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Why the Official Theory about COVID-19 Is Not Tenable

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Abstract

Notwithstanding the huge number of scientific papers published on COVID-19, there are still critically important aspects of the story that have not been sufficiently studied. The problems concern the definition, the diagnosis, and the tests used. They are all questionable. Even if they all were tacitly accepted as correct, the current mainstream theory remains untenable: it cannot explain what happened, i.e., the pandemic in the version presented, more or less, as a scientific consensus. The simple reason is that the virus named "SARS-CoV-2" – according to surveys conducted with the widely used test for its identification – was already widespread in pre-pandemic years all around the globe before its official "birth", and no one even noticed it. There is ample proof of this fact.

Keywords: *cellular immunity, COVID-19 pandemic theory, epidemiology, falsifiability theory, IgG versus IgM, RT-PCR test, SARS-CoV-2 specific antibodies*

Introduction

The number of scientific papers published about COVID-19 is impressive: by June 2025 over 480,000 articles had been registered on Pubmed in the 5 and a half years since the COVID-19 pandemic was first announced to be underway. It might, therefore, be assumed that the topic had been sufficiently explored in all its aspects. The COVID-19 phenomenon is, without question, the most studied infectious disease in the history of medicine. By comparison, there were far fewer publications on HIV-AIDS — just over 186,000, on PubMed during a period of no less than 44 years — a period beginning with the first reports of a new kind of pathology among US homosexuals (Centers for Disease Control and Prevention, 1981). Those papers establish a time frame that is 8 times longer than the time for publishing papers on COVID-19. Yet those who think

that everything has been clarified on this matter would be wrong: heavy doubts remain on the most central and pressing issues.

The sheer quantity of scientific research and production does not necessarily go hand in hand with quality. It is not disputed, for example, that more than half of the papers peer-reviewed and published have no value whatsoever (Ioannidis 2005, Lundberg 2018), and the rest are also not free from lingering problems. The exaggerated production of scientific papers in the absence of any serious examination of them remains an issue that had already been well known in the past, and since the emergence of the COVID-19 story, the dimensions of the phenomenon have only grown exponentially. There is also the human impossibility of even taking a quick look at all those published papers: a lifetime would not be sufficient for reading all of them. John Maddox, renowned Editor of *Nature* (for 22 years) was keenly aware of this problem and was deeply concerned about it. He wrote an editorial in 1988 with the evocative title, "Finding wood among the trees":

> Is there a danger, in molecular biology, that the accumulation of data will get so far ahead of its assimilation into a conceptual framework that the data will eventually prove an encumbrance? Part of the trouble is that excitement of the chase leaves little time for reflection. And there are grants for producing data, but hardly any for standing back in contemplation.

In search of such contemplation, I ask the following question: are there keys to a deeper comprehension of the COVID-19 issues buried in all the frantic scientific production that followed the announcement of the "pandemic"? What is certain is that much of it was not even peer reviewed but has, rather, consisted of preprints because of the announced and perceived "emergency".

Methods and Results

Search has been made in the scientific literature with the already mentioned key words. This is not a complete review, but a search in which only papers considered meaningful to the current debate are briefly described. The goal is to sift through some of the significant data using a sound methodological approach.

I began from the observation that there are consistent criticisms of numerous aspects of the story and they concern: a) the tests to determine the presence of SARS-CoV-2 (Gallagher 2020; Reuters 2020; Watson et al. 2020[1]; Borger et al. 2020; Kämmerer et al. 2023; Serpieri & Franchi 2024); b) the signs/symptoms of disease conditions attributed to COVID-19 (e.g., Franchi and Tomsic, 2023); c) the true-nature of the vaccines (e.g., Latypova, 2020; Segalla, 2024). The epidemiology in particular has been guided by a peculiar approach to case definition (WHO 2020; CDC 2023), which was extremely broad, and allowed the diagnosis of confirmed COVID in the presence of any clinical condition, including well-being (see Table 1). As the table shows, given a positive outcome of a debatable nucleic acid amplification test (NAAT), the condition in the first column of Table 1 would put the person in the class of patients with full blown COVID-19 disease. This table might seem like a joke, but instead it represents exactly what actually happened. An extreme example that made the news in Abruzzo, Italy, was the case of a 41-year-old man who died by drowning but a NAAT swab showed him positive for COVID-19 so he was "counted among the victims of the epidemic" on September 29, 2020 (see the report by Donat-Cattin, 2020). In spite of the absurdities and the

[1] Watson et al. wrote: "The lack of a clear-cut 'gold-standard' is a challenge for evaluating COVID-19 tests; pragmatically, clinical adjudication may be the best available 'gold standard', based on repeat swabs, history, and contact with patients known to have COVID-19, chest radiographs, and computed tomography scans. Inevitably this introduces some incorporation bias, where the test being evaluated forms part of the reference standard […]"

criticisms that followed, none of them seemed to lead to the necessary unambiguous conclusion that, I believe, it should have led to. Rather, all of the conflicting components of the COVID-19 phenomena merely gave rise to a complex of seemingly unending debates.

Table 1
Given the WHO Case Definition, Each Pathology, Condition, Injury or Death Was Classed As a "COVID-19 Confirmed Case" Whenever The Applied Nucleic Acid Amplification Test (NAAT) Obtained a Positive Result

Condition	NAAT Result	Diagnosis
Acute heart failure	POSITIVE	COVID-19
Acute heart failure	NEGATIVE	Acute heart failure
Interstitial pneumonia	POSITIVE	COVID-19
Interstitial pneumonia	NEGATIVE	Interstitial pneumonia
A pimple	POSITIVE	COVID-19 (potentially fatal)
A pimple	NEGATIVE	Only a zit
Death by drowning	POSITIVE	COVID-19 (fatal)
Death by drowning	NEGATIVE	Drowning fatality

So, here I propose another approach that escapes the fallacies of circular reasoning. Karl Popper (1934/1959) maintained that a theory is scientific only if it is exposed to the possibility of experimental testing or observations that might disprove it. A scientific theory must allow correct forecasts to be made. If its predictions are consistent with experimental observations, the theory is confirmed; otherwise, it must be discarded or modified. In fact, a good theory is judged by the past. What are its verifiable consequences? I am going to detail the following answer to these questions:

If the SARS-CoV-2 virus was a novel entity that did not exist until December 2019, it could not be found prior to that time, but it was found. The results of the present research are sufficient to conclude that the currently accepted theory is, thanks to the results I am reporting here, unequivocally falsified.

The Scientific Consensus About the Genesis of the COVID-19 Pandemic

In synthesis, the only possibilities considered for the pandemic, with its reported epicentre in Wuhan in December 2019, were 3: it had to be the outcome of a "natural, accidental, or deliberate" spread of SARS-CoV-2 (e.g., Gostin et al. 2023). SARS-CoV-2 was declared — without material proof [2]— to be the sole cause of the pathologies identified as, and/or associated with, COVID-19 [3]. Research

[2] The true isolation of the SARS-CoV-2 has been questioned by some researchers from the beginning. FOIA requests have been sent to various public institutions, asking for documents that could prove it. Not one was found and the whole collection of questions and answers was made public by Massey (2021).

[3] In fact, the question "which are the first three papers that show the causal correlation between the SARS-CoV-2 and the disease "COVID-19?" remains today without a proper answer, just as it was in 2020. The positivity of the reference test (RT-PCR) was not necessary nor sufficient for the characteristic symptoms and subsequent disease conditions of COVID-19, except by definition (WHO 2020a; CDC 2023; Franchi & Tomsic 2023), which involves the logical fallacy of circular reasoning.

in recency and primacy effects might help explain this cognitive bias toward that widely accepted explanation (Rubínová & Price, 2024).

The Widely Ignored Fourth Possibility ("the White Crow")

In fact, there is a fourth possibility, being well aware that we chose to remain in the viral paradigm without sharing it: the virus (identified by the positive result of PCR, supposedly the gold standard for the COVID-19 diagnosis) was present and widespread in Italy, even before the outbreak of the epidemic, without giving any sign of its existence: a fact absolutely not expected also according to the comments of the mainstream authors. We point out that Italy's first two cases of COVID-19 disease were recorded on January 30, 2020, when two tourists from China tested positive for SARS-CoV-2 in Rome. The first autocthonous (indigenous Italian) case was diagnosed in February, 18, 2020.

However, three studies would suggest another story, each one reinforcing the others:

1. The first study (La Rosa et al. 2021), reported in November-December 2019, concerned the presence of SARS-CoV-2-specific viral sequences in sewage water in cities far away from each other. If researchers were able to find it in the sewage water, with the dilution that is entailed, it means that the number of viruses must have been considerable, and the original diffusion must have begun much earlier.
2. In the second study (Apolone et al. 2021), antibodies were found in cryo-preserved sera from asymptomatic people, dating back to September 2019.
3. In the third study, autopsies performed in the pre-pandemic period in Milan by Lai et al. (2021) examined 169 of the deceased persons and found 5 of them to be positive for the presence of antigens/antibodies and/or viral sequences (all of which were discovered by PCR).

Apolone et al. (2021) wrote:

> We investigated the presence of SARS-CoV-2 receptor-binding-domain (RBD)-specific antibodies in blood samples of 959 asymptomatic individuals enrolled in a prospective lung cancer screening trial between September 2019 and March 2020 to track the date of onset, frequency, and temporal and geographic variations across the Italian regions. SARS-CoV-2 RBD-specific antibodies were detected in 111 of 959 (11.6%) individuals, starting from September 2019 (14%), with a cluster of positive cases (>30%) in the second week of February 2020 and the highest number (53.2%) in Lombardy. This study shows an unexpected very early circulation of SARS-CoV-2 among asymptomatic individuals in Italy several months before the first patient was identified and clarifies the onset and spread of the coronavirus disease 2019 (COVID-19) pandemic. Finding SARS-CoV-2 antibodies in asymptomatic people before the COVID-19 outbreak in Italy may reshape the history of pandemic.

LOGICAL CONSEQUENCES

One of the logical consequences is that — if we attribute meaning and reliability to antibody and genetic tests — the virus had to be present much earlier than claimed in the mainstream narrative (many months at least) in order to be so widespread in September 2019. Incidentally, the finding of IgM would mean a recent infection (within the previous 5 months, i.e. by April 2019) while IgG alone would indicate an older infection, more than 5 months earlier, perhaps even years earlier.

So, the virus (engineered or not) was found far from Wuhan and its laboratories at a time when the pandemic was far from having yet begun according to the accepted narrative, but the significance of

such findings seems to have gone unnoticed by the scientific community. It was already in circulation, while the "gain of function" (GoF) technology — applied to the coronavirus destined to become SARS-CoV-2 — was still in progress and there had not yet been any leak from the Wuhan laboratory.

In other words, the unique purported cause was present, but not the expected effect. That's an irreconcilable contradiction.

WAS IT AN ISOLATED ANOMALY?

No: there is ample proof of pre-pandemic viral presence in the world. There have been positive tests for SARS-CoV-2 in different populations, theoretically free from infection, on at least 5 continents. Here are 11 notable instances:

1. In France, Carrat et al. (2021) wrote:

> The first documented case in Europe was reported retrospectively in France in one patient with a diagnosis of pneumonia and a positive SARS-CoV-2 RT-PCR result on December 27, 2019. This report suggests that SARS-CoV-2 infection may have occurred as early as November 2019 in France.

2. In Germany, on pre-pandemic cellular immunity, Braun et al. (2020) reported:

> We identified S-reactive CD4+ T cells by flow cytometry according to their expression of CD40L and 4-1BB after in vitro stimulation with S peptides. . . . [also that] S-reactive T cells in RHDs are cross-reactive to hCoVs. Our study reveals pre-existing cellular SARS-CoV-2 cross-reactivity in a substantial proportion of SARS-CoV-2-seronegative HDs. This finding could have considerable epidemiological implications regarding herd immunity thresholds and projections for the COVID-19 pandemic.

3. In The Netherlands, T-cell reactivity dating from years before the "pandemic" was declared, was found by Grifoni et al. (2020):

> Understanding adaptive immunity to SARS-CoV-2 is important for vaccine development, interpreting coronavirus disease 2019 (COVID-19) pathogenesis, and calibration of pandemic control measures. Using HLA class I and II predicted peptide "megapools," circulating SARS-CoV-2-specific CD8+ and CD4+ T cells were identified in _70% and 100% of COVID-19 convalescent patients, respectively. [...] Importantly, we detected SARS-CoV-2-reactive CD4+ T cells in _40%–60% of unexposed individuals, suggesting cross-reactive T cell recognition between circulating "common cold" coronaviruses and SARS-CoV-2. All of the donors were recruited between 2015 and 2018, excluding any possibility of exposure to SARS-CoV- 2. Importantly, pre-existing SARS-CoV-2-cross-reactive T cell responses were observed in healthy donors, indicating some potential for pre-existing immunity in the human population.

4. In Russia, Gumanova et al. (2022) found:

> We investigated seroprevalence of anti-SARS-CoV-2-S1 receptor-binding domain (RBD)-specific antibodies in the serum samples in 2011–2021 [...]. The present study detected anti-SARS-CoV-2-S1 RBD-specific antibodies (3–6%) in the serum of participants recruited in 2011–2019, and these findings can contribute to our understanding of the origins of SARS-CoV-2.
>
> The sera were selected from recruited participants who were asymptomatic.

5. In the USA, Basavaraju et al. (2020) obtained similar findings:

> Methods. To determine if SARS-CoV-2–reactive antibodies were present in sera prior to the first identified case in the United States on 19 January 2020, residual archived samples from 7,389 routine blood donations collected by the American Red Cross from 13 December 2019 to 17 January 2020 from donors resident in 9 states (California, Connecticut, Iowa, Massachusetts, Michigan, Oregon, Rhode Island, Washington, and Wisconsin) were tested at the Centers for Disease Control and Prevention for anti–SARSCoV-2 antibodies.
>
> Results. Of the 7,389 samples, 106 were reactive by pan-Ig. Of these 106 specimens, 90 were available for further testing. Eighty four of 90 had neutralizing activity, 1 had S1 binding activity, and 1 had receptor-binding domain/ACE2 blocking activity >50%, suggesting the presence of anti–SARS-CoV-2–reactive antibodies. . . .
>
> Conclusions. These findings suggest that SARS-CoV-2 may have been introduced into the United States prior to 19 January 2020.

6. In Saudi Arabia, in October 2019 SARS-CoV-2 antibodies were unexpectedly found by Mahallawi et al. (2022) in the blood of Arabians:

> In conclusion, we provide evidence to support the unexpected early circulation of SARS-CoV-2 among persons who had visited China a few months prior to the pandemic declaration. These results support the emergence and spread of SARS-CoV-2 before the COVID-19 pandemic declaration. The detection of SARS-CoV- 2 antibodies in individuals prior to the reported pandemic eruption in China could rewrite the currently accepted timeline of the pandemic.

7. In Africa, Ioannidis and Contopoulos-Ioannidis (2023) provided a review of published papers signalling widespread pre-pandemic positivity. They wrote:

> Positivity was similar for anti-nucleocapsid (14%) and anti-spike antibodies (11%), higher for anti-spike1 (23%), and lower for anti-receptor-binding domain antibodies (7%). Positivity was similar, on average, for immunoglobulin M and immunoglobulin G.
>
> Conclusion: Prepandemic samples from Africa show high levels of anti-SARS-CoV-2 seropositivity. At the country level, cross-reactivity tracks especially with malaria prevalence. Our meta-analysis provides strong evidence for prepandemic cross-reactive humoral immunity to SARS-CoV-2 in Africa, closely tracking with malaria. Further studies of broader immunological profiles involved and of the public health implications are necessary.

Ioannidis and Ioannidis-Contopoulos (2023) asserted that "*cross-reactivity tracks especially with malaria prevalence*" implying for one thing, that it could also be present without "*malaria prevalence*" and for another that all results are considered to be "cross-reactivity" and therefore false positives. Why? The only reason is "the virus could not be there before 2020", according to the preconceived mainstream narrative. The same results in time frames after 2020 would be considered proof of the presence of COVID-19 infection. What is manifested is a spectacular example of how reality can be arbitrarily tailored to fit a preferred hypothesis instead of adjusting the theoretical hypothesis to fit the facts as should be done.

8. In Cambodia there were similar findings by Manning et al. (2022):

> Conclusions: We found in a widely used, highly specific, and validated ELISA that approximately 4% to 14% of prepandemic serum samples from malaria-infected persons in Cambodia were positive for non-neutralizing antibodies to SARS-CoV-2 spike and RBD [receptor binding domain] antigens by using various standardized optical density cutoff values.

9. In Singapore, Le Bert et al. (2020) found signs of "SARS-CoV-2 infection" among sera of unexposed donors (collected before July 2019):

> Notably, we detected SARS-CoV-2-specific IFNγ responses in 19 out of 37 unexposed donors.

GRAPHICAL ABSTRACT

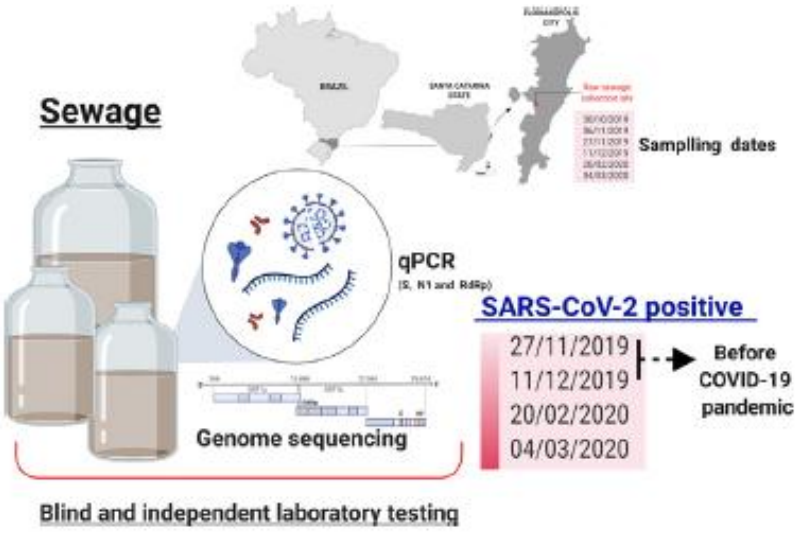

Figure 1. This is the graphical abstract from Fongaro, et al. (2021), titled "The presence of SARS-CoV-2 RNA in human sewage in Santa Catarina, Brazil, November 2019", in The Science of the Total Environment, 778, 146198 at URL https://doi.org/10.1016/j.scitotenv.2021.146198

10. In Brasil, Fongaro et al. (2021) examined sewage material for SARS-CoV-2 genetic sequences (Figure 1). They found some in November and December 2019:

> Human sewage from Florianopolis (Santa Catarina, Brazil) was analyzed for severe acute respiratory syndrome coronavirus-2 (SARS-CoV2) from October 2019 until March 2020. Twenty-five ml of sewage samples were clarified and viruses concentrated [...]. SARS-CoV-2 RNA was detected by RT-qPCR using oligonucleotides targeting N1, S and two RdRp regions. The results of all positive samples were further confirmed by a different RT-qPCR system in an independent laboratory. S and RdRp amplicons were sequenced to confirm identity with SARS-CoV-2. Genome sequencing was performed using two strategies [...] Conclusions: We have confirmed the presence of SARS-CoV-2 RNA that strongly implies the presence of SARS-CoV-2 circulation in the Americas as early as 27th November, 56 days ahead of the reports of COVID-19 cases in the continent and more than 90 days in the case of Brazil. Therefore, our findings point out that SARS-CoV-2 was circulating unnoticed in the community for some months before pandemic status was declared.

11. The virus was even captured "while swimming" in the Atlantic Ocean by La Rosa et al. (2024):

> Indeed, what is particularly novel in this study, is the discovery of viral RNA at considerable distances from the coastline.
>
> Forty-three 500-liter samples were collected between May 2022 and January 2023 from the Atlantic Ocean, the Mediterranean Sea, the Arctic region, the Persian Gulf and the Red Sea. Using molecular detection methods including real-time RT-qPCR and nested PCR followed by sequencing, we successfully detected SARS-CoV-2 RNA in 7 of the 43 marine water samples (16.3%), and specifically in samples taken from the Atlantic Ocean and the Mediterranean Sea. The estimated concentrations of SARS-CoV-2 genome copies in the positive samples ranged from 6 to 470 per 100 liters.

What quantity of viruses (dozens of pounds?) had to be continuously spilled into the ocean to obtain the observed concentrations, so far from the coast and sufficient to compensate for their presumed progressive degradation by sea water? Where were they coming from and since when? This doesn't seem to be in proportion with the mass of viruses allegedly present in the infected population, which has been estimated by Sender et al. (2021).

Discussion

The currently accepted theory — even if its erroneous premises are endorsed — has been completely falsified by the findings described above. The presence of "specific" viral sequences (attributed to the SARS-CoV-2 virus), specific IgG and IgM antibodies and specific cellular immunity in the pre-pandemic era, all over the world, make the version of the fictional escape from Wuhan implausible. Sometimes, a mysterious exception is invoked that proves the rule; however, that is not the case here because we are not talking about a single mysterious "white crow" but about entire flocks of white crows wherever anyone has looked for them. Not even an earlier escape dating back to 2018 or previous years can be invoked. It is true that laboratory experiments on the "gain of function" of coronaviruses began even before 2013-2014 (Oller 2021), and virus engineering can be traced back to the 1990s (e.g. Leparc-Goffart et al. 1997) but most of the modern research was developed after 2014.

For instance, Daszak (2014-2019) obtained funding in a series of grants to Eco-Health Alliance to collect and study the bat viruses that were viewed as good candidates for pathogens with pandemic potential (PPP). Some of them eventually came to be considered as the source of the main sequence in SARS-CoV-2. Later on, Yan et al. (2020) deemed RaTG13 to be a coronavirus that is outstandingly similar to SARS-CoV-2. It was allegedly found in Mojiang sick miners in 2013, and the same authors suspect it had been fabricated — and not discovered in bats — by the research group of Shi Zheng-Li (Zhou et al. 2020). Five years earlier, Ralph Baric and Shi Zheng-Li published a letter in *Nature*, see Menachery et al. (2015), where they described the synthesis of an infectious full-length SHC014 recombinant coronavirus, consisting of digitally assembled genetic fragments using "published sequences".

Alina Chan, a molecular biologist at the Broad Institute of MIT and Harvard, summarized her view in a review article published by the *New York Times* (2024). Among other arguments she noted that:

> In 2021, *The Intercept* published a leaked 2018 grant proposal for a research project named Defuse, which had been written as a collaboration between EcoHealth, the Wuhan institute and Ralph Baric at the University of North Carolina, who had been on the cutting edge of coronavirus research for years. The proposal described plans to create viruses strikingly similar to SARS-CoV-2.

In other words, the "gain of function" research on coronavirus began years earlier[4], but most authors accepted the idea that the new microorganism was released in November or December 2019. This supposition would become central to the mainstream narrative about the COVID-19 "pandemic". So, arguing about the date when the virus may have been created, leaves the deeper question of how SARS-CoV-2 could be the cause, would remain unanswered. In their seminal paper, Zhou et al. (2020) asserted:

> The epidemic, which started on 12 December 2019, had caused 2,794 laboratory-confirmed infections including 80 deaths by 26 January 2020.

Andersen (2020; cited by Oller & Santiago 2021) states:

> Estimates of the timing of the most recent common ancestor of SARS-CoV-2 made with current sequence data point to emergence of the virus in late November 2019 to early December 2019, compatible with the earliest retrospectively confirmed cases.

Fleming (2021) writes:

> In 2019, one of those pathogens was intentionally released upon the world in the Wuhan Wet Market.

Alina Chan, cited also by Wenstrup and Runz (2024), principal authors of the final report of the US Subcommittee on the COVID pandemic, stated:

> In December 2019, Chinese investigators assumed the outbreak had started at a centrally located market frequented by thousands of visitors daily. This bias in their search for early cases meant that cases unlinked to or located far away from the market would very likely have been missed. To make things worse, the Chinese authorities blocked the reporting of early cases not linked to the market and, claiming biosafety precautions, ordered the destruction of patient samples on January 3, 2020, making it nearly impossible to see the complete picture of the earliest Covid-19 cases. Information about dozens of early cases from November and December 2019 remains inaccessible.

In view of the evidence presented here, one might suppose that there was a natural spillover or a release of the supposedly "lethal virus" in the years 2011-2019, so as to explain its apparent spread during the time frame leading up to the "pandemic". Such a would-be explanation, however, does not mesh with the facts. In particular, there is a complete absence of the "pandemic" disease with the characteristics attributed to COVID-19, throughout the time period of interest. Besides, the findings in Russia even in the years 2011-2013 (Gumanova et al. 2022), make the hypothesis of the engineered virus rather unlikely, because most of the known gain of function research on the "new" pathogen named "SARS-CoV-2" had not yet taken place.

In any case, the findings described are incompatible with a causal role of SARS-CoV-2 of COVID-19 pandemic. In confirmation of the above, mortality from all causes remained in the same fluctuation band until February 2020 (see for instance the Italian data reported in Figure 2).

Many consequences follow:

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[4] Citing the "gain of function" research is only intended to follow the current narrative as it is. It does not imply agreement that the "gain of function" research has led to real achievements (i.e. new forms of life). In fact, "gain of function" research aiming to produce new entities actually depends on special assembler software of small RNA fragments, as detailed for instance in Islam et al. (2021). It is these computer-based procedures that have led to the registration of over 21.2 million different "isolates" of the same tiny supposedly infective COVID-19 viral entity known as SARS-VoV-2 (GISAID, July 2025).

1) The global pandemic must be explained in some way contrary to the mainstream narrative, and so must the related statistics. For example, the sudden increase of mortality from all causes in Bergamo (Italy) in March 2020, could not possibly be related to the spreading of a new virus infection because mortality spiked in too brief an interval of time. Suspicions of manipulation of statistics and other factors, including irrational clinical approaches and toxic effects due to antivirals — especially those imposed on the elderly — must be considered (Hockett & Engler 2024).

2) The role and significance of PCR, antibody and antigenic tests must be determined in this context. A realistic and correct approach would make it possible to solve the problematic observation that antibodies did not offer protection against SARS-CoV-2 infection nor against the COVID-19 disease. Besides, the mystery of "high viral loads" in asymptomatic individuals (e.g. Lavezzo et al. 2020) would also have a satisfactory explanation.

3) The cross-reactivity hypothesis has been invoked by some of the authors of the cited studies, but it is a double-edged sword: it can be argued that cross-reactivity of the tests performed and "immune reactions to variants of the corona virus" are indistinguishable. From another point of view: we started this work saying we would consider the specific tests fully reliable. The cross-reactivity hypothesis implies that it is not so. Not accepting that premise, one must conclude that the tests do not have the meaning attributed to them, so the cross-reactivity hypothesis cannot help the mainstream narrative which I have shown to be false.

4) An implication of my findings is that the "vaccine" products aimed at COVID-19 and more specifically the SARS-CoV-2 virus, could not have any meaning or usefulness, not even theoretically, because those products were directed against an agent that could not possibly be the cause of the disease they were supposed to prevent.

5) The origin and quality of the tests must be re-examined. It must be remembered that the reference *gold standard* testing, notably PCR, was prepared in a few days in a laboratory where there was no availability of the supposed causative agent. By the admission of the authors themselves, Corman et al. (2020), they had no way to validate any claims made about the virus itself. They wrote:

> We report here on the establishment and validation of a diagnostic workflow for 2019-nCoV screening and specific confirmation, designed in absence of available virus isolates or original patient specimens.

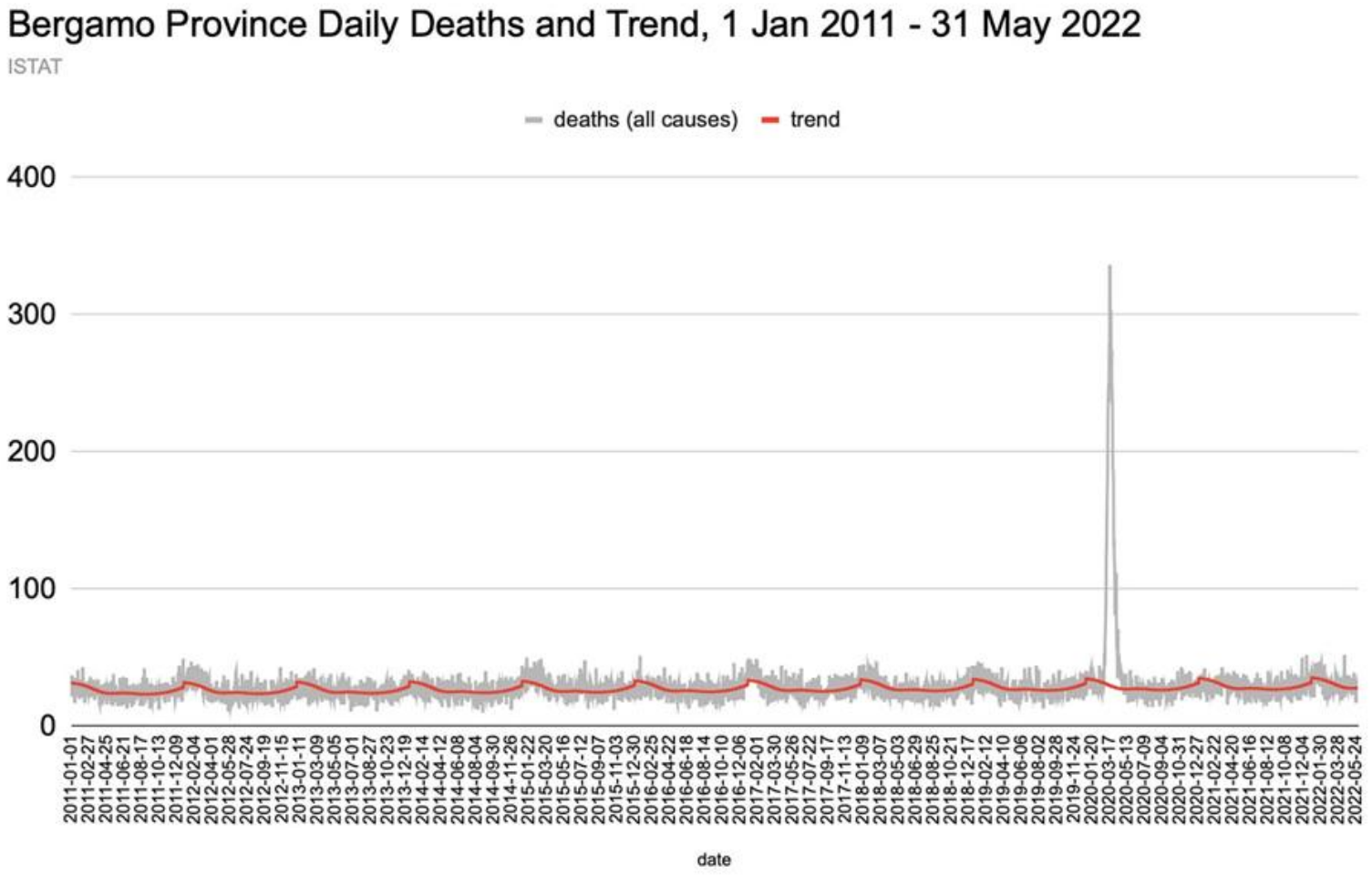

Figure 2. Bergamo province daily deaths, according to ISTAT. Bergamo has been the province with the highest mortality rate for "COVID-19" in Italy. From Hockett, J., & Engler, J. (2024, September 22). *Yes, We Believe the Bergamo (Italy) All-Cause Death Curve is Fraudulent*. https://www.woodhouse76.com/p/yes-we-believe-the-bergamo-italy.

6) Such a claim is self-contradictory. Validation in such a context cannot take place. The virus sequence was initially modelled on a computer with the help of special sequence-assembling software (Wu et al. 2020) and was hastily accepted worldwide without critical discussion. The scientific work that inaugurated the test — based upon a mere segment of the entire virus sequence (Corman et al. 2020) — was subjected to one of the fastest peer-reviews in the history of medicine. It required only a few hours ("Article submitted on 21 Jan 2020 / accepted on 22 Jan 2020 / published on 23 Jan 2020"). It is legitimate to doubt that the peer-review could have been sufficiently rigorous. The journal subsequently, refused to publish or comment on a devastating critique written by Borger et al. (2020). To demonstrate that the perplexities were — and remain — legitimate, it should be remembered that initially for this test there were descriptions of *positive* or *negative agreement* of the results (Abbott 2020; Abbott 2025; Anonymous 2020) instead of *sensitivity* and *specificity*. In fact, the specificity and sensitivity must be calculated in relation to the certain presence or absence of the virus itself (this shifting of terms corresponds to an admission that the authors had to resort to a fake validation, because the real gold standard was not at the disposal of the researchers). Subsequently, there was a silent transition to the use of the terms specificity and sensitivity, without any scientific or rational justification.

7) The just prior point, number 5, also stems from the need to revise fundamental concepts for the evaluation of the PCR testing, i.e., the calculation of false positives and negatives (Woloshin et al. 2020), according to the prevalence of the infection in the population (Wilson 2020). In this particular case, it is difficult to do any such calculation because the tests that are used to detect the virus, or the COVID-19 disease, are absurdly validated by unvalidated reference tests. They are like a dog that continues to chase its own tail, in a continuous circle (Anonymous 2020, Kämmerer et al. 2023). Besides, in the initial work of Zhou et al. (2020), the authors specifically noted that they used 40 cycles of PCR to amplify RNA, obtaining positive results with as many as 40 cycles of amplification which would later be deemed by the WHO (2020b) to be indistinguishable from background noise. It is noteworthy that Kari Mullis, the inventor of PCR testing, used a maximum of 20 cycles of amplification in his patent (Mullis et al. 1987).

Conclusion

The widespread presence of SARS-CoV-2, according to the tests prepared for its identification, in periods prior to 2020, and dating back to 2011, and geographically far from Wuhan, is incompatible with the theory officially accepted by health authorities and mainstream scientific communities worldwide. Therefore, that theory must be considered falsified. This falsification has direct consequences, for example, showing plainly the futility and irrationality of the so-called vaccine, which never, not even in theory, had any possibility of being effective. This falsification implies the need to challenge every aspect of the accepted "pandemic" narrative, that is to say, all the premises on which the contested claims have been based.

Given all the arguments that stand against the viral theory of the COVID-19 pandemic, and that exclude the possibility of SARS-CoV-2 being its cause, other reasons for its occurrence must be taken into account. Considering the abnormal breadth of the diagnostic criteria and the capriciousness of test results that made out virtually any and every health condition, or cause of death, to be COVID-19 disease, many causal factors other than the computer assembled SARS-CoV-2 must be considered. These factors can be identified in: a) every already known pathology

which was given the new name "COVID-19", including bacterial interstitial pneumonia which often went untreated because of the presumption of its being a viral infection, b) toxic factors, c) iatrogenic diseases due to the irrational clinical approaches of family doctors as suggested by the new COVID-19 protocols, or d) by the equally irrational approaches to treating patients admitted to the hospital and that included the use of dangerous "antiviral" drugs (e.g., remdesivir), as well as e) the initial terror induced by an ominous prognosis, and f) other stress inducing factors. The epidemiology of COVID-19 has been built in this biased manner and COVID-19 statistics have been inflated by the two mentioned components: pathologies normally present in the population which were given the new name, COVID-19, and actual pathologies that were poorly treated or that were induced by medical treatments directed towards a fictional cause.

Last but not least, I believe, the method of viral isolation should be completely overhauled, and the "gain of function" research with respect to the supposed infective agent of COVID-19, namely, SARS-CoV-2, should be re-examined for what I believe it to be: a digital construction, an artifact, that has little to do with the real world. Not science, but science fiction.

Conflicts of Interest

The author declares that he has no conflicts of interest. No outside funding was received for this work and no outside interest has influenced the conclusions reached.

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