# The Lancet Respiratory Medicine: Role of exosomes in false-positive covid-19 PCR tests

**Preprint** · November 2021

CITATIONS
0

READS
68,954

**3 authors:**

P. Stallinga
Universidade do Algarve
**99** PUBLICATIONS **2,081** CITATIONS
SEE PROFILE

Leslie V. Woodcock
Universidade do Algarve
**163** PUBLICATIONS **4,499** CITATIONS
SEE PROFILE

Igor Khmelinskii
Universidade do Algarve
**305** PUBLICATIONS **3,488** CITATIONS
SEE PROFILE

The Lancet Respiratory Medicine

# Role of exosomes in false-positive covid-19 PCR tests: non-specificity of SARS-CoV-2-RNAin vivodetection explains artificial post-pandemic peaks

--Manuscript Draft--

| | |
|---|---|
| **Manuscript Number:** | thelancetrm-D-21-01205 |
| **Article Type:** | Article (Original Research) |
| **Keywords:** | COVID-19; PCR test; false positive; SARS-CoV-2-RNA; exosomes |
| **Corresponding Author:** | Igor Khmelinskii<br>UALG: Universidade do Algarve<br>Faro, Algarve PORTUGAL |
| **First Author:** | Igor Khmelinskii |
| **Order of Authors:** | Igor Khmelinskii |
| | Peter Stallinga |
| | Leslie V. Woodcock |
| **Manuscript Region of Origin:** | UNITED KINGDOM |
| **Abstract:** | Summary<br><br>Background<br><br>The COVID-19 pandemic priorities have focused on prevention by detection and response. National governments' prevention response decisions are based upon detection statistics from PCR tests that are used to define numbers of (i) COVID-19 infected persons, (ii) COVID-19 hospitalisations, and (iii) COVID-19 deaths. These statistics assume a priori that PCR tests are nigh 100% true detectors of COVID-19 infections. Here we will provide an alternative interpretation, along with the compelling evidence, that false positives have distorted to some degree the statistics of the primary outbreaks, and account for almost the whole of the 2nd and subsequent apparent COVID-19 outbreak peaks in various countries.<br><br>Methods<br><br>We extract from the published literature on PCR-test outcomes graphical data that reveals the evidence for a very large percentage of false positive results. We review the role of exosomes in the immune response to all respiratory viral infections and its effect on PCR tests. We hypothesise that exosomes, triggered by all viral respiratory infections, are largely responsible for positive outcomes from PCR-tests for COVID-19. We test our alternative interpretation for consistency with the empirical epidemiological trends as published by WHO.<br><br>Findings<br><br>We find that PCR testing data for the second and following waves of COVID-19 pandemic indicate that these waves are mainly artefacts of false-positive results. We find that this interpretation provides a more consistent explanation of the known epidemiology of COVID-19 than the hitherto consensus notion of extremely contagious and rapidly mutating viruses.<br><br>Interpretation<br><br>The RNA code counted in PCR tests, previously attributed to SARS-CoV-2, belongs instead to a respiratory-virus-induced immune system response by human cells that liberate exosomes, and that vitiate PCR test results. PCR tests have zero specificity in vivo due to the exosome RNA. PCR tests exhibit excellent specificity in vitro on pure samples of other respiratory viruses. Low success rate of vaccines is explained by |

| | |
|---|---|
| | inexact identification of the SARS-CoV-2 RNA. |

THE LANCET Respiratory Medicine **Article** (to be submitted 2021)

# Role of exosomes in false-positive covid-19 PCR tests: non-specificity of SARS-CoV-2-RNA *in vivo* detection explains artificial post-pandemic peaks

**Igor Khmelinskii[1,4,5,6], Peter Stallinga[2,4,5] and Leslie V. Woodcock[3,5]**
University of Algarve, Faculty of Science and Technologies, Faro, Portugal
[1]Department of Chemistry and Pharmacy
[2]Department of Electronic Engineering and Informatics
[3]Department of Physics
[4]Centre of Electronics, Optoelectronics and Telecommunications
[5]Ossónoba Philosophical Society, Faro, Portugal
[6]Email correspondence: ikhmelin@ualg.pt

## Summary

Background: The COVID-19 pandemic priorities have focused on prevention by detection and response. National governments' prevention response decisions are based upon detection statistics from PCR tests that are used to define numbers of (i) COVID-19 infected persons, (ii) COVID-19 hospitalisations, and (iii) COVID-19 deaths. These statistics assume *a priori* that PCR tests are nigh 100% true detectors of COVID-19 infections. Here we will provide an alternative interpretation, along with the compelling evidence, that false positives have distorted to some degree the statistics of the primary outbreaks, and account for almost the whole of the 2nd and subsequent apparent COVID-19 outbreak peaks in various countries.

Methods: We extract from the published literature on PCR-test outcomes graphical data that reveals the evidence for a very large percentage of false positive results. We review the role of exosomes in the immune response to all respiratory viral infections and its effect on PCR tests. We hypothesise that exosomes, triggered by all viral respiratory infections, are largely responsible for positive outcomes from PCR-tests for COVID-19. We test our alternative interpretation for consistency with the empirical epidemiological trends as published by WHO.

Findings: We find that PCR testing data for the second and following waves of COVID-19 pandemic indicate that these waves are mainly artefacts of false-positive results. We find that this interpretation provides a more consistent explanation of the known epidemiology of COVID-19 than the hitherto consensus notion of extremely contagious and rapidly mutating viruses.

Interpretation: The RNA code counted in PCR tests, previously attributed to SARS-CoV-2, belongs instead to a respiratory-virus-induced immune system response by human cells that liberate exosomes, and that vitiate PCR test results. PCR tests have zero specificity *in vivo* due to the exosome RNA. PCR tests exhibit excellent specificity

*in vitro* on pure samples of other respiratory viruses. Low success rate of vaccines is explained by inexact identification of the SARS-CoV-2 RNA.

Funding: NONE: the Ossónoba Philosophical Society (OPS) investigates topical issues in physical, chemical, and biological sciences, in-keeping with the scientific method protocol, objectively, and without regard of any financial motive, political control or any subjective pressures. www.ossonobaphilosophicalsociety.org

Key Words: COVID-19; PCR test; false positive; SARS-CoV-2-RNA; exosomes

## INTRODUCTION

Recently the World Health Organisation (WHO) published a document[1] that called attention to the relevance of false positive results of Reverse transcription (RT-) Polymerase Chain Reaction, (PCR) tests for a SARS-CoV-2 virus, the causing agent of respiratory disease commonly known as COVID-19. PCR tests are used to directly screen for the presence of viral RNA, which will be detectable in the body before antibodies form or symptoms of the disease are present. During PCR testing for COVID-19, substances known as reverse transcriptase or DNA polymerase are added to a nasopharyngeal sample in a lab. These substances work to make numerous copies of any viral RNA that may be present. This procedure ensures enough copies of the RNA are present to signal a positive result, as specifically designed primers and probes attach themselves to sequences of the genetic code of the virus to signal that a pathogen has been found.

The WHO publication[1] is a reminder that the disease prevalence alters the predictive value of test results. As disease prevalence decreases, the risk of false positive increases. This means (*quote*) "that the probability that a person who has a positive result (SARS-CoV-2 detected) is truly infected with SARS-CoV-2 decreases as prevalence decreases, irrespective of the claimed specificity" (underlined by us). The WHO alarm notification has been vindicated by a field-study investigation in UK.[2]

In layman terms, technical specifications provided by test producers become irrelevant at low SARS-CoV-2 prevalence. The producers may claim their test is 100% specific to the SARS-CoV-2 virus, but in practice if COVID-19 prevalence in the population is low or zero – as may well happen seasonally – each and every positive result will be a false positive, reducing the amount of information contained in it to zero.[3] Note, however, that since the start of the pandemic, the WHO has been proclaiming that more extensive testing is necessary[4], which could conceivably generate ever repeating unreal 'COVID-19 outbreaks'.

The warning signals already have important consequences for public health institutions, which could have far-reaching damaging effects not just to public health services but to the general economy, of political decisions based upon PCR test statistics. We note the following 4-point established facts at the outset.[1-4]

i) Irrespective of the test specificity claimed by the test manufacturer, PCR tests produce false positive results in appreciable numbers;

ii) The fraction of false positive results in all positive results increases as the prevalence of the SARS-CoV-2 virus in the population decreases, e.g. due to seasonality of all other respiratory infections;

iii) In the limiting case of zero prevalence of SARS-CoV-2 virus, all positive PCR test results will inevitably be false positives; hence,

iv) PCR tests can never indicate that SARS-CoV-2 virus went out of circulation, as false positives will be appearing indefinitely, indicating alleged presence of the virus.

Accordingly, we have investigated a more plausible explanation for the development of COVID-19 pandemic data since 2019, based upon the alternative interpretation that exosomes play a role in false positive tests of patients that do not carry the COVID-19 virus. We find that the prevailing interpretation of PCR test results does not withstand scientific scrutiny in the light of the experimental or factual observations.

## Research in Context

| Evidence before this study | Added value of this study |
|---|---|
| The COVID-19 pandemic appears to be caused by a highly contagious and quickly mutating virus, with new variants, rapidly emerging all over the world, causing new outbreaks of the pandemic, including the disease in vaccinated persons, and repetitive infection in previous COVID-19 patients. The implication is that the pandemic is a never-ending cycle of new waves, mass PCR-testing and vaccinations programs. | The first wave of COVID-19 that developed in Europe and US in March-May 2020 was largely real, while two or more subsequent waves are an artefact of false-positive results of PCR tests that are not indicative of rampant, or indeed significant, levels of infection by the SARS-CoV-2 virus in population. The implication is that we can safely stop PCR-testing and mass vaccination of healthy people and end restrictions. |

## EXOSOMES

The important role of exosomes in the functioning of the immune system challenged by a viral or bacterial respiratory disease is established science[5], although the precise function and molecular mechanisms remain a topical research question[6]. Cells challenged by viruses or bacterial toxins produce exosomes, apparently signaling the immune system into action. Exosomes are structurally like a flu virus and contain some information in form of RNA and some attached proteins that provide structural integrity and acceptance by the target cells of the immune system. The immune response to respiratory infections is evidently triggered by the appearance of exosomes[5,6].

The trigger may also involve individual airway epithelial cells, at least partly, as exosomes were found to contain viral proteins, although only not in the beginning of the illness, indicating that interaction with the immune is necessary before that happens. Generally, foreign viral RNA may be identified by comparing it to cell's own

genetic material. Such function requires specialized cellular machinery, hardly compatible with normal physiological function of epithelial cells.

Exosomes, however, contain the answer to the 'where' question, and information contained in the exosome RNA should uniquely identify the virus-challenged airway epithelial cells, allowing the immune system to prepare its targeted response. The exosome RNA structure is probably independent of the pathogenic virus and varies only in conformity with the patients' individual genome.

These patient genome variations consistently explain several observations, which the consensus hypothesis attributes to the special properties of SARS-COV-2. Indeed, any test would produce negative outcome for exosomes whose RNA is different from that of the exosomes used for developing the test. This explains the 40% false negatives reported in China at the outset of the pandemics, in patients with classical clinical symptoms of COVID-19.

On the other hand, sequencing of the perceived viral RNA in remote locations necessarily reveals new exosome strains, caused by human genetic variability, but misidentified as new SARS-COV-2 variants. Once tests detecting such exosomes are developed and deployed, these perceived virus variants get instantly discovered everywhere, creating a false and terrifying impression of their fast propagation. The apparent propagation rate of such new variants will only be limited by the throughput of the testing system. Note that many differing human genomes coexist in various geographic locations due to modern population mobility.

These variant-specific tests would also produce false negatives on patients with differing genomes, albeit on genomes different from the undetectable genomes of the original tests. The exosome origin of the RNA attributed to SARS-COV-2 and its variants explains the fact that PCR tests used on patient biological samples are completely non-specific as regards the virus. Indeed, these tests are specific to exosome RNA, which probably carries no virus information, as postulated above. This, in turn, explains a misdiagnosis phenomenon, that manifests itself in countless observations, the more salient of which we itemise and outline in the following section.

## OBSERVATIONS EXPLAINED

We describe some perceived infection cases as 'false positive multiplication by compulsory contact testing' (FPMCCT), which requires no further explanation. The second class of false positives is one of the key points of the present paper and is described as illnesses caused by seasonal respiratory viruses, including variants of influenza A and B, common cold rhinoviruses, human corona viruses, etc.[7] all being 'misdiagnosed by PCR tests as SARS-CoV-2 virus' (MDSCV), with very high probabilities, close to 100%. This phenomenon will be discussed below in more detail.

The following published records of information were obtained from mainstream media and verified with Our World in Data[8], wherever quantitative data was called for. The order in which the following salient questions are asked is arbitrary.

| WHO Observation[8]/Question | OPS* Explanation |
|---|---|
| **1) New variants** like English (alpha), South-African (beta) or Indian (delta) very quickly propagate all over the world causing new outbreaks of COVID-19.<br>Has COVID-19 virus propagated via asymptomatic virus carriers, who were not detected by PCR tests, suggesting more testing is required to isolate and quarantine ALL virus carriers? | Once PCR tests detecting new variants are deployed, false positives appear due to MDSCV enhanced by FPMCCT producing exponential growth of false positives, and an apparent outbreak of the new virus variant in all locations where new tests are used. |
| **2) Patients previously infected or vaccinated** against Covid-19 are diagnosed with SARS-CoV-2 or its variants. Is the virus mutating so quickly that immunity created by a previous contact with SARS-CoV-2 or vaccination cannot prevent new illness? | One or both positive results of SARS-CoV-2 test could be false, therefore neither the existence of previous immunity, nor that an immunised person has contracted COVID-19 can be confirmed |
| **3) More testing** is not reducing SARS-CoV-2 apparent prevalence; e.g. Lisbon implemented a free testing program but produces higher rates of new cases than the rest of Portugal. Lisbon is a large place where virus propagation is facilitated by numerous contacts each person experiences daily.<br>Is more testing required to isolate and quarantine all asymptomatic virus carriers? | Free testing inevitably leads to more testing; more testing produces more false positives, with FPMCCT generating an exponentially growing outbreak. Lisbon has been hit by the 2nd wave harder than most other EU cities. |
| **4) New cases** in the second and later waves are frequently not traceable to contacts with any detected Covid-19 cases because there are so many asymptomatic carriers that it's impossible to detect them all. Is more testing required to stop the pandemic? | False positives appear in absence of SARS-CoV-2 in random persons who never had any contact with it and therefore are not traceable. |
| **5) Transmission frequency:** most frequently SARS-CoV-2 transmissions have occurred within the household, since at home people are not required to isolate from each other. Were public health measures successful in reducing virus transmission outside families? | Repetitive testing of family members in contact with symptomatic patients produces more SARS-CoV-2 false positives than normal. Public health measures can't affect numbers of false positives, nor the apparent development of new COVID-19 outbreaks. |
| **6) Severity of contact restrictions** does not affect the pandemic scale of the second and subsequent waves; e.g. | Numbers of false positives are only affected by number of tests made, but not by public health measures. The scale |

| | |
|---|---|
| compare Sweden (least-severe measures) to Netherlands (most-severe measures). Is SARS-CoV-2 so contagious it manages to percolate through any public health measures and develop another full-blown outbreak? | of the apparent outbreak is thus only limited by the throughput of the testing system. All EU countries are similar in this regard, making Swedish outbreaks statistically similar to the Dutch outbreaks. |
| **7) Wave dynamics** of COVID-19 has been very different between the first and subsequent pandemic waves. Public health measures were not deployed fast enough to affect the development of the first wave, which was a classic flu wave – 4 weeks up, 7 weeks down in EU.<br>Have subsequent waves developed much slower because of adequate public health measures already in place? | Dynamics of the first wave, a classic flu wave, was generated by SARS-CoV-2 transmission and little affected by public health measures, including lockdowns. Influenza A (H1N1) propagation was also little affected by equivalent public health measures in US in 1918. The dynamics of subsequent COVID-19 pandemic waves was slower, because it was generated by MDSCV enhanced by FPMCCT. |
| **8) Mortality rates** of subsequent infection waves are an order-of-magnitude lower than the first wave.<br>Is SARS-CoV-2 virus is quickly evolving to become less deadly and more transmissible? | Most cases in the subsequent waves were generated by MDSCV enhanced by FPMCCT, irrespective of the actual presence of SARS-CoV-2. Mortality statistics are therefore incorrect as regards attribution of deaths to a particular virus. Many are attributed to the wrong virus, or may have non-viral causes. |
| **9) Faster evolution** worldwide of SARS-CoV-2 virus variants, compared to other flu viruses, makes it impossible to create a durable immunity. Will more frequent vaccination be required? Illness-acquired immunity is presently valid for 6 months only e.g. in Russia. Do we need yet stricter public health isolation and distancing policies to stop the variants? | Virus variants pop up apparently faster due to MDSCV enhanced by FPMCCT. Any variant-sensitive PCR test generates false positives and an apparent outbreak once used for mass testing. |
| **10) Other respiratory viruses**, including, influenza A, B, drastically reduced their prevalence when SARS-CoV-2 spread, according to WHO-FLUNET. Were public health measures successful in also reducing the prevalence of all respiratory viruses, excepting SARS-CoV-2, which is so virulent it propagates regardless of attempts to stop it? | All symptomatic patients with respiratory illness are PCR-tested for SARS-CoV-2, and once it is detected due to MDSCV, not tested for anything else. Placed into wards with patients that do have SARS-CoV-2, their false positives become real positives, thereby increasing the prevalence of COVID-19 in pandemic data. |

| | |
|---|---|
| **11)** In **Israel and UK** (e.g.) almost entire adult population has been vaccinated, but an outbreak of Covid-19 is apparently starting in both, as of June 28, 2021.<br>Does SARS-CoV-2 mutate so quickly, that the vaccines are no more efficient against its new upcoming variant(s)? | New Covid-19 outbreak in Israel and UK results from MDSCV enhanced by FPMCCT and therefore unaffected by SARS-CoV-2 vaccines. |
| **12) China** never had serious national-scale outbreaks of Covid-19. Did strict public health measures, implemented in China eliminated SARS-CoV-2 from Chinese population? Is it necessary to continue to test and quarantine arriving travellers to keep the virus out of China? | China declared successful its combat against SARS-CoV-2 and stopped mass testing; hence no false positives and thus no false subsequent outbreaks are possible without mass testing. |
| **13) India** started mass testing with western aid, in summer 2021, which caused a fast growth in recorded Covid-19 cases.<br>Did this extended testing lead to the discovery of more cases, helping to isolate and eradicate the disease? | More testing results in more false positives due to MDSCV enhanced by FPMCCT producing an exponentially growing outbreak of false positives, only limited by throughput of testing system. |
| **14) Asymptomatic cases** of COVID-19 are prevalent, apparently making illness detection and containment more difficult. If SARS-CoV-2 "hides" in the organisms of asymptomatic carriers, avoiding detection, will yet more testing be needed to combat the pandemic? | Most of asymptomatic carriers are false positives due to MDSCV with no SARS-CoV-2 in their body. FPMCCT causes exponential growth of false positives and new outbreaks. |
| **15) First wave of COVID-19** occurred in spring 2020, which is the season for human corona viruses, but there was no similar outbreak in spring 2021.<br>Could COVID-19 outbreaks appear in any season due to high contagiousness of SARS-CoV-2 and its variants? | The first wave was real but inflated by false positives later. Subsequent waves are generated by MDSCV enhanced by FPMCCT, producing an apparent outbreak of COVID-19 for each of the respective seasonal respiratory viruses. |
| **16) Ratio of mortalities to infections** dropped so dramatically, up to the point that the March 2021 wave had no accompanying excess deaths whatsoever.<br>Is SARS-CoV-2 evolving and mutating and | Second and subsequent waves are mostly caused by less dangerous seasonal respiratory viruses, generating apparent SARS-CoV-2 'outbreaks' by MDSCV enhanced by FPMCCT, with lower mortality rates. In the extreme |

| | |
|---|---|
| so quickly becoming more contagious and less deadly? | case with only FPMCCT 'outbreaks' appear anytime, while mortality is zero. |
| **17) Vaccinations are ineffective** on dynamics of subsequent waves (see UK last wave as of July 2021). R values continue similar, etc. With 70% of people vaccinated, one would expect much slower growth, R' = 0.3R. In some cases the latest outbreaks are even larger.<br>Is SARS-CoV-2 is evolving so quickly, that it leaves the existing immunity behind, so that new outbreaks are little affected by vaccination? | Currently developing outbreaks are caused by seasonal respiratory viruses, generating apparent SARS-CoV-2 outbreaks by MDSCV enhanced by FPMCCT. These seasonal viruses are not stopped by currently available COVID-19 vaccines. |
| **18) Viral pneumonia** patients produce negative PCR test results for SARS-CoV-2. with probability of about 40%.<br>In the course of the illness does the virus move to where it cannot be sampled by the testing equipment, leading to negative test results? | PCR tests are not sensitive to this virus, but are sensitive to the response produced by human airway epithelial cells challenged by the virus. The RNA contained in this response (exosomes) varies from patient to patient and is not detectable by tests in some patients. Apart from high false-positive rate, PCR also has high false-negative rate. |
| **19) Repetitive positive test** results could be obtained for weeks and months, with total absence of any symptoms and in continuing isolation.<br>Does virus survive for a long time in asymptomatic carriers who slip thought the testing net and infect many other people? | Tests do not detect the virus but the response produced by human airway epithelial cells challenged by the virus, see the discussion below; this response may persist in some virus-free persons for many weeks. |
| **20) The WHO 'start testing' directive** was announced by WHO Feb. 2020 to be ready as virus propagates very quickly and cases of COVID-19 started appearing in March 2020 efficiently in Western Europe, US and all over the world, propagated by asymptomatic carriers. So when testing was started, was the COVID-19 virus already rampant in many countries before it started to be detected? | Initially tests mostly detected other respiratory viruses circulating in the population due to MDSCV amplified by FPMCCT, so that propagation of SARS-CoV-2 had little effect on the apparent onset of the first Covid-19 wave in Western Europe and US. Other countries did not have many tests available already in March, which delayed the apparent start of the first wave till mass testing could be implemented. |
| **21)** N**ew COVID-19 outbreak** is growing in European countries in July 2021, | What is observed is a vaccination-caused illness. Patients vaccinated against the |

| | |
|---|---|
| causing hospitalization of patients in the 30 to 50 age range.<br>Is highly virulent Delta variant of SARS-CoV-2 spreading so quickly all over the world, and yet the existing vaccines are not very efficient against it? | immune response of their own cells develop autoimmune reaction. Younger people with stronger immune system are at higher risk of autoimmune reaction. |

## SCIENTIFIC METHOD CRITERIA

Based upon observation, question, hypothesis criteria of the scientific method, we can analyse the pandemic data, with WHO observations/questions, and alternative OPS explanations, itemised in the previous section, objectively. From the point of view of the scientific method defined above we note that is has not been adhered by scientific advisors to many national governments that have already answered "yes" to the many questions raised by the WHO in *ad hoc* knee-jerk responses to the pandemic.

The interpretations provided by OPS explanations are compliant with the scientific-method criteria. Unlike WHO, we are not using any alleged or unsubstantiated properties, e.g., hypothetical rate-of-transmission, of SARS-CoV-2 variants. We refer only to information demonstrably correct; however, we question the reliability of analytical tools existing at the present time, like dubious PCR tests[1]. However, the proposed role of exosomes in the false-positive PCR test results will remain a hypothesis that needs to be investigated with some circumspection for more direct laboratory experimental evidence that it is the agent responsible for vitiating PCR test results.

The WHO interpretations require the following hypothetical assumptions for which there is presently no compelling evidence, and which go against the principle of Occam's razor:

(i) Transmission by asymptomatic COVID-19 carriers
(ii) Tests working and being 100% specific in clinical *in vivo* practice
(iii) Vaccines are effective: vaccinated people are getting 'infected'
(iv) Effectiveness of vaccines defined by PCR test and/or antibody test?
(v) The same virus generating completely different outbreak dynamics in the first and subsequent waves

On the other hand, the OPS explanations only assume established scientific knowledge that has been conclusively demonstrated:

(vi) PCR tests produce false positives both on different respiratory viruses and on patients having no respiratory viruses at all
(vii) Contact PCR testing amplifies number of tests and of false positives
(viii) Classic, or nearly Gaussian, dynamics of the first (SARS-CoV-2) wave in 2020 (March-May in Western Europe), and the slower-skewed testing-generated dynamics in the subsequent waves

The observations, and consensus interpretations that we question above, are somewhat contradictory. Compare, for example, items 5, 7 and 10, in which the

assumptions that public health measures were both sufficient and insufficient, to reduce propagation of SARS-CoV-2 are questioned. Also, WHO-observations of item 7 are contradictory in themselves, because strict self-confinement and isolation were imposed already at the onset of the first wave, but inexplicably turned out entirely inefficient, resulting in a classical dynamic of epidemic flu, i.e., 4 weeks up and 7 weeks down, instead of the publicly promised “flatten the curve” with “reduced load on health service system”.

Considering items 2, 9 and 11, SARS-CoV-2 is physically akin to other flu viruses, and its RNA mutates at a similar rate due to RNA reproduction errors in human cells. Therefore, it is unable to produce significantly different variants capable of overcoming the existing immunity much faster than common cold and other seasonal respiratory viruses, where such major variants come up no more frequently than once in a few years at least. Indeed, dozens of variants of influenza A exist, with immunologically different updates to those major variants taking many years to appear, which explains large intervals between severe outbreaks of influenza A. Therefore, the explanation provided by WHO-consensus hypothesis must be erroneous.

Moreover, considering items 6 and 10, any public health measures cannot affect SARS-CoV-2 any differently from common cold and other seasonal respiratory viruses, for the simple reason that all these viruses are physically indistinguishable and can only propagate within aqueous droplets, losing virility once the droplets dry out. Therefore, if a surgical mask can stop one of the viruses because it stops all droplets, it will similarly stop all others. In fact, we have known for more than 100 years that masks do not affect influenza propagation[9]. Therefore, the explanation provided by the WHO is quite demonstrably untrue.

From experimental evidence item 11, infection statistics for UK and Israel, is inconsistent at the time of writing. Figure 1 shows the plot of new COVID-19 cases in Israel and UK, which were the most vaccinated countries, almost all adult population, at the time of writing.

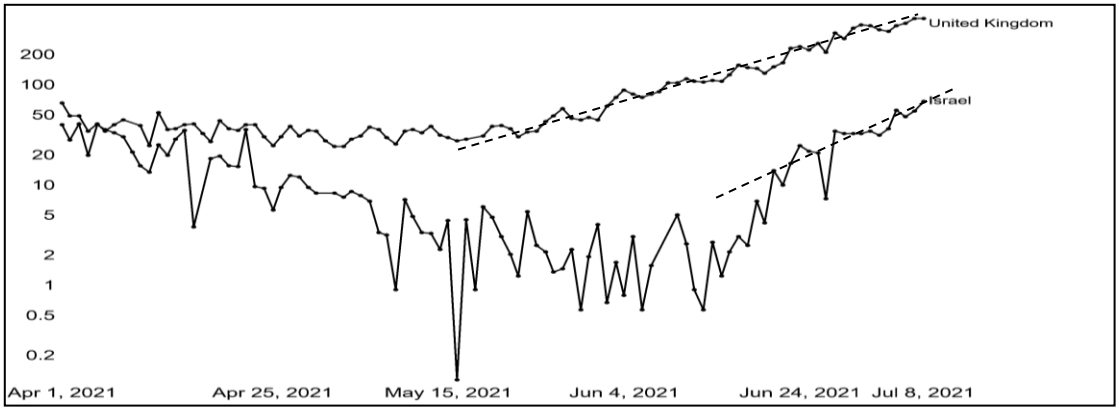


**Figure 1: Daily new Covid-19 cases in Israel and UK, per million population; data taken from Johns Hopkins University, and the plot adapted from Our World in Data [5]. Note the logarithmic scale. Dashed lines indicate the rate of outbreak development, similar in the two countries. Both countries almost completed their vaccination programs.**

Note that Israel has slightly higher percentage of complete vaccination than UK, yet lower PCR-positive case numbers at present. However, in Israel, the apparent case infection rate is growing faster than in UK and is set to overcome UK numbers in the matter of weeks. Therefore, vaccination has no apparent effect on the development of the current case numbers, exactly as predicted by the false-positive explanation.

Finally, we also note that WHO-consensus and OPS-explanation produce very different predictions of the future development of the Covid-19 pandemics, and very different public health recommendations. We discuss these differences in the last section on conclusions and recommendations.

## EXCESS MORTALITIES

Note that there are no valid RNA tests capable of discriminating covid-19 from other flu-like diseases, as demonstrated above. Therefore, no information on epidemiology of covid-19 may be obtained from published case statistics, which is all based on RNA tests. For these reasons, excess mortality statistics was considered instead. Excess mortality usually exhibits peaks associated with viral respiratory diseases in autumn and winter seasons. However, additional peaks appeared in spring in the years 2020 and 2021, attributable to SARS-COV-2 as will be discussed below.

### Eastern Europe

Figure 2 shows excess mortality in selected Eastern European countries in 2020-2021. Apparently, these countries did not have adequate meteorological conditions for covid-19 transmission in spring of 2020, and therefore had no significant associated mortality in that period. This demonstrates that even in its first outbreak, SARS-COV-2 was transmitted as a seasonal flu virus, requiring adequate meteorological conditions, and was not transmitted outside its preferred season, contrary to what may be inferred from statistics of the new cases derived from invalid RNA tests. It is therefore concluded that SARS-COV-2 is only prevalent in spring, in the same way as all of the previously known human coronaviruses.

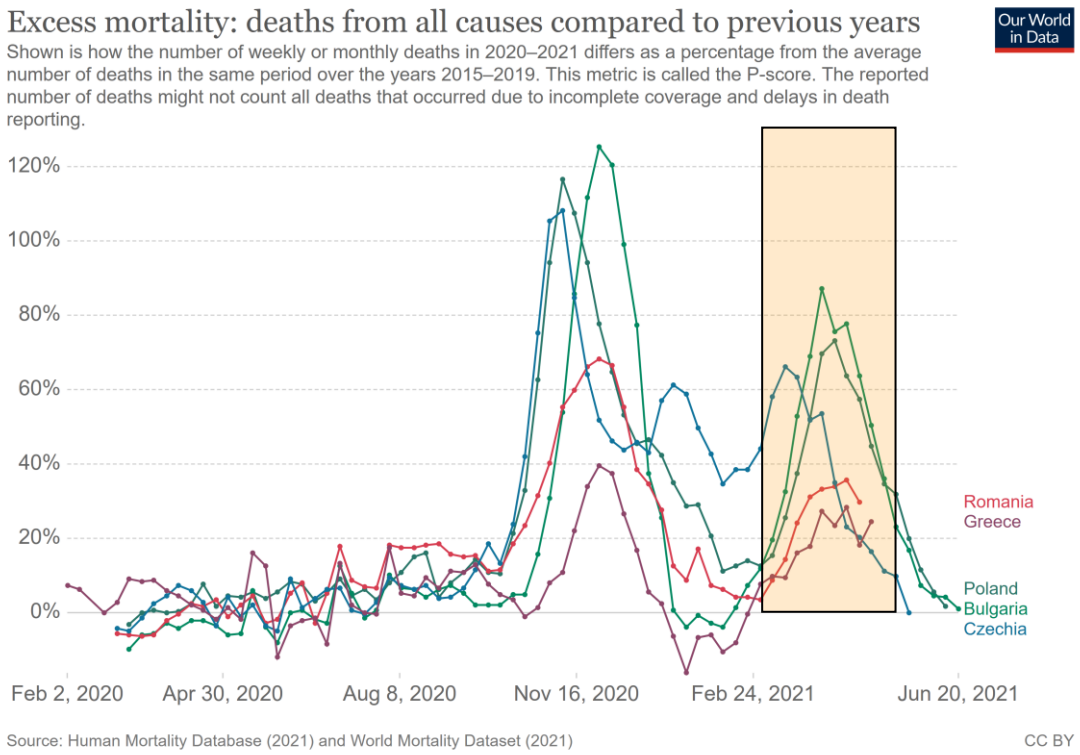


**Figure 2: Weekly excess mortality in representative Eastern European countries in 2020-2021. Plot adapted from Our World in Data https://ourworldindata.org/explorers/coronavirus-data-explorer Rectangle: February 28 to May 16, showing expected coronavirus prevalence in spring 2021.**

Thus, within the time interval considered, Eastern European countries had a rhinovirus peak in autumn of 2020 and their first coronavirus peak in spring 2021. Czechia also had a discernible flu A peak, with its maximum on January 10. It is possible that rhinovirus mortality was amplified by clinical diagnostic errors induced by results of invalid PCR tests that signalled covid-19 in rhinovirus cases.

## Western Europe

Figure 3 shows excess morality in selected Western European countries. Contrary to Eastern European countries, Western European countries apparently had suitable meteorological conditions for covid-19 outbreak already in spring of 2020 and strong peaks of excess mortality in this period. These peaks occurred during the first wave of covid-19 in Europe. The mortality peaks of the second covid-19 outbreak in spring of 2021 were significantly smaller, about 25% of the first outbreak, indicating that population-wide immunity was already quite advanced after the first outbreak. Note that other human coronaviruses do not produce strong mortality peaks in spring; therefore, their mutations should be significantly slower than those of flu A. It is therefore reasonable to expect that spring mortality peaks associated with covid-19 will also disappear in a year or two, after sufficient immunity is accumulated in the population. On the other hand, it seems reasonable that stronger coronavirus mortality peaks were associated to the newly appeared coronavirus, as has happened with SARS-COV-2. The second outbreak in UK did not appear at the same time as that in other countries, occurring probably about 2 months earlier, due to significantly different climate. The excess mortality peaks in autumn 2020 and winter

2020-2021 are attributable to rhinovirus and flu A. The small and sharp mortality peak appearing in Germany, Netherlands and France in August 2020 is attributable to the heat wave.

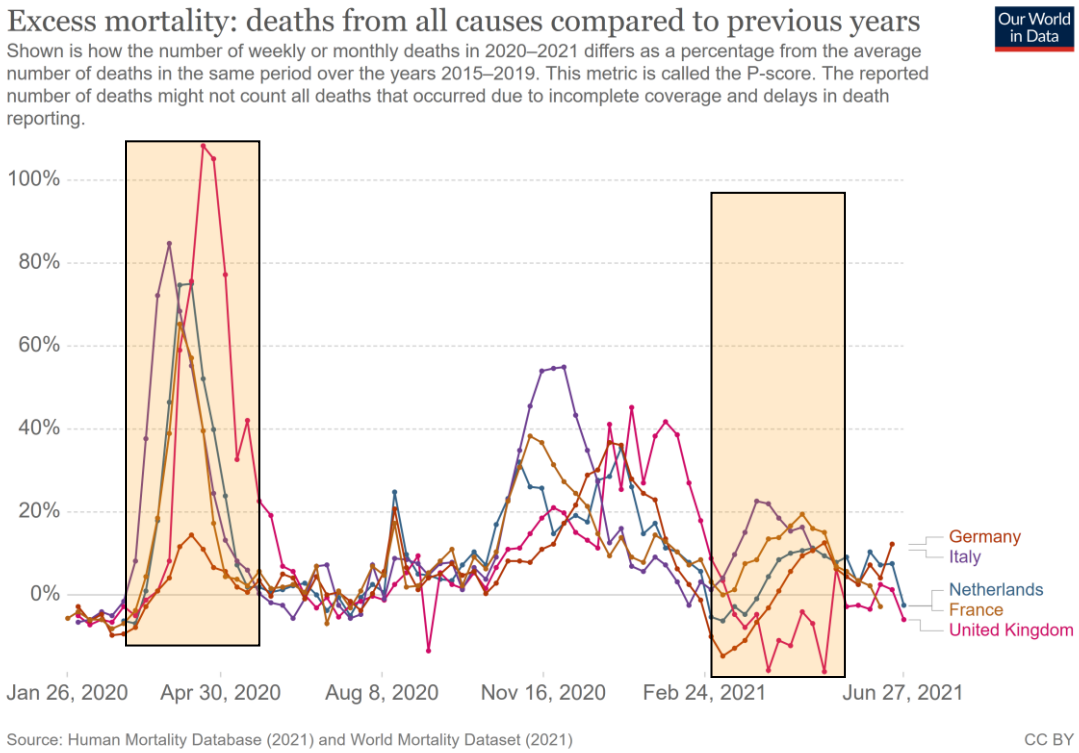


**Figure 3: Weekly excess mortality in representative Western European countries in 2020-2021. Plot adapted from Our World in Data https://ourworldindata.org/explorers/coronavirus-data-explorer Rectangles: March 1 to May 24, 2020 and February 28 to May 16, 2021, showing expected coronavirus prevalence during spring season.**

It is therefore concluded that SARS-COV-2 is quite similar to other coronaviruses, in that it appears only seasonally, in most European countries in spring, and its associated excess mortality is on the way to extinction in a few more years, indicating rapid acquisition of population-wide immunity. There are no indications of rapid mutations of this new virus, which would slow down the observed reduction in the spring excess mortality. Additional excess mortality in seasons with low SARS-COV-2 prevalence is apparently induced by utilization of the results of invalid RNA tests in the clinical practice, which leads to exaggerated scale of pandemics and misdiagnosis of other respiratory viruses as SARS-COV-2. This may cause contamination of patients already carrying a different respiratory virus by SARS-COV-2 in hospitals, where the virus may be surviving and propagating even outside its preferred season, due to the air-conditioned environment facilitating virus transfer from authentic virus carriers.

## CONCLUSIONS WITH RECCOMENDATIONS

Our revised explanation for the various observations of the COVID-19 epidemiology, along with predictions for the development of COVID-19 pandemic are so far entirely consistent with all known experimental observations as evidenced by published statistical data. We have been unable, as yet, to conduct clinical experiments to confirm the reality of the MDSCV, however, researchers in St. Petersburg have obtained experimental evidence that a large fraction of COVID-19 patients has additional respiratory viruses in their system[10]. To avoid an infinite sequence of pseudo-COVID-19 outbreaks and constant mass revaccinations, mass testing for SARS-CoV-2 and its variants should be immediately discontinued, along with futile attempts to identify asymptomatic carriers, and the respective resources used to provide remedy for chronic patients, some with other pathogens, who were left without medical help by reorienting all medical service towards COVID-19 pandemics.

MDSCV phenomenon may be tested for in the laboratory by infecting human volunteers with known respiratory viruses other than SARS-CoV-2 and using standard PCR tests for SARS-CoV-2 on those who develop symptoms, in a strictly quadruple-blind experimental design. In an approach not requiring volunteers, patients diagnosed with SARS-CoV-2 may be retested for seasonal respiratory viruses, which will be present in most cases producing what is erroneously interpreted as COVID-19 due to false-positive results of PCR tests.

We are finally left with a striking contradiction between excellent specificity of SARS-CoV-2 tests demonstrated on *de facto* samples of other respiratory viruses *in vitro* as per the information provided by the test producers, and the apparently zero specificity of the same tests revealed *in vivo* in clinical practice, as demonstrated here. To address this contradiction, note that all tests (and vaccines) were produced using the genetic information published by the SARS-CoV-2 discoverers in the appropriate databases.

Consulting the respective seminal publications[11], we find that the respective genetic material had been identified computationally without preparing an isolate of the respective virus particles, and without separating them physically from other carriers of genetic material that may be present in the biological samples[11]. Noting that tests apparently produce false positive results in people carrying some respiratory virus different from SARS-CoV-2, we must conclude that the alleged genetic code of the SARS-CoV-2 virus had been wrongly identified, belonging instead to something generated by human airway epithelial cells challenged with respiratory viruses and containing RNA, for instance, to exosomes, as explained above. Note that identical exosomes would be detected in pneumonia of bacterial origin.

It is no surprise, therefore, that the tests are totally non-specific in the clinical practice, while demonstrating excellent specificity *in vitro*: samples of other respiratory viruses used for *in vitro* trials were not contaminated with products of human cells, whereas all biological samples used to identify RNA code of SARS-CoV-2 have been in contact with such cells[11]. Note also that SARS-CoV-2 RNA had been identified by its similarity to that of another virus, which casts reasonable doubts on that previous identification.

It appears also that the RNA codes of SARS-CoV-2 variants, very similar to that of the original COVID-19 virus, have also been wrongly identified. Given that the alleged SARS-CoV-2 RNA is in fact generated by human airway epithelial cells used

for virus culturing[11], it is possible to explain high rates of false negative results in Covid-19 patients. RNA induced by the virus in challenged human cells may vary from patient to patient, due to individual genetic differences, making it not recognizable by the test.

Having established an erroneous identification of the genetic material belonging to SARS-CoV-2, we can interpret low success rates of all the existing vaccines, requiring multiple doses to produce reasonable immune response. Indeed, the vaccines are based on the genetic material, probably exosomes, generated in human airway epithelial cells challenged by respiratory viruses, and not on the genetic material of the SARS-CoV-2 virus itself. Immunity generated by such vaccines can only act in advanced stages of Covid-19 illness, amplifying the already existing response of the patient immune system, which could be too late for patients whose immune reaction is belated. These vaccines may also exacerbate problems in patients with other diseases that induce cell response akin to that generated by respiratory viruses, probably explaining some of the adverse reactions to vaccination amongst younger recipients.

**Contributors** IK and PS guided and performed all the initial observation and data analysis. IK guided the conceptualisation and investigation, wrote the first draft of the Article. LVW guided the writing, review, and editing, and contributed to the interpretation sections. All authors contributed to the analysis of the data used and/or referenced, and to the acquisition, and drafting results and revising it critically for important intellectual content. All authors had full access to all the data in the study and take full responsibility for the decision to submit for publication.

**Declaration of interests** We declare no competing interests.

***Acknowledgments** This study was performed under the auspices of The Ossónoba Philosophical Society (OPS). www.ossonobaphilosophicalsociety.org

**References**

1 WHO Information Notice for IVD Users 2020/05, Nucleic acid testing (NAT) technologies that use polymerase chain reaction (PCR) for detection of SARS-CoV-2, 20 January 2021, https://www.who.int/news/item/20-01-2021-who-information-notice-for-ivd-users-2020-05, consulted 25/03/2021.

2 Healy, P.,Khan, A., Metezai, H., Blythe, I., Asad,H., The impact of false positive COVID-19 results in an area of low prevalence, Clinical Medicine (Lond.) 21(1) e54-e56 (Jan. 2021) https://doi.org/10.7861/clinmed.2020-0839

3 Nickbakhsh, S. , Mair, C., Matthews, L. , Reeve, R., Johnson P. C. D., Thorburn, F. , von Wissmann, B., Reynolds A.. McMenamin, , J., Gunson, R. N., Murcia, P. R., Virus–virus interactions impact the population dynamics of influenza and the common cold, PNAS December 26, 2019, **116** (52) 27142-27150; first published December 16, 2019; https://doi.org/10.1073/pnas.1911083116

4 BBC, WHO head: 'Our key message is: test, test, test', March 16, 2020, https://www.bbc.com/news/av/world-51916707, consulted on July 6, 2021

5 Elrashdy, F., Aljaddawi,A.A., Redwan, E. M., Uversky, V., On the potential role of exosomes in the COVID-19 reinfection/reactivation opportunity, Journal of Biomolecular Structure and Dynamics 07 09 (2020) (see also references cited) https://doi.org/10.1080/07391102.2020.1790426

6 Bedford, J. G., Infusini G, Dagley L.F., Villalon-Letelier F, Zheng M.Z.M., Bennett-Wood, V, Reading P.C., Wakim, L.M. Airway Exosomes Released During Influenza Virus Infection Serve as a Key Component of the Antiviral Innate Immune Response. *Front Immunol.* 2020 May 12; 11:887. https://doi.org/10.3389/fimmu.2020.00887. PMID: 32477358; PMCID: PMC7236881.

7 Wikipedia, List of infectious diseases causing flu-like syndrome, https://en.wikipedia.org/wiki/List_of_infectious_diseases_causing_flu-like_syndrome, Consulted on July 6, 2021

8 Our world in data, Coronavirus data explorer https://ourworldindata.org/coronavirus-data-explorer, Consulted on July 5, 2021.

9 Lynch, C., and Cumming J.G., The distribution of influenza by direct contact – hands and eating utensils, American Journal of Public Health 1919, 9(1): 25-38 (p. 37, end of point 6). https://ajph.aphapublications.org/doi/pdf/10.2105/AJPH.9.1.25

10 Nikitin, N. A., Where has the flu A gone, Interview *in Russian*, Consulted on July 6, 2021. https://mirtesen.naukatv.ru/blog/43825814888/Kuda-delsya-gripp-?utm_referrer=mirtesen.ru&utm_source=mirtesen&utm_medium=partner&utm_campaign=65032&utm_content=10726261&utm_term=42910

11 R. Lu, X., Zhao, J. Li, P. Niu, B. Yang, H. Wu, et al., *Lancet* 2020; 395: 565–74, Genomic characterization, and epidemiology of 2019 novel coronavirus: implications for virus origins and receptor binding, https://doi.org/10.1016/S0140-6736(20)30251-8