Will Jones PhD, Editor *The Daily Skeptic*

Sunday 8 Jan 2023

Dear Will:

Thank you for publishing your excellent article at *The Brownstone Institute* [1]. Brilliant. It was sent to me by my friend and colleague Heath Goddard. I benefited enormously from this article - my only short critique is on your statement right at the end re virus is man-made. I will deal with that below, but what I say on that *in no way detracts* from the excellent analysis of the rest of your article.

I am a scientist (74 yr), PhD thesis plus 6 books and over 150 papers in 50 years at the coal face in research and teaching in microbiology (mucosal immunology secretory IgA), molecular and cellular immunology ( reverse transcriptase-coupled somatic and germline evolutionary genetic or inherited diversity mechanisms of immunity) and more recently these past 7 years (since 2015) with Professor N Chandra Wickramasinghe and colleagues (cosmic biology, evidence demonstrating panspermia, origins life on Earth).

So, as luck would have it, I have both a broad and deep understanding of biomedical science - and particular in relation to all issues relating to COVID-19 including the most plausible infection and immunity aspects; and, of course, I have been following the voluminous data on the now clear lack of safety and prior lack of safety testing by the Pharmaceutical industry on the mRNA LNP expression vector vaccine, e.g. my recent interview on Friday 6th Jan 2023 with moderator Ben Bornstein and Professor Ian Brighthope. see substack URL

<https://thenobodywhoknowseverybody.substack.com/p/screwed-up-lnp-mrna-an-off-target?sd=pf>

So, I have benefited enormously from reading your analysis because we have also been following the pandemic at many key levels of understanding since mid-Jan 2020, and publishing continuously in peer review and fast accelerated journal outlets. Many of our key Covid papers are now curated in a book recently published (Wickramasinghe C, Gorczynski RM, Steele EJ (Eds) Understanding the Origin and Global Spread of COVID-19 World Scientific Publ. Co Pte Singapore, 2022. Many of the same papers with URL links are in the attached list. I will refer to some of these and others here.

It is our conviction that understanding *both* the plausible explanation for COVID-19 origins (natural or man-made?) and its main global epidemiological spread process igniting explosive epidemics on the ground or at sea (viral-laden dust clouds transported both short, local and regional , as well as vast distances via global upper and lower prevailing wind systems and then brought down by local weather to contaminate the terrestrial or ocean environment).

But I want to keep this short as possible. I want to comment on the really new reference you brought to our attention Amendola et al 2022 [2]. I do not dispute your conclusions- I just want to sharpen what the paper is saying particularly with respect to the *exact timing* of emergence COVID-19 prior to the main explosive epidemic across China through January 2020 We would agree pretty well with everything you have concluded.

**Formal Report: Amendola et al 2022, Time of Arrival of COVID-19, Deaminase-Driven Haplotype-Sorting**

So, this is an important paper providing COVID-19 genomic RNA sequence evidence with a direct bearing on the time of arrival of *first detections* of COVID-19 genomes on Earth, in the Lombardy region of Italy after October 11, 2019 (the time of the putative life bearing meteorite strike in the stratosphere over Jilin NE China on the 40o N Latitude line). There have been other spot sightings of COVID-19 from November 2019 in China and elsewhere prior to the main explosive epidemic across China from late December 2019 and through January 2020. Those reports are mainly from serology which are equivocal, but believable in some cases, see our discussions of Althoff et al 2020 data brought to our attention by George Howard in [3], and our analysis of the highly questionable data of Apalone et al 2021 about to be published in [4].

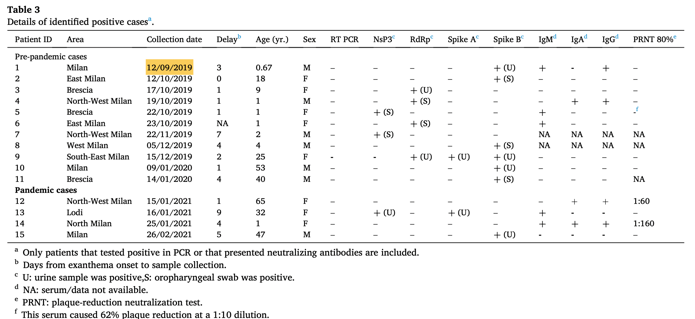
The best evidence is genomic RNA sequence of known regions in COVID-19 or full-length 29,903 nt sequencing if possible i.e. those nest PCR recovered sequences that map to the China Hu-1 reference genome . Some of that type of evidence is in Amendola et al 2022- but it also requires proper interpretation.

In Amendola et al the critical regions to read are the Methods, then key results on Sequence Analysis in Table 3 and Figure 2.

Table 3 is pasted below. Figure 2 is pasted after references. In Pre- Pandemic samples it is important to note

“Samples became positive after nested PCRs, and none of the samples tested positive with the Real-Time PCR diagnostic protocol. This suggests a low viral load at the detection threshold.” This is crucial. In all these patients the evidence for viral replication suggest *the very earliest stages* of gaining traction in the mucosal cells of the respiratory tract of a human host. All patients are asymptomatic for COVID-19 itself.

The patient that scores positive after nested PCR in Urine before October 11, 2019 is a baby. They provide no sequence data for that result, and that patient could be the “one of six” where they just state “Six out of the seven partial S sequences (fragment B) were 100% identical to the reference sequence Wuhan-Hu-1.” But they do not tell us which one. So, we have data for a positive nested PCR detection in a baby’s urine 12 September 2019 that is equivocal and no genomic sequence data.



But the rest of the positive detection data from October 17 2019 is very interesting. I accept it and believe it to be essentially correct but not their speculative model-based explanation of what it implies for the timing of emergence of COVID-19.

Section 3.2 in *Sequence Analysis* and Figure 2 identifies that pre-pandemic patients #4,#5,#6,#7,#8,#10,#14 have genomic sequence evidence consistent with the non-China haplotype data ‘coordinated mutation’ patterns (C3037T, C14408T, and A23403G) first analysed and reported by Steele and Lindley 2020 [5]. (Many other independent reports consistent with our paper and these are appended as an Appendix). They are diagnostic of L214c or L241f haplotypes observed outside China in our analysis. I am now of the conviction that our explanation [5] for appearance of new haplotypes via APOBEC and ADAR deaminase-mediated riboswitching (producing *coordinate*d mutations) to achieve the best replicative genome for that host genetic back ground *is still the best interpretation of the data* [5]. This means that immediately on arrival into a patient’s mucosal epithelium of the respiratory tract in the first 24 hr of potential infection the haplotype sorting riboswitch process allows a full length RNA genome to emerge and gain replicative traction.

The actual evidence they get supports this - because as they make clear from the data (Table 3) “ Samples became positive after nested PCRs, and none of the samples tested positive with the Real-Time PCR diagnostic protocol. This suggests a low viral load at the detection threshold.”

So, we are dealing with events *very early* in infection. The haplotype sort has *to take place quickly* or that attempted infection focus of virus will be eliminated by the host’s Innate Immune Interferon Stimulated Gene (ISG) cascade attack on all facets of the virus life cycle ( it involves almost 1000 gene products , protein and RNA directed at pathogen life cycles). Thus, I do not accept their speculation on timing as it ignores these crucial Host-Parasite interaction factors viz.

“Since all three β mutations were present in samples collected on October 17, 2019, we estimate that the progenitor of SARS-CoV-2 could have already existed 11.6–16.2 weeks earlier than October 17, i.e., in late June 2019 to late July 2019, using a simple extrapolation assuming six mutations from the ancestral genome, constant substitution rate, and the published range of mutation rate estimates (Kumar et al., 2021; Pekar et al., 2021).”

So, within 24hr (maybe 48 hr at latest) of arrival into a human nose or throat, the first evidence of mature genome replicating virus with *the correct replicative haplotype* will be apparent. So we come back to the original first report work of Steele and Lindley 2020 [5] - the riboswitching haplotype sorting model described in that paper needs to be taken seriously, indeed it is actually supported now by these sequence data recovered from very low viral loads in asymptomatics.

The Amendola et al 2022 data thus are consistent with all the other evidence we have analysed suggesting the putative meteorite strike of October 11 2019 over Jilin is indeed the likely first cause of the COVID-19 pandemic. For a full comprehensive review see [6] and the paper list attached with this email letter.

However I actually date the first critical nested PCR recovery to 19 Oct 2019, I am certain there is a typo error they have made if you look at Fig 2 and my summary Table below:

**Highlight indicates recovered sequence information mapped to Hu-1. Thus sequences from 6/11 samples ; and 5/11 are false positives (nested PCR**)

Pre Pandemic

Patient ID & Collection Date

1 12/09/2019

2 12/10/2019

3 17/10/2019

4 19/10/2019

5 22/10/2019

6 23/10/2019

7 22/11/2019

8 5/12/2019

9 15/12/2019

10 9/1/2020

11 14/1/2020

The situation is therefore better than I thought as 5/11 pre-pandemics are false positives by nested PCR i.e. only 6/11 recovered sequence that could be mapped to Hu-1 reference genome. That begins from 19 October 2019. So day of first detection is 19 October 2019 eight days after meteorite strike over Jilin NE China October 11 2019.

On the interesting excess of COVID-19 deaths globally you highlighted, a plausible explanation is the total viral load in a given dust cloud coming down to contaminate a given terrestrial or ocean region. That is a big unknown given what we have seen in global new cases per day epidemiology patterns. Indeed we mention first strike viral loads in the explosive Wuhan and New York epidemics as undefined factors in Steele and Lindley 2020 [5].

**Natural or Man-Made?**

This is a very important question and must be addressed carefully. The most plausible scientific answer to it will allow us to handle the inevitable future pandemics that strike the Earth (usually cold and flus) more rationally rather than in a ‘wild animal stampede’ as has unfolded with COVID-19.

The key point to understand is that pandemics have always arrived suddenly throughout recorded history, and most plausibly from Space. In quite reflection it is important to take time out and purchase and read

Hoyle F, Wickramasinghe NC *Diseases from Space* J.M.  Dent Ltd, London,1979,  now out in a 2nd edition reprint with some Covid update Wickramasinghe C. *Diseases from Outer Space* World Scientific Publ. Co. Pte , Singapore, 2020

And it is important you embrace all the data, not just the sequence peculiarities in Hu-1 reference sequence such as Gain-Function sequence features in Covid-19 genomes ( features like this will be found in *any* viral genome you inspect carefully) - they may be interesting but they are *completely  irrelevant facts* (they are Red Herrings )† not able to explain both current and historical pandemic data nor the epidemiology local, regional, country wide or global ( clear global synchrony strike events- Space Weather event), and certainly not the natural adaptive genetics and haplotype switching of the virus ( see list papers and in our big review [6].

**Key Observations that any Human Engineered/Lab Leak theory must handle or try to explain**

• Sudden emergence in China, and eruption all across China at same time (see attached case incidence map through January 2020 . A similar event happened in India in April 2021 [see new cases per day epidemiology plot in 3].

• The putative life-bearing meteorite strike in the stratosphere on the 40o N latitude line over Jilin NE China on the night of October 11 2019 is the most likely first cause of Covid-19 [6].

• How and why the hundreds and thousands of known ‘mystery cases’, that defy contact-tracing an original patient X and any type of person-to-person spread epidemiological model [3,6].

• How could the Spanish Flu 1918-19 have emerged suddenly under a Lab leak bioweapon theory of pandemic emergence?

• Back then there was no knowledge DNA, RNA, Protein, molecular biology, genetic engineering, and certainly no bioweapon labs or bioweapon research back in 1918.

• Back then there was no air travel, yet like Covid-19 now, strikes on cities in India and USA on the same day and in isolated regions and e.g. for Flu Alaska 1918-19, for Covid in Antarctica .

• Strikes by Flu virus on ships at sea in 1918-1919, just like Covid-19 now [see list and 6].

• So the Human Engineered Bioweapon Lab Leak theory, or anything like it, cannot explain the sudden emergence and epidemiology of Spanish Flu – that strong obvious conclusion of ours cannot be dismissed, as what happened in 1918-19 *is a relevant scientific fact* if the claim is that human engineered viruses and pathogens explain the sudden emergence of pandemic phenomena.

 Thus, all ‘Lab Leak Human Engineered’ explanations which depend on ‘Gain of Function’ ideas or any other supposed plausible genetic manipulation in the laboratory are incredulously implausible if you are a serious scientist reacting to relevant evidence about emergence pandemics. They are complete Red Herrings.† I cannot emphasise that strongly enough. I cannot understand as a scientist dealing with reality how the ‘China Virus’ story, a cold war conspiracy theory, has had such a grip, particularly on the American mind.

But this critique does not detract at all from the rest of your article- please understand that.

Best for New Year

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†**“Red Herrings”** – What are they?

The art of all scientific analysis and inquiry is the detection of “Red Herrings” , sifting the ‘wheat from the chaff’ – otherwise you can spend your entire scientific life thrashing about analysing a never-ending set of Red Herrings or even technical artefacts (or fabricated data).

In the Universe of Facts, only a very few are Relevant to explaining or understanding any given phenomenon or observation. The Wuhan /Human Engineered Lab Leak theory is based on Red Herrings - so it can be immediately dismissed. Why? Because none of the theory explains

• What happened a 100 years ago in the Spanish Flu 1918-1919.

• What happened suddenly all across China in Dec 2019-Jan 2020.

• Nor does it grapple with the early genetics of the virus I early months nor its rapid early spread in global prevailing wind systems.

This is the way to proceed scientifically in understanding the origins of the COVID-19 Pandemic. The alternative with the impossible odds on animal jumps and implausible lab leaks *versus* the most plausible theory that addresses all the relevant facts is the panspermia scientific explanation we offer. See all references to many of our scientific papers, and attached and specifically again at DOI: [10.31038/IDT.2021223](https://doi.org/10.31038/IDT.2021223) Please take the time to read and evaluate the relevant scientific evidence I bring to your attention.

**References**

[1] Jones W. (2023) **The evidence COVID-19 was spreading around the world in late 2019**. *Brownstone Institute*, Jan 2 2023. <https://brownstone.org/articles/the-evidence-covid-19-was-spreading-around-the-world-in-late-2019/>

[2]. Amendola A, Canuti M, Bianchi S, Kumar S, Fappani C, Gori M, et al 2022 **Molecular evidence for SARS-CoV-2 in samples collected from patients with morbilliform eruptions since late 2019 in Lombardy, northern Italy**. *Environ Res*. 2022 Dec;215(Pt 1):113979.doi: 10.1016/j.envres.2022.113979. Epub 2022 Aug 25. <https://pubmed.ncbi.nlm.nih.gov/36029839/>

[3]. Steele EJ, Gorczynski RM, Carnegie P, Tokoro G, Wallis DH, Temple R, Wainwright M, and Wickramasinghe, NC (2021) **COVID-19 Sudden Outbreak of Mystery Case Transmissions in Victoria, Australia, May-June 2021: Strong Evidence of Tropospheric Transport of Human Passaged Infective Virions from the Indian Epidemic.** *Infectious Diseases and Therapeuptics* 2021 Volume 2 Issue 1 ID at Infectious Diseases and Therapeutics: IDT-2021-214. DOI: [10.31038/IDT.2021214](https://doi.org/10.31038/IDT.2021214)

[4]. Steele EJ, Gorczynski RM, Lindley RA,and Wickramasinghe NC (2022) **Natural Antibodies and SARS CoV-2 Specific Antibodies in Healthy Asymptomatic Individuals** *Clinical Infectious Diseases* Letter to Editor accepted as December 6 2022

Posted at Academia.edu December 26 2022

[Natural Antibodies and SARS CoV-2 Specific Antibodies in Healthy Asymptomatic Individuals- Critique Apalone et al 2021](https://www.academia.edu/93671920/Natural_Antibodies_and_SARS_CoV_2_Specific_Antibodies_in_Healthy_Asymptomatic_Individuals_Critique_Apalone_et_al_2021)

Or

<https://www.academia.edu/93671920/Natural_Antibodies_and_SARS_CoV_2_Specific_Antibodies_in_Healthy_Asymptomatic_Individuals_Critique_Apalone_et_al_2021>

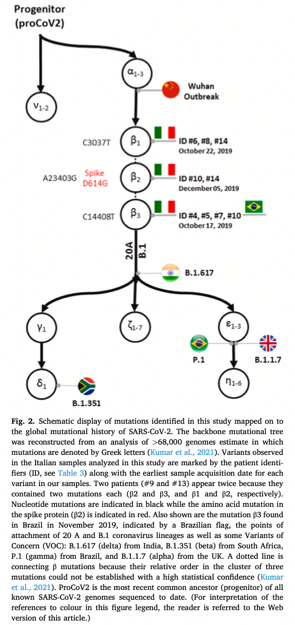
[5]. Steele EJ, Lindley RA (2020) **Analysis of APOBEC and ADAR deaminase-driven Riboswitch Haplotypes in COVID-19 RNA strain variants and the implications for vaccine design**. *Research Reports* doi:10.9777/rr.2020.10001

<https://www.companyofscientists.com/index.php/rr>

<https://www.companyofscientists.com/index.php/rr/article/view/177>

[6].Steele EJ, Gorczynski RM, Lindley RA, Carnegie PR, Rebhran H, et al. (2022**) Overview SARS-CoV-2 Pandemic as January-February 2022: Likely Cometary Origin, Global Spread, Prospects for Future Vaccine Efficacy**. *Infect Dis Ther* Volume 3(1): 1-16. DOI: [10.31038/IDT.2022311](https://doi.org/10.31038/IDT.2022311)

<https://www.academia.edu/69066451/Overview_SARS_CoV_2_Pandemic_as_January_February_2022_Likely_Cometary_Origin_Global_Spread_Prospects_for_Future_Vaccine_Efficacy>



**Appendix**

**Papers on COVID -19 Consistent with Steele & Lindley 2020-21 papers on Host APOBEC and ADAR responses to replicating SARS CoV-2 Genome**

Steele EJ, Lindley RA (2020) Analysis of APOBEC and ADAR deaminase-driven Riboswitch Haplotypes in COVID-19 RNA strain variants and the implications for vaccine design. *Research Reports* doi:10.9777/rr.2020.10001

<https://www.companyofscientists.com/index.php/rr/article/view/177>

Key words: SARS CoV-2; Innate Immunity; APOBEC and ADAR RNA editing; C-to-U RNA editing; A-to-I RNA editing; Early months 2020 COVID-19 pandemic ; Putative Riboswitch haplotypes; Vaccine design.

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Liu X, Liu X, Zhou J, Dong Y, Jiang Jiang W. 2022. Rampant C-to-U deamination accounts for the intrinsically high mutation rate in SARS-CoV-2 spike gene. RNA. 2022 Jul;28(7):917-926.doi: 10.1261/rna.079160.122. Epub 2022 May 4.

<https://pubmed.ncbi.nlm.nih.gov/35508354/>

Keywords: C-to-U deamination; Spike protein (S) gene; SARS-CoV-2; mutation rate; single-stranded RNA.

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Martignano F, Di Giorgio S, Mattiuz G, Conticello SG. 2022. Commentary on "Poor evidence for host-dependent regular RNA editing in the transcriptome of SARS-CoV-2". J Appl Genet. 2022 May;63(2):423-428.doi: 10.1007/s13353-022-00688-x. Epub 2022 Mar 12.

<https://pubmed.ncbi.nlm.nih.gov/35279801/>

Keywords: ADARs; APOBECs; RNA editing; Viruses.

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Song Y, He X, Yang W, Wu Y, Cui J, Tang T, Zhang R. 2022. Virus-specific editing identification approach reveals the landscape of A-to-I editing and its impacts on SARS-CoV-2 characteristics and evolution. Nucleic Acids Res. 2022 Mar 21;50(5):2509-2521.doi: 10.1093/nar/gkac120.

<https://pubmed.ncbi.nlm.nih.gov/35234938/>

Key Words: A-to-I RNA editing; SARS-CoV-2 characteristics and evolution; intensity of innate immune response;

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Ringlander J, Fingal J, Kann H, Prakash K, Rydell G, Andrersson M, Martner A, et al. 2022. Impact of ADAR-induced editing of minor viral RNA populations on replication and transmission of SARS-CoV-2, Proc Natl Acad Sci U S A. 2022 Feb 8;119(6):e2112663119.doi: 10.1073/pnas.2112663119.

<https://pubmed.ncbi.nlm.nih.gov/35064076/>

Keywords: ADAR; RNA deamination; RNA mutation; SARS-CoV-2.

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Picardi E, Mansi L, Pesole G. 2021. Detection of A-to-I RNA Editing in SARS-COV-2. Genes (Basel). 2021 Dec 23;13(1):41.doi: 10.3390/genes13010041.

<https://pubmed.ncbi.nlm.nih.gov/35052382/>

Keywords: ADAR; RNA editing; SARS-COV-2; transcriptome.

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Liu R, Wu P, Ogrodzki P, Mahmoud S, Liang K, Liu P, et al. 2021. Genomic epidemiology of SARS-CoV-2 in the UAE reveals novel virus mutation, patterns of co-infection and tissue specific host immune response.

Sci Rep. 2021 Jul 7;11(1):13971.doi: 10.1038/s41598-021-92851-3.

<https://pubmed.ncbi.nlm.nih.gov/34234167/>

Erratum in

[Publisher Correction: Genomic epidemiology of SARS-CoV-2 in the UAE reveals novel virus mutation, patterns of co-infection and tissue specific host immune response.](https://pubmed.ncbi.nlm.nih.gov/34526633/)

Key Words: Origin SARS Co-2 in United Arab Emirates 2020; UAE genomic variants; Up regulation APOBEC4 expression; ADAR and APOBEC response , tissue specifity

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Kockler ZW, Gordenin DA. 2021. From RNA World to SARS-CoV-2: The Edited Story of RNA Viral Evolution. Cells. 2021 Jun 20;10(6):1557.doi: 10.3390/cells10061557.

<https://pubmed.ncbi.nlm.nih.gov/34202997/>

Keywords: ADAR; APOBEC; RNA editing; RNA world theory; genome stability; hypermutation; messenger RNA; viral RNA; viral evolution.

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Vlachogiannis NI, Verrou K-M, Stellos K, Sfikakis PP, Paraskevis D. 2021. The role of A-to-I RNA editing in infections by RNA viruses: Possible implications for SARS-CoV-2 infection. Clin Immunol. 2021 May;226:108699.doi: 10.1016/j.clim.2021.108699.Epub 2021 Feb 25.

<https://pubmed.ncbi.nlm.nih.gov/33639276/>

Keywords: A-to-I RNA editing; Innate immunity; Mutations; SARS-CoV-2; Viral infections

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Crooke PS, Tossberg JT, Porter KP, Aune TM. 2021. Cutting Edge: Reduced Adenosine-to-Inosine Editing of Endogenous Alu RNAs in Severe COVID-19 Disease. J Immunol. 2021 Apr 15;206(8):1691-1696.doi: 10.4049/jimmunol.2001428. Epub 2021 Mar 29. <https://pubmed.ncbi.nlm.nih.gov/33782089/>

Abstract

Severe COVID-19 disease is associated with elevated inflammatory responses. One form of Aicardi-Goutières syndrome caused by inactivating mutations in ADAR results in reduced adenosine-to-inosine (A-to-I) editing of endogenous dsRNAs, induction of IFNs, IFN-stimulated genes, other inflammatory mediators, morbidity, and mortality. Alu elements, ∼10% of the human genome, are the most common A-to-I-editing sites. Using leukocyte whole-genome RNA-sequencing data, we found reduced A-to-I editing of Alu dsRNAs in patients with severe COVID-19 disease. Dendritic cells infected with COVID-19 also exhibit reduced A-to-I editing of Alu dsRNAs. Unedited Alu dsRNAs, but not edited Alu dsRNAs, are potent inducers of IRF and NF-κB transcriptional responses, IL6, IL8, and IFN-stimulated genes. Thus, decreased A-to-I editing that may lead to accumulation of unedited Alu dsRNAs and increased inflammatory responses is associated with severe COVID-19 disease.

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Azgari C, Kilinc Z, Turhan B, Circi D, Adebali O. 2021. The Mutation Profile of SARS-CoV-2 Is Primarily Shaped by the Host Antiviral Defense. Viruses. 2021 Mar 2;13(3):394.doi: 10.3390/v13030394.

<https://pubmed.ncbi.nlm.nih.gov/33801257/>

Keywords: ADAR; APOBEC; COVID-19; ROS; SARS-CoV-2; ZAP; evolution; mutation; phylogenetics.

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Siqueira JD, Goes LR, Alves BM|, de Carvalho PS, Cicala C, Arthos J, et al . 2021. SARS-CoV-2 genomic analyses in cancer patients reveal elevated intrahost genetic diversity. Virus Evol. 2021 Feb 16;7(1):veab013.doi: 10.1093/ve/veab013. eCollection 2021 Jan.

<https://pubmed.ncbi.nlm.nih.gov/33738124/>

Keywords: COVID-19; SARS-CoV-2; cancer; full-length genome; single nucleotide variant.

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Mourier T, Sadykov M, Carr MJ, Gonzalez G, Hall WW, Pain A. 2021. Host-directed editing of the SARS-CoV-2 genome. Biochem Biophys Res Commun. 2021 Jan 29;538:35-39. doi: 10.1016/j.bbrc.2020.10.092. Epub 2020 Nov 5.

<https://pubmed.ncbi.nlm.nih.gov/33234239/>

Keywords: ADAR; APOBEC; Genome editing; ROS; SARS-CoV-2; Virus evolution.

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Wang R, Hozumi Y, Zheng Y-H, Yin C, Wei G-W. 2020. Host Immune Response Driving SARS-CoV-2 Evolution. Viruses. 2020 Sep 27;12(10):1095.doi: 10.3390/v12101095.

<https://pubmed.ncbi.nlm.nih.gov/32992592/>

Keywords: ADAR; APOBEC; COVID-19; SARS-CoV-2; gene editing.

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Di Giorgio S, Martignano F, Torcia MG, Mattiuz G, Conticello SG. 2020. Evidence for host-dependent RNA editing in the transcriptome of SARS-CoV-2. Sci Adv. 2020 Jun 17;6(25):eabb5813.doi: 10.1126/sciadv.abb5813. eCollection 2020 Jun.

<https://pubmed.ncbi.nlm.nih.gov/32596474/>

Key Words: COVID-19 outbreaks ; SARS-CoV-2 virus; Host Response; Mutational analysis; APOBEC; ADAR; RNA editing