biological Chemistry, 2002, 277, 11306-11313.	3.4	33
53	Down-regulation of MHC class I antigen presentation by HCMV; lessons for tumor immunology. Immunological Investigations, 2000, 29, 97-100.	2.0	39
54	Viral Subversion of the Immune System. Annual Review of Immunology, 2000, 18, 861-926.	21.8	764