

COVID-19: THE FALSE PANDEMICS, THE NEVER ISOLATED VIRUS AND THE FAKE SWAB TESTS.

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THE ISSUE OF DISTANCING AND MASKING

The most shocking and devastating element for our lives in 2020 was the so-called lockdown and the related measures (masks, distances, traffic ban, quarantine), all based on the principle that, to keep the expansion of the viral infection under control, it is necessary to maintain a sufficient distance between people, because suddenly closeness, community and sharing have become dangerous practices.

We have always been told that man is a social animal, but today we are told that our social being is dangerous, because we are all potential carriers of disease. We've had some hints of this thinking with previous pseudo-pandemics, but that was preliminary evidence that hadn't dented our natural tendency to social living, and to build economies based on exchange and sharing.

Not that there hasn't always been room for individuality and privacy as a distancing from others and from the world.¹ But these spaces have always been experienced, historically and before modern solitude, as moments of return to interiority, to spiritual knowledge, and to the formation of individuals so strong and independent that they are, precisely for this reason, more capable of contributing to the health of human communities. The right approach to separation from social life has always been to foster the process of individual growth and maturation as a prerequisite for a fuller and truer ability to participate and contribute to social life. In traditional communities, such as those of Native Americans, adolescents had to be able to spend a day and night alone amidst the dangers of the forest as a prerequisite for becoming full members of the community. The engine of that healthy individual separation was courage.

Today, however, the engine that leads individuals to withdraw away from others is fear: the other is a danger, and in this way individuality becomes a prerequisite for the destruction of the community, because in a similar world of individuals closed in on themselves and distant from the others, the community becomes simply impossible, a threat to be avoided at all costs. Unfortunately, the media manipulation unleashed with Covid-19 has managed to spread such levels of terror that the idea that the other is a danger to be avoided becomes normal and too widely accepted.

The World Health Organization, that today is increasingly in the hands of Bill Gates (as before, historically, it had been in the hands of the Rockefellers, of whom Gates is in many ways an emanation), prefers to use the term "physical distancing", perhaps so as not to make it too clear that the distancing measures

¹ Scoglio Stefano, *Transforming Privacy. A transpersonal philosophy of Rights*, Praeger, 1998.

adopted affect and tend to destroy sociality: the closure of schools and workplaces; the closure of public places such as restaurants and bars; the rigid limits imposed on mobility; home isolation and quarantine; the obligation to keep the distance between people of at least 1 or 2 meters (and this spatial uncertainty already shows that they do not even know what they are talking about); they are all forms of distancing aimed at ensuring that people do not assemble and therefore stop being people, to become a collection of individuals closed in their cocoon, both dangerous and unsafe, potential victims and potential executioners.

Behind this approach is the idea that viruses, and in particular the SARS-Cov2 virus believed to be responsible for the respiratory disease Covid-19, is transmitted by air or, as has been said more often, through the nebulized droplets in the air from those who cough or sneeze or, according to some, just speak. In reality we have heard all and its contrary about this virus, even that it settles on surfaces and can be contracted through the contact of the hands, which would then carry it inside our respiratory system when we touch our mouth or nose (from here the obligation of gloves, then withdrawn). We have heard of all these different contradictory theories because the truth is that all these theories on the transmission of the virus are only hypotheses that have never been proven:

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Is the coronavirus airborne? Experts can't agree

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Is the coronavirus airborne? Experts can't agree

The World Health Organization says the evidence is not compelling, but scientists warn that gathering sufficient data could take years and cost lives.

Dyani Lewis

As reported by this recent article, even experts do not agree that the coronavirus is airborne, and according to the WHO itself "the evidence is not convincing". The only studies in which the transmission of a coronavirus (not SARS-Cov2) by air has been preliminarily "proven" have been carried out only in hospitals and nursing homes, in places that are known to produce all types of infections due to hygienic conditions.² No study has ever proved that there is transmission of viruses in open environments, or in closed but well ventilated environments. Even assuming that there is this transmission by air, it has been stressed that, for the "contagion" to occur, it is necessary that the people between whom the alleged transmission occurs are in close contact for at least 45 minutes.³

² Morawska L, Junji C. Airborne transmission of SARS-Cov2, Environ Int 2020, Jun; 139: 105730.

³ Lewis D., Is the Coronavirus airborne? Experts can't agree. <https://www.nature.com/articles/d41586-020-00974-w>, p. 3

In short, all the radical distancing measures imposed by the various governments, aimed at preventing the transmission of the virus, such as maintaining the minimum distance, closing places even if adequately ventilated, and so on, are based on a hypothesis that has never been proved and that the WHO itself defines without convincing evidence to support it.

Incidentally, if there is no convincing evidence that the virus circulates in the air, the only apparently plausible reason in support of the masks also falls: it is established that the masks are unable to filter incoming viruses of the size of nanometers, therefore the only reason to keep them is to prevent the entry of droplets, which contain "our" viruses, into the air. But if we are not even sure that viruses travel in the air, on what basis is the obligation to wear masks imposed? Moreover, if the way the virus is supposed to reach someone is from the aerosol exhaled by someone else, this aerosol is exhaled only when we cough or speak animatedly, certainly not when we breathe through our nose, given that no droplets of saliva exit our body through the nose. Yet, they insist that we need to cover also our noses with the masks. It is inevitable to think that the goal of this imposition is different than the declared one: by blocking both mouth and nose, the mask blocks proper respiration, they make it more difficult for the air and thus the oxygen to get in, while keeping trapped in the CO₂ that we are supposed to exhale, generating that process of hypercapnia that can actually lead to respiratory problems, the very same problems that can be easily interpreted as Covid-19.

Moreover, there is a long standing literature on the antibacterial and anti-viral properties of lysozyme, the enzyme that is mainly present in our saliva, which again raises the question of how saliva droplets could be carriers of a virus that our saliva itself may inhibit, thanks to its lysozyme and other enzymes content.⁴

Even admitting, and not granting, that it was necessary to establish precautionary rules with respect to a never-isolated virus, it would have been enough to ask not to come into direct physical contact with anyone showing symptoms of interstitial pneumonia to keep the presumed contagion under control; without resorting to a useless and devastating universal lockdown. This is a first symptom of the fact that the reasons for the lockdown were completely unrelated to health reasons, and instead firmly founded on political-economic reasons (which I cannot discuss here).

The reality is even worse, however, because in fact SARS-Cov2 has never been truly isolated and never truly identified, and its pathogenicity has never been proven. From this primary fact the consequence derives that the swab tests to verify the positivity to the virus, based on finding the SARS-Cov2 in the patient's pharyngeal or broncho-alveolar fluid, cannot but be meaningless, given that the original and golden standard of the isolated virus is missing. These are strong but easily demonstrable statements.

⁴ Hardestam J et al, *Antiviral effect of human saliva against hantavirus*, J Med Virol, 2008 Dec; 80(12):2122-6.

ISOLATION OF THE VIRUS AND ITS PATHOGENICITY: THE VERIFICATION ACCORDING TO THE KOCH POSTULATES.

In microbiology there is a golden rule, Koch's postulates, developed by microbiologist Robert Koch:

- 1) the presumed agent responsible for the disease in question must be present in all cases of that disease;
- 2) it must be possible to isolate the microorganism from the diseased host and make it grow in a culture;
- 3) each time a culture of the microorganism is inoculated into a healthy host, the disease is reproduced;
- 4) the causing organism must be isolated again from the experimentally infected host.

These postulates are in fact elementary logical principles, because the only way to demonstrate the pathogenicity of a microbe is to isolate it from a sick patient, put it in culture, and check whether this culture is pathogenic (disease-producing). Since virology began, the microbiological world has divided between those who try to satisfy such postulates (with the appropriate adjustments), and those who try to make people believe that such postulates are no longer applicable, which amounts to say that in science there is no longer a need for logic. As we shall see, much of modern science has in fact decided to dispense with elementary logic.

One of the objections that this second group of microbiologists has raised is that viruses cannot reproduce in a "pure culture", as they do not reproduce on their own but need the host cell to reproduce. But it is clear that this is an objection easy to overcome, as it's enough to include in the concept of culture viruses presumable growing on host cells; and subsequent pathogenicity testing can be done with such a culture, as long as it can be proven that the virus is indeed the major component by far of that culture, and the host cells to be infected are healthy and non-pathogenic. But the first requirement, even before starting any culture, is the isolation of the virus, because without it there will be no virus to "grow".

Let's see how the alleged isolation of the SARS-Cov2 virus is described in the most important study, the first and the one cited by all subsequent ones as evidence of virus isolation.⁵ In the study by Zhu et al., as always, they start with taking samples of liquid from patients:

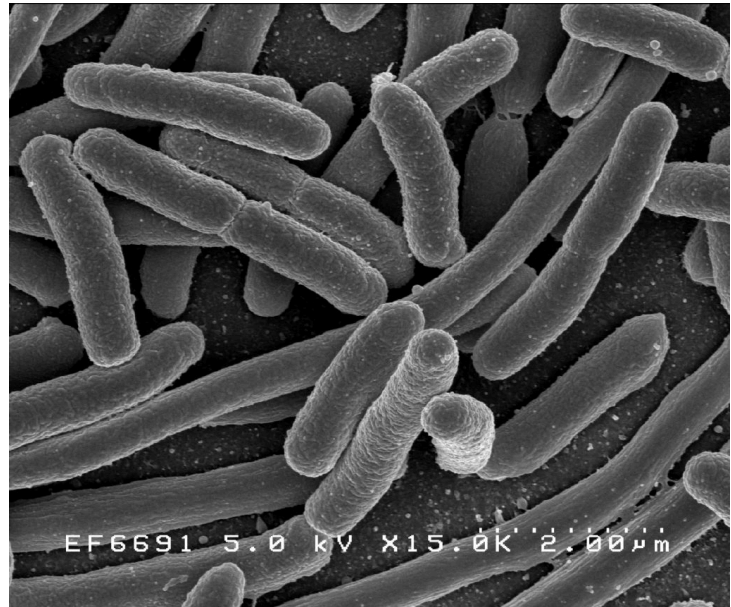
"Four lower respiratory tract samples, including bronchoalveolar-lavage fluid, were collected from patients with pneumonia of unknown cause who were identified in Wuhan on December 21, 2019..."

Now, one would expect that from this liquid, especially considering that in the infected liquid the share of SARS-Cov2 virus should be largely in the majority,

⁵ Zhu N et al, A Novel Coronavirus from Patients with Pneumonia in China, 2019, N Engl J Med. 2020 Feb 20; 382(8): 727-733.

through centrifugation and filtration procedures the researchers go to physically isolate the virus, as is done for bacteria. In the case of bacteria, the physio-chemical isolation is fairly standard, and it is possible to verify how the cultured bacteria form dense colonies that almost do not admit foreign organisms (see photo below); and this is because they are living organisms endowed, like all forms of life, with the fundamental impulse of self-preservation (self-preservation) and self-reproduction (self-reproduction).

Colony of E. coli



But viruses are not living organisms, they do not eat, they do not produce excrements, they do not reproduce. They are described as tiny organic molecules (comparable to infinitesimal fragments of our skin) made up of a strand of RNA (more rarely of DNA) and a lipoproteins the outer shell (capsid). The size of a virus is at the nanometer level, while bacteria are, like our cells, at the micrometer level, which obviously makes it much more complicated to isolate a single virus, given that nanoparticles are visible only under the electron microscope.

However, if it is true what they say about viruses, that in order to cause a disease they must reach a very high concentration or viral load, then in a patient's fluid there should be so many of those viruses that it should not be complicated to isolate the colonies of viruses produced, and thus also obtain electron microscope photos formed almost entirely by virus colonies, as in the photo above by E. Coli.

Instead, none of this happens, and researchers, God knows why, don't even try to physically isolate the virus, looking instead for what they call indirect evidence, such as determining and concentrating the specific nucleic acids (RNA) of the virus they are looking for; an attempt that, as we are about to see, is pretty much impossible. However, so far we have established that the virus has never been physically isolated in its entirety.

Indeed, Zhu et al proceeded to extract the nucleic acids present in the patient's liquid, assuming that it would composed mainly of the specific RNA from the SARS-Cov2 virus:

“Extraction of nucleic acids from clinical samples (including uninfected cultures that served as negative controls) was performed with a High Pure Viral Nucleic Acid Kit, as described by the manufacturer (Roche).”⁶

The study assumes that the kit only extracts viral RNA (after all, it calls itself High Pure Viral Nucleic Acid - high amount of pure viral nucleic acids). However, the manufacturer Roche, in the related documentation, states, referring to the kit, that:

“...total nucleic acids are isolated...Isolate both RNA and DNA”⁷

That’s the level of specificity! This means that the kit extracts a ton of undifferentiated nucleic acids. Let's do the math, however approximate. In the body there are 30-40 trillion (billions of billions) cells; and according to most virologists, about 400 trillion viruses, 10 times the number of human cells. Whereas there are, in each cell, about 360,000 units of messenger RNA (mRNA); and considering that mRNA constitutes only 1-5% of total RNA, in each mammal cell there are 7.2 to 36 million RNA molecules; let's average that up, and let's say 20 Million. As for DNA, DNA being about 2.5 times less than RNA, there are on average 8-10 million DNA units per cell. In total, therefore, in each cell we have on average 30 million human nucleic acids.

There are approximately 5 billion cells per 1 ml of human blood or fluid. Zhu et al. used 150 microL of broncho-alveolar fluid, which therefore contains about 750 million cells; each cell contains approximately 30 million nucleic acids of human origin; which means that in the supernatant used as sample there are about 22.5 billions nucleic acids of human origin, in addition to all those of presumed viral origin. There are thought to be around 400 trillion virus in the human body. Each virus has a strand of RNA (very rarely DNA). If we take the 5 billion cells per ml of human liquid, we can say that in the same amount there must be, given the above ratio of 1:10, about 50 billion viruses per ml, each with an RNA strand. This brings the total amount of nucleic acids in the 150 microL to: 7.5 billion of viral origin + 22.5 billion of human origin = a total of 30 billion nucleic acids!

It is in this ocean of nucleic acids that researchers want to identify the SARS-Cov2, but without knowing anything about the virus, as it is the first time they meet with it. And that virus is an infinitesimal particle inside a huge sea of infinitesimal particles: in short, it's really like looking for a needle in a haystack!

Sure, researchers might answer that in a sick person pneumonia the virus must have proliferated in such quantities as to cause disease, and therefore it must constitute a large share of the total nucleic acids present. But the existence of the disease in itself cannot be proof of its cause, the cause must be proven to being responsible for the development of the disease; and you can't establish the causal responsibility of a virus, if you do not even know its structure, that is without first isolating it.

⁶ *ibid.*, p. 28-29

⁷ https://lifescience.roche.com/en_it/products/high-pure-viral-nucleic-acid-kit.html

How do you go hunting for this infinitesimal fragment inside the ocean of billions of nucleic acid particles? Through RT-PCR:

“Extracted nucleic acid samples were tested for viruses and bacteria by polymerase chain reaction (PCR), using the RespiFinderSmart22kit (PathoFinder BV) and the LightCycler 480 real-time PCR system, in accordance with manufacturer instructions. Samples were analyzed for 22 pathogens (18 viruses and 4 bacteria)...In addition, unbiased, high-throughput sequencing, described previously, was used to discover microbial sequences not identifiable by the means described above.”

Before analysing each of these passages, let's see how the RT-PCR works. Polymerase Chain Reaction (PCR) is a technique invented by Kary Mullis, 1993 Nobel Prize in Chemistry, and is based on the laboratory reproduction of the DNA duplication and multiplication process in nature. Through high temperature, the DNA is split into its two filaments, each of which becomes a template on which the enzymes polymerases mounts free nucleotides to recompose the missing strand. In this way, from one unit of DNA you get two, from two four, and so on; thus being able to amplify small quantities of DNA into large quantities. Mullis has always declared that his technique was to serve only as a research tool, to amplify DNA that is known up to levels that can be more easily studied.⁸

Mullis has always warned against using his technique for diagnostic purposes, especially in relation to viruses unknown and emerging: Mullis was a very vocal critic of the HIV as the cause of AIDS theory, and always strongly affirmed that the HIV virus had never been isolated, contrary to what Gallo and Montagnier said, claiming that it could not have been isolated with its PCR.⁹

Let's think for a moment. When I try to amplify a virus, I start generally from RNA molecules, which have only one strand. The PCR, though, works with DNA, so it has to transform the RNA strand into a double stranded DNA: this is the RT component in the RT-PCR, and stands for Reverse Transcriptase - the operation of transcribing RNA into DNA. Now if I know the virus, I take its RNA strand and transcribe it by adding a small part of the genetic sequence, a primer, corresponding to the strand; but if the virus is an unknown, because it is a new and newly emerging virus (and in some ways all viruses are actually unknown), I have no defined strand of viral RNA available, but only a sea of billions of RNA and DNA nucleic acids, within which to go search for the genetic sequence of the virus I'm looking for. How can I do that?

I make a hypothesis, I try to guess: I choose some genetic sequences that I think can hopefully correspond to the RNA I am looking for. Here we already face a big problem. Having no proof that the interstitial pneumonias of Wuhan or Bergamo were caused by one specific virus, I could only speculate that the cause was a virus; and still further speculate that it was a virus from the coronavirus family. And so I went to look for primers, artificial gene sequences, which corresponded to the family of coronaviruses; but always and only by guessing,

⁸ Mullis K et al (Editors), The Polymerase Chain Reaction, Birkhauser, 1994.

⁹ <https://www.youtube.com/watch?v=lifgAvXU3ts>

because of coronaviruses there are countless sequences available, and this new virus would anyway be a new and different virus. Thus, I put these artificial and entirely hypothetical primers in contact with the supernatant of the pharyngeal or broncho-alveolar fluid of the patient, that is with tens of billions of RNA and DNA molecules; and if, as it is likely, my primers attach (anneal) to something in that broth, I concluded that whatever attached to my primers, then forming a DNA molecule by reverse transcriptase, it is the new and unknown SARS-Cov2. In other words, I threw a casual and hypothetical hook into the sea, and whatever unknown fish I pull up, be it even a small and not very threatening fish, I define and call that the killer fish, just like that, without any proof of its killing ability.

As if that weren't enough, the primers used are just an infinitesimal fragment of the alleged genome of the virus: they are in fact made up of only 18-24 bases (nucleotides) each; while the SARS-Cov2 virus is assumed to consist of 30,000 bases; so that the primers represent only 0.07% of the virus genome.

All that we explained above, has two complementary effects: it greatly reduces the probability that that priming sequence is specific to the virus in question, thus at the same time making it more probable that it will stick to anyone of the billions of gene sequences present in the supernatant, in a purely casual way. And the shortness and minimal size of the priming sequence greatly increases the chance of fishing up anything, including fragments of ordinary human genome.

Let's imagine that all English literature, including many poems and short stories unknown to the public, are collected in a huge database, and that I want to look for an unknown poem which, however, I believe was important at a certain historical period. I don't know anything about this poem, except that it is a love poem. I will therefore have to enter keywords in the computer that make me find the poem, but I can't use more than 18-24 letters. So I type "my love I miss you", a phrase of 18 characters (spaces included), and with this phrase I should find my poem among the about 28 billion poems contained in the database, half of which love poems. What are my chances of bringing out the specific poem I am looking for, and not one already known or still unknown but different from what interests me? I would say next to zero...and this is what happens with RT-PCR in relation to a virus that is new and therefore unknown.

This is not just my idea, but it is also recognised in the relevant scientific literature:

"The most commonly used PCR-based methodologies require the knowledge of the microorganism's genome sequences; however, this knowledge is not always available. A typical case is represented by the outbreaks of emerging pathogens, where the causative agent was never before identified..."¹⁰

¹⁰ Calistri A., Palù G., Unbiased Next-Generation Sequencing and New Pathogen Discovery: Undeniable Advantages and Still-Existing Drawbacks, *Clinical Infectious Diseases*® 2015;60(6):889-91.

This is why alternative methods are being developed today, such as the method called "unbiased" NGS (Next Generation Sequencing), which instead of going to fish in the sea of gene sequences for the specific sequence defined by the artificial and hypothetical primers, tries to sequence all the nucleic acids present, to then discard all the known ones via a comparison with the known sequences contained in the data banks, in order to leave, if possible, only the unknown ones. But this method also has strong limitations, limitations that are also applicable to RT-PCR:

“Because random/unbiased amplification amplifies the host nucleic acids along with the microbial ones, searching for the microbial nucleic acids is like looking for a needle in a haystack.”

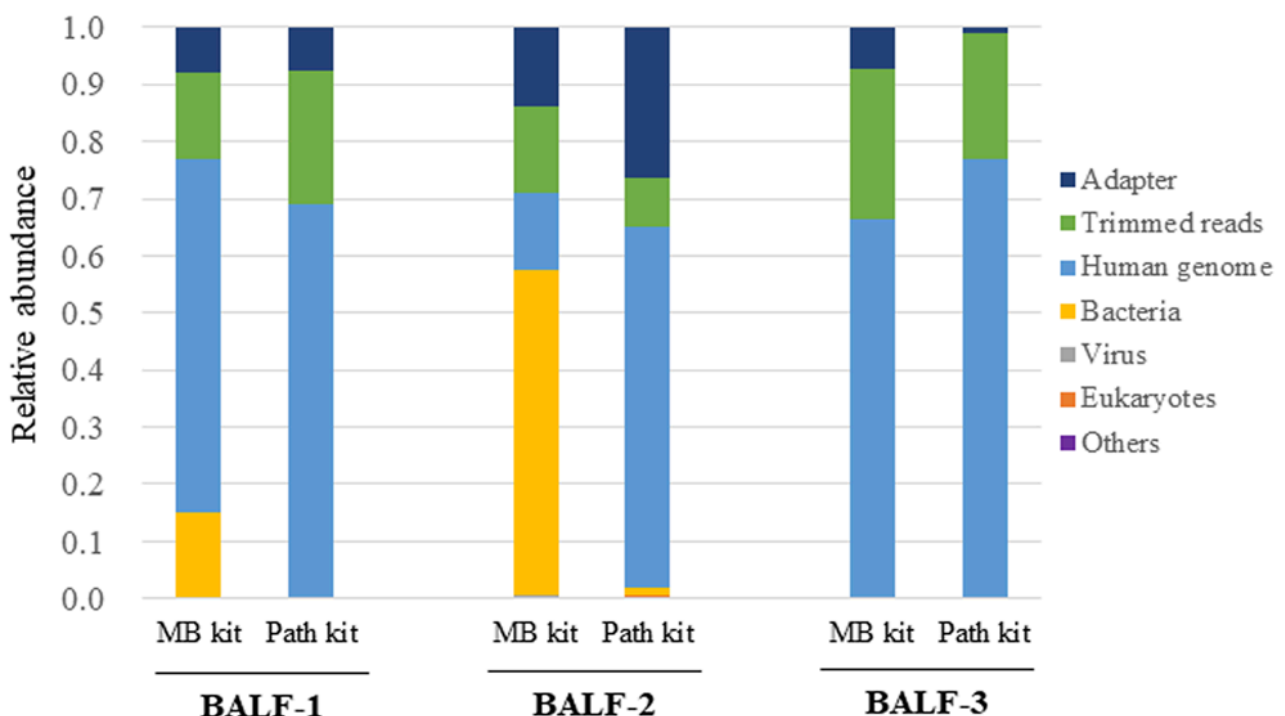
This is so much more true, considering that the human host’s nucleic acids constitute the great majority of the billions of nucleic acids contained in the supernatant:

“In the study by Brown and coworkers, only 0.4% of the total reads could not be assigned to the human genome”.¹¹

Brown et al.’s study focuses on brain tissue, and it is likely that in the brain, given its high level of protection, there are far fewer microbes than in the rest of the body. However, even if the number of microbes were higher, the human host’s nucleic acids would still represent the majority of the total nucleic acids; and if other compartments we involved, such as those in G.I. tract, we would have to face also the huge number of bacterial genomes present.

Let’s take a NGS study on the bronchoalveolar fluid (BALF) of patients with very serious respiratory conditions, thus patients whose BALF should have a very high percentage of viral or bacterial nucleic acids. This is the chart that reports the presence of nucleic acids in the BALF of 3 of such patients:

A



The count has been done with two different kits, with different results. However, one can see that in 5 out of the 6 bars, the light blue bars, representing the human genome, are greatly prevalent, except for the MB Kit result for the BALF-2. To the light blue bars we have to add the green bars of the Trimmed reads, as "...92.9-99.6% of trimmed NSG reads were derived from human DNA"¹² (the Adapter reads remain unassigned). So, when we add all the light blue and green bars, we obtain an average percentage of human genome nucleic acids equal to 86%. And this is in patients with very serious respiratory diseases! In ordinary people with no symptoms, this percentage is bound to increase to above 90-95%! In other words, the human genome always constitutes the great majority of nucleic acids, and this makes the haystack even bigger, and the search for the needle even more improbable; and this even in the specific field of respiratory diseases, to which COVID-19 belongs.

Zhu et al tried to reduce this huge sea to a smaller sea through the RespiFinder Smart22 kit, which checks for the presence of 22 microbes, 18 viruses (including the various influenza viruses and all other coronaviruses) and the 4 bacteria (*Bordetella pertussis*, *Chlamydia pneumoniae*, *Legionella pneumophila*, and *Mycoplasma pneumoniae*) potentially involved in respiratory disease of the patients from whom the bronchoalveolar fluid sample was taken. However, here too insurmountable difficulties arise. The main kit, the RespiFinder Smart22, was tested by an independent study, which concluded:

*"The RespiFinder-19 and RespiFinder-SMART-22A did not detect influenza viruses."*¹³

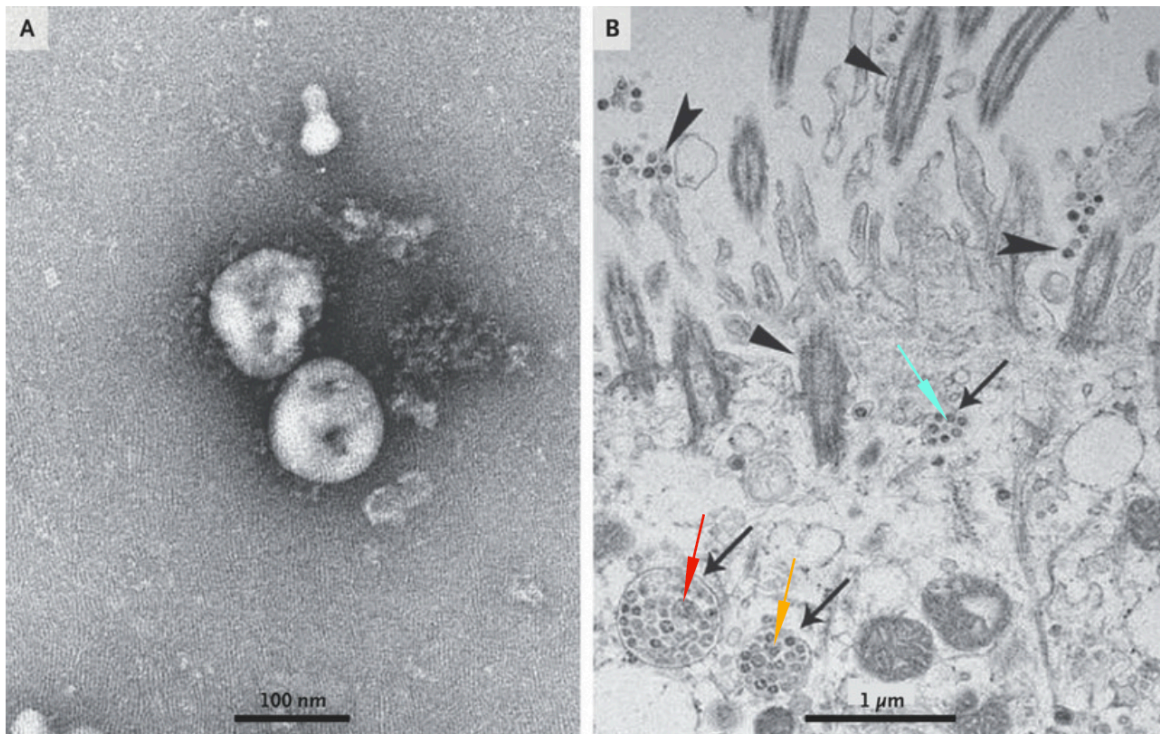
Hence, Zhu et al's attempt to eliminate other potential pathogens from the sea of nucleic acids, fails, not being able to eliminate even the fundamental influenza viruses. But this is secondary to the huge problem pointed out above, the fact that the patient's fluid is an ocean of billions of nucleic acids, most of which of human origin; with the almost certainty that the mini sequences, primers of 18-24 nucleotides, may have "caught" one of the host's endogenous nucleic acids, from an exosome or an extracellular vesicle (EVs).

That Zhu et al did not perform any isolation of the virus, neither physical nor by gene sequencing, it is further proven by what has come to be considered the essential proof of the identification of the virus, i.e. photographs of the same under the electron microscope (indispensable precisely because physical isolation does not seem possible). Below is the photo that Zhu et al present as the photo of SARS-Cov2:

"Electron micrographs of negatively stained 2019-nCoV particles were generally spherical, with some pleomorphism (fig. 3). The diameter ranged from about 60 to 140 nanometers (nm). The viral particles had quite distinctive "spikes" (spikes), about 9-12 nm, and gave aivirions the appearance of a solar corona. Free

¹²Takeuchi S et al, Metagenomic analysis using next-generation sequencing of pathogens in bronchoalveolar lavage fluid from pediatric patients with respiratory failure, Scientific Reports, Nature, 9/09/2019.

¹³ Dabisch-Ruthe M et al, Comparison of three multiplex PCR assays for the detection of respiratory viral infections: evaluation of xTAG respiratory virus panel fast assay, RespiFinder 19 assay and RespiFinder SMART 22 assay, BMC Infectious Diseases, volume 12, Article number: 163 (2012), p.2.



extra-cellular particles and inclusive bodies full of viral particles ... The observed morphology is compatible with the Coronaviridae family."

A first big problem, which emerges with these electron microscope photos, is that of size. On Wikipedia a few months ago it was written, in reference to SARS-Cov2: "The average diameter of the virus particles is about 125 nanometers (.125 μm) This is in line with what the authors of the article state, that: "The diameter varies from about 60 to 140 nanometers". But it is one thing to say that the virus is about 125 nM large, which is quite specific; another thing is to give a range as wide as that from 60 to 140 nm. For example, it can be said of human beings that they vary from about 1.50m to 2.10m, as there are several individuals of different heights. Now, saying that viruses as a whole range from 60 to 140 nM would make sense; but to say that the individual SARS-Cov2 virus varies so much it would be like saying that John varies his height from 1.60 to 2 meters depending on the circumstances!

One could reply that viruses are not human individuals, but it is also true that, according to virology, each virus has a fairly stable structure: for example, after 30 years, of the alleged HIV virus it is said that it has a size of about 120 nM. So, with SARS-Cov2 they are taking liberties of definition which further confirm that everything on this specific virus is even more random than usual. And that license of unlimited definition led to the fact that Wikipedia itself changed its text on the coronavirus, and now reports that "Each SARS-CoV-2 virion has a diameter of about 50-200 nanometers"¹⁴: that would be like saying that John varies his height from 1 to 4 meters according to circumstances!

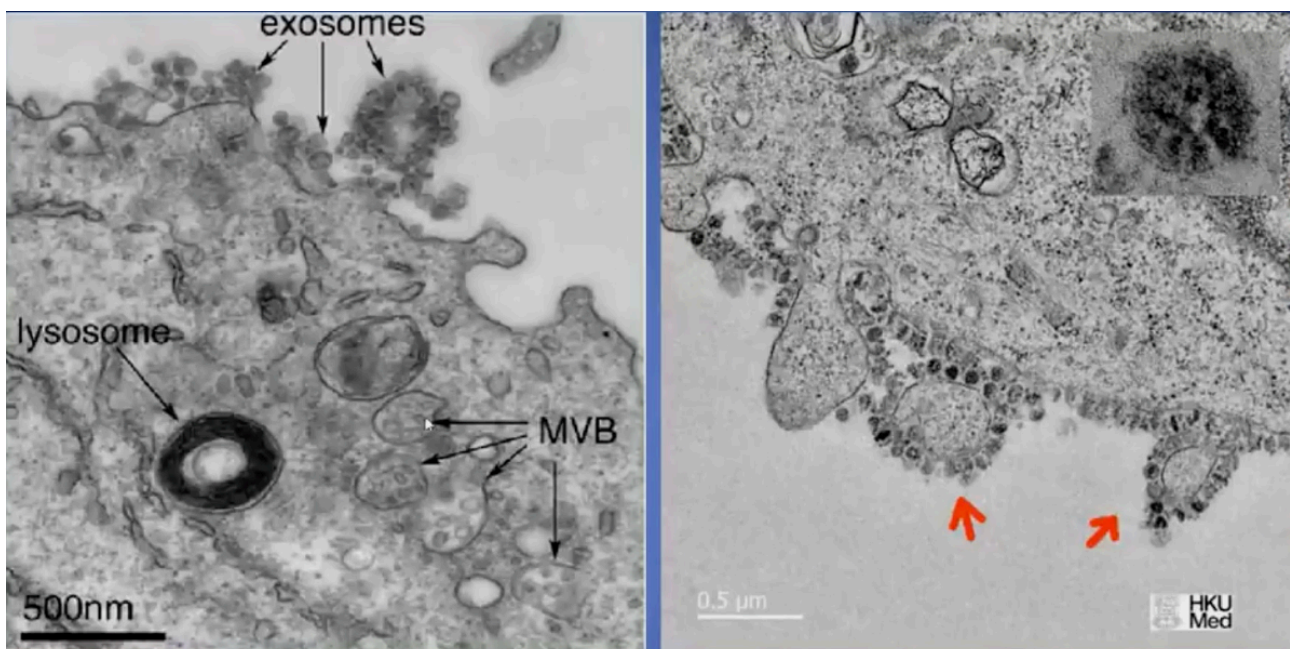
What is passed off as SARS-Cov2 are actually particles of all kinds, as can also be seen from the images provided by Zhu et al. Through a screen size meter

¹⁴ <https://it.wikipedia.org/wiki/SARS-CoV-2>

(FreeRuler), the particles that the authors assign to the SARS-Cov2 can be measured. The enlarged particles of the left side photograph measure about 100 nM each (on a 100 nM scale). But in the right side image, all the small particles indicated with arrows as SARS-Cov2, measured on a scale of 1 MicroM (1000 nM), have totally different sizes. The black arrows actually indicate vesicles containing viral particles. When we go to measure with the ruler some of these particles, we have that in the central vesicle the highest particle at the center (light blue arrow) measures approximately 51.87 nM, thus below the range proposed by Zhu et al ; the particle immediately to its right measures a little more, about 57.5 nm, but still below limit; while, almost at the center of the lowest vesicle, the largest particle (yellow arrow) measures approximately 73.7 nM, falling within the broad margins of Zhu et al; finally, in the lower left vesicle, the largest particle (red arrow) measures a good 155.6 nM, again well above the maximum limit defined by Zhu et al. (140 nM) . It is likely that the correction made lately by Wikipedia was aimed precisely at covering this problem.

There are other strong confirmations that the particles referred to as SARS-Cov2 may actually be those harmless and useful particles, called "extracellular vesicles" (EVs), which have extremely variable dimensions (from 20 nanoM to 10 microM), but which for the most part range from 20 nm to 200 nm, and which include, as a sub-category, that of "exosomes". These are particles produced by our cells which, as with the alleged viruses, contain nucleic acids, lipids and proteins, and are involved in various activities useful to our body, such as transport of immune molecules and stem cells, as well as the elimination of cell 's catabolic debris.¹⁵

Exosomes account for perhaps the largest share of EVs, and have been the object of numerous studies for over 50 years. Although few have heard of these beneficial particles, the scientific literature on them is huge, and only on PubMed, if one types "exosome", over 14,000 studies are provided! We cannot go into detail about EVs and exosomes here, but it is important to point out how they are indistinguishable from viruses, and several think that in reality what is defined as a dangerous virus is nothing but a beneficial exosome. This is immediately visible under the electron microscope¹⁶:



As can be seen, the largest of the exosomes is of the same size and structure of the corona-virus, and it is therefore plausible to believe that, in the large sea of particles contained in the supernatant of the Covid-19 patient's broncho-alveolar fluid, what is taken to be SARS-Cov2 is but an exosome. After all, it is the same researchers on exosomes who admit the impossibility of distinguishing viruses from extra-cellular vesicles and exosomes:

“In recent decades, the similarity between EVs and viral particles has become increasingly evident. Viruses and EVs share different aspects such as size, structural and biochemical composition, and the transport of bioactive molecules within cells...EVs and enveloped viruses also share similar biogenesis processes since both are generated in the endosomal network or bud from the plasma membrane...The remarkable resemblance between EVs and viruses has caused quite a few problems in the studies focused on the analysis of EVs released during viral infections. Nowadays, it is an almost impossible mission to separate EVs and viruses by means of canonical vesicle isolation methods...to date, a reliable method that can actually guarantee a complete separation does not exist.”¹⁷

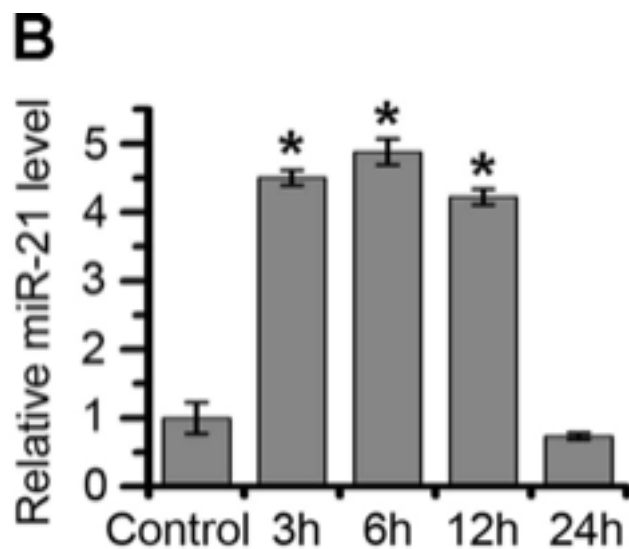
What else do you need? What is taken for virus may very well be an extra-cellular vesicle or an exosome. What is certain is that SARS-Cov2, if it exists at all, has never been isolated, which for a novel and unknown and only speculative virus is equal to a sentence of non existence.

In a recent discussion with an Italian biologist, she admitted that viruses and exosomes are in fact indistinguishable, but she claimed that there is a fundamental difference between the two, namely that while viruses replicate and multiply inside a cell, exosomes do not.¹⁸ However, if we look at the original Chinese studies that supposedly isolated the virus, there is no mention of any replication, nor any quantitation of the virus's multiplication. As we are about to see, the only proof offered for the presence of the virus was the damage that the culture containing the virus has done on the cells *in vitro*. The reality is the images of the virus entering a cell and exiting the cell in a multiplied numbers are not different from the what happens with exosomes. Exosomes are known to use the same methodology of entry into cells that is attributed to viruses, for instance through the “clathrin-mediated endocytosis”; and the multiplied numbers exiting the cell could very well be exosomes rather than viruses, not only because they are indistinguishable, as we have seen, but also because exosomes do multiply too inside a cell before exiting it. In fact, they can multiply even 5-fold (+500%) in just 3-6 hours, as shown by this graph¹⁹:

¹⁷ Giannessi F et al., The Role of Extracellular Vesicles as Allies of HIV, HCV and SARS Viruses, *Viruses* 2020, 12, 571; pp. 572-4.

¹⁸ <https://www.databaseitalia.it/dott-stefano-scoglio-dove-sbaglia-la-bolgan/?fbclid=IwAR2BsVawv7bKC9FqhDccZawmAd34LOZDL4NFu3PrFVRONk22ASLvyNPhZoo>

¹⁹ Tian T et al, Exosome Uptake through Clathrin-mediated Endocytosis and Macropinocytosis and Mediating miR-21 Delivery, *THE JOURNAL OF BIOLOGICAL CHEMISTRY* VOL. 289, NO. 32, pp. 22258–22267, August 8, 2014.



The intracellular proliferation of exosomes has been evaluated by measuring the exosomes's miRNA

With this, falls the only argument that could be used to claim that viruses can be distinguished from exosomes.

But lets' go back to Zhu et al's study. Its pathogenicity test, and thus its proof that in the culture used to "infect" there was a virus, was limited to testing for the cytotoxicity (toxicity on cultured cells) of the supernatant of the bronchoalveolar fluid, used by the authors as if it were the isolated SARS-Cov2. Apart from the limits of a purely *in vitro* test, the only result obtained was that while the group of cells where broncho-alveolar fluid was injected had a mild toxicity after 96 hours (4 days), the control group of cells that received the injection of an inert liquid, had the same level of toxicity, only after 6 days instead of 4. This was probably due to the fact that cells of the test group were human lung cancer cells, thus "sicker" and weaker than those used for the control group, which were E6 Vero cells (which are more susceptible to toxicity of normal human cells, but certainly less susceptible than human cancer cells).²⁰

But there is a further element that shows that Zhu et al. did not isolate any specific virus, the fact that indeed they sequenced not one but three different "viruses", each from each of three different patients:

"The novel coronavirus was identified from all three patients. Two nearly full-length coronavirus sequences were obtained from bronchoalveolar-lavage fluid, and one full-length sequence was obtained from a virus isolated from a patient (BetaCoV/Wuhan/IVDC-HB-01/2020|EPI_ISL_402119). Complete genome sequences of the three novel

²⁰ Zhu N et al, A Novel Coronavirus from Patients with Pneumonia in China, 2019, N Engl J Med. 2020 Feb 20; 382(8): 727-733, p.6.

coronaviruses were submitted to GISAID...and have a 86.9% nucleotide sequence identity to a previously published bat SARS-like CoV (bat-SL-CoVZC45, MG772933.1) genome. The three 2019-nCoV genomes clustered together within the sarbecovirus subgenus.”

That is, each of the three patients gave rise to a different viral sequence, and yet, given the 86,9% similarity of each of the three genomes with a previously published bat coronavirus, the three different sequences were clumped together and said to be all three the new SARS-Cov2. The novel coronavirus is indeed triune, like some other divinity we have heard of!

And this original sin of viral sequencing continues unimpeded as of today: at GISAID now there are almost 150.000 different sequences of the SARS-Cov2, from all over the world, and no amount of mutation of a single virus could achieve such amazing feat in just 10 months! If each of the sequences were indeed a true sequence of the SARS-Cov2, as they are officially recognised to be, that would mean that this virus mutates about 500 times a day! And if that were the case, how would you ever hope to be able to catch it?!?

But the reality is that there is nothing to catch, as admitted even by the two most important health bodies the two sides of the Atlantic Ocean, the European Commission and the US CDC. Let's start with the European Commission, which in its document of 16 April last wrote:

“Since no virus isolates with a quantified amount of the SARS-CoV-2 are currently available...”.²¹

Before analysing in detail this statement, which however seems to me self-evident, let's see what the CDC writes:

“Since no quantified virus isolates of the 2019-nCoV are currently available...”.²²

In short, both Europe and the US say the same thing: they call "isolated virus" a material in which the virus has not been quantified. But if it hasn't been quantified, how can it be an isolated virus? In any language, “isolated” means separate from any other substance, and therefore it should constitute 100% of the isolate, while here not only it does not constitute 100% of the “isolate”, you don't even know how much of it there is in the presumed “isolate”! When you make an extract, for example of phycocyanins, an 80% concentration is enough to say that it is “pure” phycocyanin: it is not really like that, but you accept it as a convention because you are satisfied with 80%. Phycocyanin, though, is known in great detail, has been fully identified and characterised, and that is why it can be quantified. In any case, all this proves that what is called an "isolated virus" is, as I have always maintained, a complex matrix of which the virus would constitute only a percentage. But what percentage: 1%, 5%, 50%? Dunno, no one knows, it

²¹ European Commission, Working Document of Commission Services, Current performance of COVID-19 test methods and devices and proposed performance criteria, April 16 2020, p.19.

²² Center for Disease Control and Prevention, Division of Viral Diseases, CDC 2019-Novel Coronavirus (2019-nCoV) Real-Time RT-PCR Diagnostic Panel, 13/07/2020, p.39

could be made up of 99% of something else, such as human genome fragments, but we continue to call it an “isolated virus”!

Maybe is because they also know very well that this is the case, that the EU Commission document poses the following parameters as a requisite for any valid swab test:

“A search for significant homologies with other SARS coronaviruses, the Bat SARS-like coronavirus genome and the human genome and human microflora should be performed to evaluate and predict potential false positive RT-PCR results.”

For the EU Commission this is presented as an auspice, something that in the future will need to be done, and that for now is only wishful thinking. Which confirms that in what is called a “virus isolate”, in which no virus is quantified, many other things are present as prevalent: other coronaviruses’s genomes, human genome particles (EVs and exosomes), and even the genome of our human microflora!

Now, when I say this, and I have been saying it from the beginning, I am insulted as a conspiracy theorist and irresponsible denier; I wonder of the same insults could be directed to the EU Commission and the US CDC!

The reality, one that is undeniable, is that lacking a virus isolate, all the SARS-Cov2 sequences are but hypothetical computer generated artefacts, nothing more. This implies that all the gene sequences that are presented to us as the “isolated virus” are but hypothetical constructions elaborated on the computer, mere artifices!

So, the question is: what is in the swab tests? What is in the vaccines being prepared and pushed by the pharmaceutical industry? And above all, how can we say that this alleged virus, which at the present state of knowledge is completely unknown, is responsible for whatever pathology?

And talking of pathology, let's analyse the study which is indicated as the one that would have satisfied Koch's postulates in relation to SARS-Cov2. Published in the prestigious Nature journal, and so sanctioned at the highest levels of official science, in the article the authors claim:

“Our results demonstrate the pathogenicity of SARS-CoV-2 in mice... completely satisfies Koch’s postulates, and confirms that SARS-CoV-2 is the pathogen responsible for COVID-19.”²³

In this study, the authors used as the SARS-Cov2 virus the nucleic acid extract previously prepared by the team of dr. Tan, who made Zhu et al's study, which we have discussed above. So, Bao et al continue in the cheating tradition of selling a nucleic acids extract as an isolated virus. But the study by Bao et al is interesting because it tested the pathogenicity of the alleged virus not in vitro and on cell cultures, but on mice, therefore at a higher level of realism.

²³ Bao L et al, (2020). The pathogenicity of SARS-CoV-2 in hACE2 transgenic mice, Nature, vol 583, pp. 830–833.

Let's clarify that, even if the virus had not been isolated, the study of Bao et al on pathogenicity is however interesting because if there really was a virus responsible for the pulmonary symptoms of the Wuhan patients, even if not isolated, it must have been present in significant amounts in the extract of the broncho-alveolar fluid (BALF) of gravely ill pneumonia patients.

By injecting that extract into mice, the mice should be getting sick with the same pneumonia-like symptoms of the Wuhan patients. Let's see if that is what happened. The authors used two groups of mice to inject the extract presumably full of the SARS-Cov2 virus:

a) a “wild”, natural type group, which has been injected intranasally with the BALF extract HB-01;

b) a group of hACE2 transgenic mice, specially modified at the level of the mothers' ovaries, so that they became generators of large quantities of the ACE2 enzyme, considered essential for the entry of coronaviruses into human cells; these mice were also injected intra-nasally with the BALF extract HB-01;

c) a third control group, consisting of hACE2 mice, to which a placebo was given.

The results of the alleged infection were the following:

*"We observed slight bristled fur and weight loss only in the HB-01-infected hACE2 mice—and not the HB-01-infected wild-type mice or mock-treated hACE2 mice—during the 14 days of observation; other clinical symptoms, such as an arched back and decreased response of external stimuli, were not found in any of the mice. Notably, the weight loss of HB-01-infected hACE2 mice was up to 8% at 5 days post-infection."*²⁴

Therefore:

a) natural mice, those not genetically modified, from receiving the allegedly very pathogenic virus had zero effects, nothing, naught...and since we humans are not, at least for now, genetically modified, this is what counts for us: **the alleged virus is unable to produce any pathogenic effect on natural beings.**

b) But even the devastating effects of the virus on genetically modified mice were pretty ridiculous: a slightly bristling hair and a minimal and temporary weight loss (-8%); in practice nothing special, no true pathological effect; in fact, if anything this “virus” should be proposed as a weight loss aid!

If this is proof of the pathogenicity of SARS-Cov2, I would say it actually demonstrates the absolute harmlessness of this alleged virus, which besides not having been isolated, if it really existed, once concentrated in the BALF extract, it should have generated seriously pathogenic effects in the mice, which evidently it did not do.

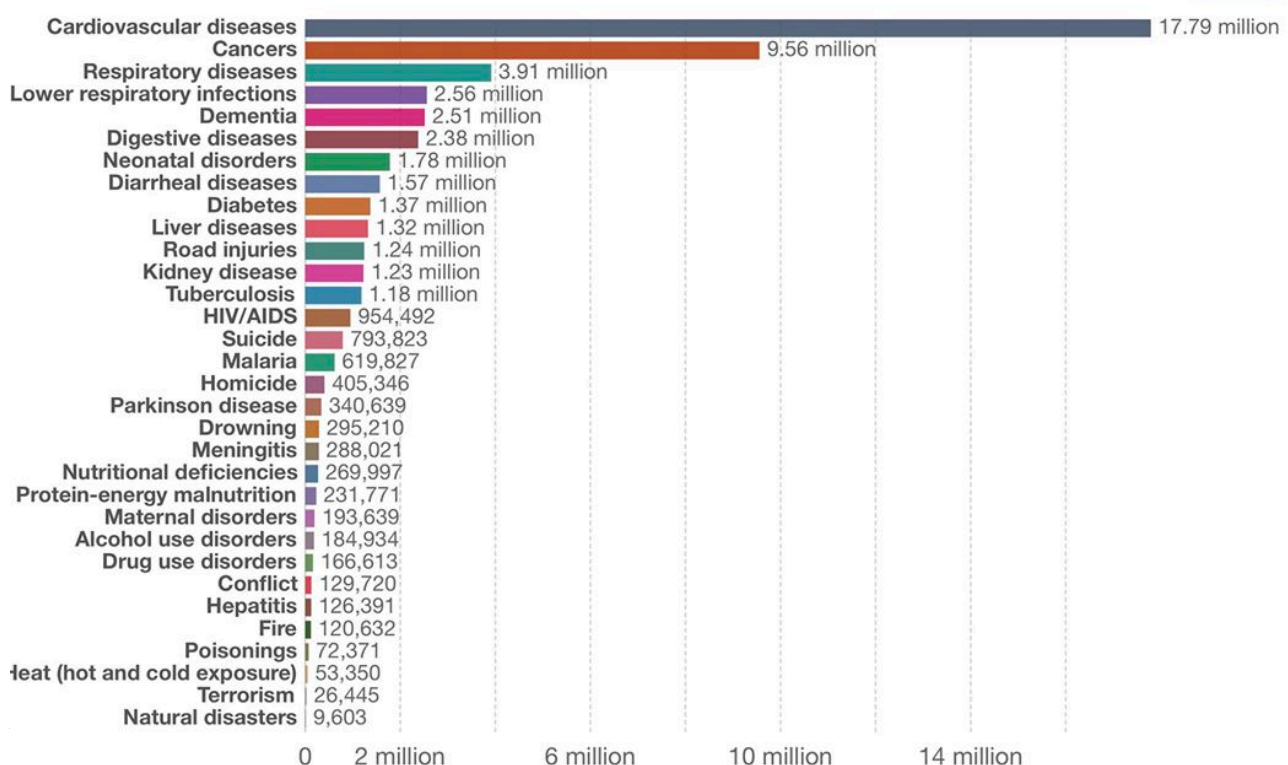
²⁴ Bao L et al, (2020). The pathogenicity of SARS-CoV-2 in hACE2 transgenic mice, Nature, vol 583, pp. 830–833.

Bao et al could argue that some pathogenic effects, such as tissue injuries, were found in the lungs of the genetically modified mice, even though not in the wild (natural) and placebo-treated mice. However, the hystological damage has been relatively modest, certainly not such as to be able to generate subsequent pathological effects. And anyway, it is also possible to think of alternative mechanisms for such minimal lung lesions: hACE2 mice are genetically modified to hyper-produce the ACE2 enzyme, a proteolytic enzyme found mainly in the lungs which, if in excess, can generates excessive vasodilation, which in turn, if prolonged can lead to hypoxia (reduction of blood oxygen levels); and prolonged hypoxia is in itself sufficient to cause some pulmonary tissue damage. The objection that the same hACE2 mice given placebo did not have the same lesions can be explained by the fact that the placebo was actually a saline phosphate buffer, which has a high content of sodium and chlorine, substances which have been shown to inhibit the action of ACE2 enzymes: "...a high-salt diet significantly decreased urinary angiotensinogen, ACE, and ACE2".²⁵

A FEW NOTES ON EPIDEMIOLOGY

We will focus, later on, on the Covid swab test, the real engine of this pseudo-pandemic. Why pseudo? The Covid-19 has presumably done something more than 1 million deaths in a year, most of them, as confirmed by both the US CDC and Italian ISS, involving people above 80 years of age and with mainly 3 concomitant very serious and lethal diseases, the only difference being their positivity to the swab test (and often they have been declared a Covid cases even without a positive swab test). How can it be a pandemic when every year, for Respiratory Diseases and Lower respiratory infections, the categories to which Covid-19 belongs, die every year nearly 7 million people in the world?

Number of deaths by cause, World, 2017



When I say that the numbers do not support the idea of a pandemic, I get the answer: what about all people who died in Bergamo? Sure, In Bergamo and a few other cities there was a significant increase in the number of deaths in March 2020, which was greatly compensated during the rest of the year. Interestingly, I discovered that most of the deaths in excess, though, were not even attributed to Covid but to other causes:

PROVINCIA	% comuni diffusi	% popolazione	variazione % gennaio+febbraio 2020/ media 2015-2019	variazione % marzo 2020/ media 2015-2019	Decessi totali 20 febbraio-31 marzo 2020	Decessi totali 20 febbraio 31 marzo media 2015-2019	Decessi covid 20 febbraio 31 marzo 2020	Decessi covid / decessi totali 20 febbraio-31 marzo 2020
Alessandria	95,7	98,2	-12,8	91,0	1.199	693	222	18,5
Ancona	76,6	84,3	-10,7	49,4	704	528	86	12,2
Aosta	91,9	91,2	-9,4	60,1	231	160	70	30,3
Asti	93,2	88,8	-13,9	38,5	382	299	38	9,9
Belluno	83,6	63,9	-11,1	9,9	205	201	14	6,8
Bergamo	97,5	98,4	-6,5	567,6	6.238	1.180	2.346	37,6
Biella	97,3	96,5	-9,5	84,0	471	279	74	15,7
Bologna	85,5	92,7	-8,4	20,0	1.525	1.289	183	12,0
Bolzano/Bozen	93,1	93,9	2,1	65,3	767	499	125	16,3
Brescia	98,0	98,9	-8,9	290,6	4.450	1.385	1.574	35,4
Como	94,6	95,6	-5,8	64,2	1.008	668	174	17,3
Cremona	99,1	99,8	-6,3	391,8	1.999	496	687	34,4

This is taken from a Table published by ISTAT, the Italian governmental statistical service²⁶, and shows that in the province of Bergamo, in the crucial period February 20-March 31, 2020, **the number of deaths increased from 1,180 in 2019 to 6,238 in 2020, a whopping 567% increase.** However, we also see that of the 6,238 deaths, **only 2,346, or 37.6%, have been attributed to Covid-19.** In reality, the distribution of causes death in Bergamo is as follows:

2346 : 1180 = 1.98 Increase of deaths assigned to Covid-19= 198% (+98%)

3892 : 1180 = 3.29 Increase of deaths for other causes = 329% (+229%)

Which begs the question of to what all these other excess deaths were due; and if in fact the excess of deaths is due to something other than an infectious disease, and so if all the measures taken to stop the spread of the virus (lockdown, masks, distancing) have been and are in fact completely misguided.

In any case, if one looks at all the tables of mortality of all of Italy for the same crucial period February 20-March 31, it is very clear that these excess of deaths, for whatever reason, are limited to a relatively small number of cities, spread over Northern Italy, which in itself is a proof that there no pandemic at work, or else all the cities surrounding Bergamo, and the other cities with excess mortality, should also have been hit. Instead, while Bergamo and Brescia were supposedly hit by the pandemic, the much larger Milan, which is very close to

²⁶ ISTAT, Impatto dell'epidemia Covid-19 sulla mortalità totale della popolazione residente. Primo trimestre 2020, May 4, 2020 (https://www.istat.it/it/files//2020/05/Rapporto_Istat_ISS.pdf).

Bergamo and has usually 2.5 times as many deaths as Bergamo, had less deaths than Bergamo (5,990 vs. 6.238).

While it is clear that whatever has happened was due to no pandemics, we may ask why this significant increase in deaths in such a limited number of cities, and in such a limited time. I have some possible answers:

- In Italy weather has been unusually warm during January and February, and the real winter cold has arrived only in March, which in itself contributed to all the deaths from flu-like symptoms, which since always have been potentially fatal for octogenarians that have been taking pharmaceutical drugs for years, which were then concentrated in the month of March,

- Given the central position of Bergamo in the morbid imagination of Italian people, it is important to underline how the types of respiratory Covid-like deaths were in fact happening well before the beginning of Covid. As reported in an interview to local doctors, they stated that their older patients had been dying of pneumonia, and pneumonia like symptoms, already since the previous October 2019. And, they added in the interview, that all those people were vaccinated, which the doctors took it to be strange, but in fact may very well be a major cause of those deaths.²⁷

- In fact, what stands out in the two most hot cities of Bergamo and Brescia is the fact, that due to a very strong campaign for mass vaccination against both flu and meningitis, in the two provinces, in the months before Covid-19, were administered, mainly to the older population, 185.000 flu vaccines and about 80.000 meningitis vaccines. This negative synergy of two toxic vaccines at the same time may very well explain, at least in a significant part, the excess of deaths.

- Another essential cause is the wrong therapy administered to patients hospitalised for Covid-19, a therapy essentially imposed to doctors by the Italian Government on a mandate from the WHO. We can safely state that the same therapeutic mistakes have been done all over the world. Before looking at the wrong therapy adopted, let me focus on another mistake which I hope has been done only in Italy: the Italian pharmaceutical regulatory agency, AIFA, in March 2020 authorised the intravenous use of interferon, even though in the very official leaflet of the drug it is specified that such a use could be lethal! AIFA withdrew the authorisation after one week, probably due to reports of people dying straight from the injection. No one has even been held accountable for such a horrific “mistake”.

- But the real crux is the wrong WHO Covid therapy, which consists in intubation and subsequent ventilation of Covid patients, at the first, even just temporary symptoms of respiratory distress. Intubation + ventilation are very extreme measures, usually done with people at risk of imminent death and generally already in a comatose state. This, because the insertion of a ventilator into the lungs is a very intrusive and violent practice, so much that in the case of Covid patients with respiratory distress but perfectly conscious, they had first to

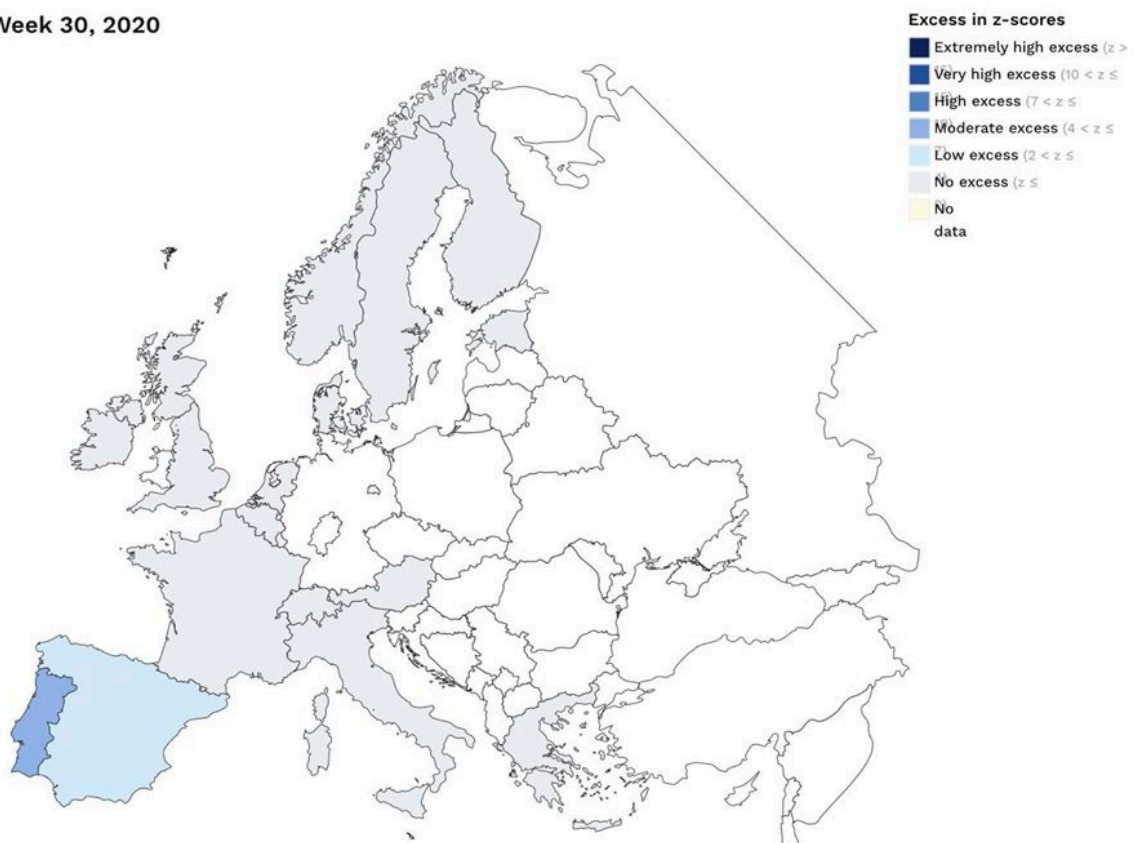
²⁷ <https://www.avvenire.it/attualita/pagine/a-bergamo-il-giallo-dei-focolai>

be put into a pharmacological coma. Besides this, the problem is that it was discovered, alas only after two-three months of its widespread use, that the practice was completely wrong and may have contributed directly to the death of the patients. In fact, initially the Covid-19 disease was described as interstitial pneumonia, an inflammation of the lungs accompanied by a cytokine-storm that would lead to respiratory distress. Based on this diagnosis, the WHO protocol prescribed the use of ventilators that would shoot high pressure oxygen into the lungs. And this was applied, with a further element, at least in Italy: the Ministry of Health advised all the doctors and hospitals not to do any autopsy, with the justification that that was necessary to stop the spreading of the disease among doctors. This was pretty ludicrous, as doctors may be the only ones who have all the necessary equipment to prevent contracting an infectious disease (leaving aside that there was no infectious disease at work at all).

When finally some doctors, both in the USA and Italy, decided to do autopsies, they discovered that the individuals who had died with Covid-19 for the most part were not affected by interstitial pneumonia, but from what is defined a pulmonary thromboembolism, that is their pulmonary vases were blocked by thrombotic blockages. It thus became evident that to shoot high pressure oxygen into blocked or partially blocked vases was equivalent to shoot high pressure water into a blocked water hose: the hose will burst!

When this very troubling fact became evident, many doctors switched to other therapies, onto drugs or natural products endowed with anti-aggregation and blood thinning properties. However, as far as I know, the WHO has not retracted its protocol.

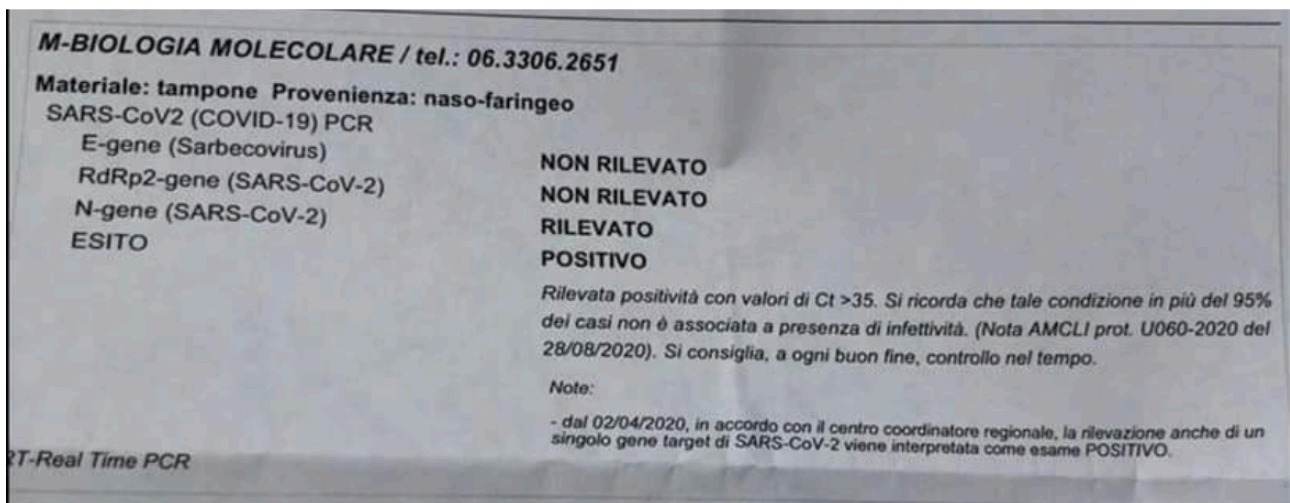
Week 30, 2020



In spite of all of this, in Europe, in the first seven months of 2020, there was no excess death rate, as shown by this graph from EuroMoMo, the European agency on mortality:

As we can see, as of July 30, 2020, with the slight exception of Portugal and Spain, the large European countries do not show any increase in mortality. This is why “asymptomatic disease” (an oxymoron) has now come to the fore as the new engine of the pseudo-pandemic. As there are no more excess deaths to show, while the politicians and their masters want to continue enforcing measures of strict social control and furthering the economic destruction of the small-medium independent companies and entrepreneurs, they now treat positive swab results, even if wholly asymptomatic, like the new sick, and most of all like the new infecting agents, capable of spreading disease, and most important terror, among the population.

Never mind that in all mental sane medical traditions the person who does not have symptoms is by definition healthy. It is not mental sanity or concern for health that leads politics, medicine and the media, these days. If it were, it would be evident to all, even when reasoning from within the basic viral-disease paradigm, that lack of symptoms, even in an “infected” individual, signifies that the viral load is so low as to make it impossible to infect anyone. This is actually openly declared in some swab tests certificate, such as this one:



Here it is written that: “Positivity has been found with values above 35 PCR cycles. In such a condition, 95% of the times, the condition is not associated to infectiousness.”

Alas, most Covid swab tests certificates do not report this evident truth, and the media do all they can to spread the opposite of this truth, always declaring the asymptomatic both sick and infectious.

Finally, even some mainstream scientists, such as Dr. Mike Yeadon, ex CEO of Pfizer, claim strongly that asymptomatic swab test positives cannot be infectious, and that it is time to stop mass swab testing!²⁸

THE CENTRAL QUESTION OF TESTING: COVID-19 SWAB AND SEROLOGICAL TESTS

The reality is that, for many reasons, including the excessive cycles of PCR, swab tests are wholly unreliable, and as we shall see they are equivalent to a lottery. This was immediately recognised by Chinese researchers themselves, who reported how the swab tests based on the genetic sequences created by the virologists were so unreliable as to generate over 80% of false positives.

[Potential false-positive rate among the 'asymptomatic infected individuals' in close contacts of COVID-19 patients].

[Article in Chinese; Abstract available in Chinese from the publisher]

[Zhuang GH¹](#), [Shen MW](#), [Zeng LX](#), [Mi BB](#), [Chen FY](#), [Liu WJ](#), [Pei LL](#), [Qi X](#), [Li C](#).

⊕ Author information

understand the robustness of the findings. **Results:** When the infection rate of the close contacts and the sensitivity and specificity of reported results were taken as the point estimates, the positive predictive value of the active screening was only 19.67%, in contrast, the false-positive rate of positive results was 80.33%. The multivariate-probabilistic sensitivity analysis

Alas, after publication, this study was later withdrawn, I wonder why...

The massive submission of the population to as many swab tests as possible is the only guarantee to find a large number of Covid-19 positives, and thus the justification needed to extend social alarm and the various forms of lockdown.

That swab tests, as well as serological tests, are completely unreliable, it should be clear by now: swab test use the same PCR methods used in alleged virus isolation studies, and should use as a parameter of identification of the presence of the virus the virus isolated and correctly sequenced. But since we know that the virus has never been isolated, all that the swab tests use are artificial, computer-generated genetic sequences, with the result that the action of the swab is completely random, a real lottery.

²⁸ <https://www.thelibertybeacon.com/there-is-no-asymptomatic-spread-mass-testing-can-stop-study/>

This is true also of serological tests. As opposed to what most people think and the media promote widely, namely that there actually is a SARS-Cov2 antibody, the antibodies present in every human beings are always and only 5, the immunoglobulins; and the serological tests search always and only for two of them: IgG and IgM. IgM are an early response of the immune system (4-6 days); IgG is a later response (9-12 days). But IgG and IgM are the answer of the immune system to any immune challenge, to any type of infection, intoxication, injury, etc., not just to viral infections. So how do you know whether those IgM and IgG are specific for SARS-Cov2, as they could be present for any other possible reasons? They put the samples of blood serum containing the immunoglobulins from the patient in contact with the presumed antigen of the SARS-Cov2 through an ELISA test. In practice, the ELISA test uses as a marker a protein that is supposedly related to the SARS-Cov2, to see if the antibodies of the tested serum respond to it and get activated. However, as no SARS-Cov2 virus has ever been isolated, whatever protein of the virus is used cannot but a laboratory artefact. This means that even the serological tests are meaningless, like the swab tests, they're just a lottery tied to the bad luck of having in your own body nucleic acids belonging to one's own Extra-vesicular cells or exosomes that match with the casual protein-antigen used in the ELISA test.

If we analyse one of the most common swab tests, that of the Institut Pasteur, which has been adopted by the WHO, we even find that among the primers it uses for the PCR there is one that corresponds to the genetic sequence of the chromosome 8 of human DNA. The human DNA chromosome 8 was sequenced in a specific article with the following genetic formula: CTCCCTTTGTTGTGTTGT. And below is the WHO table relative to the Institut Pasteurs' swab test:

Primers and probes

Name	Sequences (5'-3')	Length (bases)	PCR product size	Ref.
RdRp gene / nCoV_IP2				
nCoV_IP2-12669Fw	ATGAGCTTAGTCCTGTTG	17	108 bp	1
nCoV_IP2-12759Rv	CTCCCTTTGTTGTGTTGT	18		
nCoV_IP2-12696bProbe(+)	AGATGTCTTGTGCTGCCGGTA [5']Hex [3']BHQ-1	21		
RdRp gene / nCoV_IP4				
nCoV_IP4-14059Fw	GGTAACTGGTATGATTTTCG	19	107 bp	1
nCoV_IP4-14146Rv	CTGGTCAAGGTTAATATAGG	20		
nCoV_IP4-14084Probe(+)	TCATACAAACCACGCCAGG [5']Fam [3']BHQ-1	19		
E gene / E_Sarbeco				
E_Sarbeco_F1	ACAGGTACGTTAATAGTTAATAGCGT	18	125 bp	2
E_Sarbeco_R2	ATATTGCAGCAGTACGCACACA	20		
E_Sarbeco_P1	ACACTAGCCATCCTTACTGCGCTTCG [5']Fam [3']BHQ-1	20		

1/ National Reference Center for Respiratory Viruses, Institut Pasteur, Paris.

2/ Corman et al. Eurosurveillance¹

In theory, with a swab test like this, everyone who does it should test positive. However, why this is not the case, and the reasons why it is not so may be:

a) The WHO-sponsored swab test is just one of many different swab tests, though it's one of the most widespread;

b) the swab tests still uses pairs of different primers simultaneously (gene RdRp/Ip2, geneRdRp /Ip4 and gene E), and, since as we shall see the WHO advises now that the test can result as positive if just one gene is found, then it becomes a lottery which of the primers it will find for first a gene sequence to anneal. Which means that the chance of being positive with the specific primer containing the chromosome 8 sequence is only about 1/3, or 33%.

In any case, the fact that with one of most widespread swab tests contains the sequence of a human gene shows how the swab tests really search for any gene sequence, no matter if human or not; and that is why these types of swab tests guarantee that a certain number of tested people will always be positive, so that the pseudo-pandemic and its real terrors can be extended at will by the WHO and its acolytes.

In addition to this shameless pursuit of positivity at all costs, another factor which supports a high probability of testing positive with the swab test even in absence of specific Covid disease, is the link between being stressed out, sick and/or older, and an increased production of exosomes. These are in fact generated by our cells also as a result of oxidative stress on the cell. Thus, it has been emphasized

“...a role for exosomes in the selective secretion of harmful/damaged proteins and RNAs and thus in the maintenance of cellular fitness...the emerging function of exosomes as a means of alleviating intracellular stress conditions, and how secretion of harmful or unwanted material in exosomes, in coordination with the autophagy-lysosomal pathway, is essential to preserve intracellular protein and RNA homeostasis.”²⁹

Therefore, the higher the levels of stress and disease of any type, the higher the age, the greater the oxidative stress; the greater the production of extracellular vesicles and exosomes, and therefore of nucleic acids; and finally the greater the probability that the genetic sequences of the swab test will find a match, a correspondence, even if completely random, resulting in a positive swab test.

The normative-regulatory status of swab tests

That the swab tests are completely unreliable has even been sanctioned by the European Commission through its Working Document of 16 April 2020. The document date is relevant: April 16, 2020, the peak of the pandemic was already passed, so the EU evaluated the question of the reliability of the swab tests only after the supposed big wave of Covid-19. This means that all the original

²⁹ Baixauli F et al, *Exosomes and autophagy: coordinated mechanisms for the maintenance of cellular fitness*, *Frontiers in Immunology*, August 2014, Volume 5, Article 403.

lockdown period has been handled on the basis of swab tests that no one had even wondered if they were valid or not! That this is a problem it's also implicitly acknowledged by the EU:

“Timely and accurate COVID-19 testing is an essential part of the management of the COVID-19 crisis...after being placed on the market the performance of devices may be validated, i.e. confirmed by additional testing that the manufacturer’s specifications are indeed satisfied, e.g. in reference laboratories, academic institutions or national regulatory agencies. Such validation is not legally obligatory but highly recommended for public health decision making, especially in the context of the current COVID-19 crisis.”³⁰

The EU, which splits the hair in order to deliberate on the curvature of bananas or on the size of clams, here authorises the placing on the market of the most important tests of human history without any preliminary verification, and only hoping for optional ex post validation checks? It is so evident that the validation of tests is essential for making correct political decisions, that the “Strong recommendation” to perform validation tests looks like the fig leaf to cover the shameful waiver of proper checking before authorising tests on which the fate of nations depended! The EU document reveals that:

“In total, 78 devices based on RT-PCR...101 for the detection of antibodies and 13 for the detection of antigens were assessed.”

So, while everyone thinks there is 1 test for the Covid-19 swab, in Europe there are 78 different ones (as of April 16, today many more), PCR-based tests placed on the market by manufacturers or importers (also from China) without any prior validation! In fact, continues the EU document, of the 78 appliances on the market

“...only the ones from the Institut Pasteur, the Hong Kong Faculty of Medicine and the Charité were in-house validated...”

So, only 3 out of 78 swab tests have been validated, and even those only internally, which is equal to the innkeeper declaring that his wine is good. As the EU document explains:

“The most crucial information concerning RT-PCR based methods developed for the detection of SARS-CoV-2 are the sequences of the oligonucleotides (primers and probe) used for the amplification of the cDNA...It is important to note that, except for a few cases, no information on the actual sequences of the primers and probes in the device could be found.” (p. 13)

This is a very serious admission, because it confirms that we have entrusted the decisions about a devastating lockdown to 78 different types of tampons, none of which previously evaluated, validated, nor authorised, and that for the most part (75 out of 78) do not even declare what they contain!

³⁰ European Commission, Working Document, *Current performance of COVID-19 test methods and devices and proposed performance criteria*, April 16 2020.

And they can do that because of a huge hole in European legislation. Swab tests fall under the new legislation REGULATION (EU) 2017/746 OF THE EUROPEAN PARLIAMENT AND OF COUNCIL of 5 April 2017 relating to in vitro diagnostic medical devices, which repeals Directive 98/79/EC of 1998. In the previous legislation repealed, in general it was sufficient to affix on the device the CE mark, which is a mark relating above all to the device's safety; and only for some in vitro diagnostic devices listed in Annex II, and dealing with viruses already known (HIV 1 and 2, HTLV I and II and hepatitis B, C and D), it was necessary the technical and effectiveness evaluation by a Validation body recognised by the EU. So, while testing devices dealing with viruses, up to 2017, were required to have a specific authorisation based on a technical evaluation, in 2017 a new law was passed, which paradoxically introduced even stricter requirements (similar to the ones indicated in the EU Commission document) and strict preliminary validation procedures, but with a very important caveat: **the 2017/46 Regulation of 5 April 2017 will enter into force, for in vitro diagnostic devices, only on May 26, 2022!** Which means that Covid-19 swabs tests enjoyed an interregnum of suspended legality, not being included, relating to a new virus, in the 1998 Regulation; and not being yet subjected to the 2017 Regulation that would have outlawed them all, but which will not come into force until mid-2022!

The question that needs to be asked, and that cannot fail to have legal relevance is: these tests completely missing any prerequisite evaluation and validation, and being in circulation only thanks to a regulatory vacuum, thanks to a normative anomaly, and considering that in 2022 they would be completely illegal; it is admissible that such almost-illegal tests have the power to constrain and destroy the life and economies of entire nations and in fact of the whole of Europe?

The question of virus mutation

One of the fundamental problems with the mainstream narrative is the presumed constant mutation of the virus. Writes the Italian Istituto Superiore di Sanità :

“The virus can in fact mutate and new nucleotide sequences deposited in databases can reveal whether **these mutations may in turn make a particular test less effective or even ineffective** ... It is important to point out that for the diagnostics of this emerging virus, with a state of the art in evolution, the actual observed device performance can differ from those determined by the initial study of performance conducted by the manufacturer for the purpose of CE marking, in one previous state of the art.”³¹

This is what I, and many other critics, have been saying all along. To understand this, it would suffice to say that most of the swab tests in circulation were structured on the fake virus sequenced by the Chinese in Wuhan. But in Italy, both the Spallanzani and the San Raffaele hospitals developed different gene sequences of the virus, claiming that they had isolated (with the same fallacious

³¹ Gruppo di Lavoro ISS Test Diagnostici COVID-19, Dispositivi diagnostici in vitro per COVID-19. Parte 2: evoluzione del mercato e informazioni per gli stakeholder , Rapporto ISS COVID-19 n. 46/2020, 23 Maggio 2020, p. 8.

methodologies I have described in relation to Zhu et al.) an Italian virus different from the one isolated in China.³² Plus, in a study organised by several Italian medical centres (Sacco, San Raffaele, etc.), when they analysed 59 biological samples from Covid-19 patients from different hospitals in Center and Northern Italy, they found a notable mutation, to the point of finding:

“A mean of 6 nucleotide substitutions per viral genome was observed, without significant differences between synonymous and non-synonymous mutations, indicating genetic drift as a major source for virus evolution.”³³

Besides the miracle of the Triune Virus by the Chinese, we now find that it mutates country by country, possibly province by province (given that the Spallanzani in Rome and the San Raffaele of Milan sequenced different viruses), and maybe city by city. And in spite of the fact that even a national health authority such as the Italian ISS stresses how “... these mutations may in turn make a particular test less effective or even ineffective”, no political authority has ever even to curious to find out about the reliability of the Covid tests that are circulating in their country!

The constant mutation of SARS-Cov2, such as to make it de facto unrecognisable, has also been confirmed internationally: an American article, which also includes Robert Gallo among the authors, found dozens of mutations, increasing over time in parallel with the alleged spread of the virus from Asia to Europe to the USA³⁴; while an Asian author analysed 85 different SARS-Cov2 genomic sequences available from GISAID, and found 53 different SARS-Cov2 strains from various areas of China, Asia, Europe and the United States.³⁵

In conclusion, if the virus constantly changes, then the swab test is useless, because it looks for a virus that is always previous and always different than the one currently in circulation. This alone would be enough to understand that the Covid-19 buffer the test is completely, 100%, fallacious!

The issue of the Covid-19 test genes

The “Drosten PCR Test” and the test of the Institut Pasteur, the two tests considered the most reliable (although none of the two has been externally validated), both test for the E gene, although the Drosten test uses it as a preliminary test, while the Institut Pasteur uses it as a definitive test. According to

³² Capobianchi M.R. et al., Molecular characterization of SARS-CoV-2 from the first case of COVID-19 in Italy, Clin Microbiol Infect, 2020 Jul;26(7):954-956.

³³ Lai A. et al., Molecular Tracing of SARS-CoV-2 in Italy in the First Three Months of the Epidemic, Viruses 2020, 12, 798; doi:10.3390/v12080798.

³⁴ Pachetti M. et al., Emerging SARS-CoV-2 mutation hot spots include a novel RNA-dependent RNA polymerase variant, J Transl Med (2020) 18:179 <https://doi.org/10.1186/s12967-020-02344-6>.

³⁵ Phan Tung, Genetic diversity and evolution of SARS-CoV-2, Infection, Genetics and Evolution, 81 (2020), 104260.

the authors of the Drosten test, the E-gene test is capable of detecting all Asian viruses, thus being very non-specific at the same time (all viral strains) and limited to a geographical area (Asia). Again, the test of Institut Pasteur, one of the most adopted in Europe, uses the E-Gene test as a final test, although it is now known that the SARS-Cov2 virus (or virus) believed to be circulating in Europe would be different from the Asian ones. In reality, as we are about to see, the E gene is the less specific of the 3-4 genes searched for with the Covid-19 swab tests, been common to all coronaviruses, and thus generating a lot of cross-reactivity.

A main problem with the testing of he SARS-Cov2 is the fact that last April, while before the tests in order to produce a positive results, were required to find all 3 genes of the presumed virus, the WHO changed the algorithm,

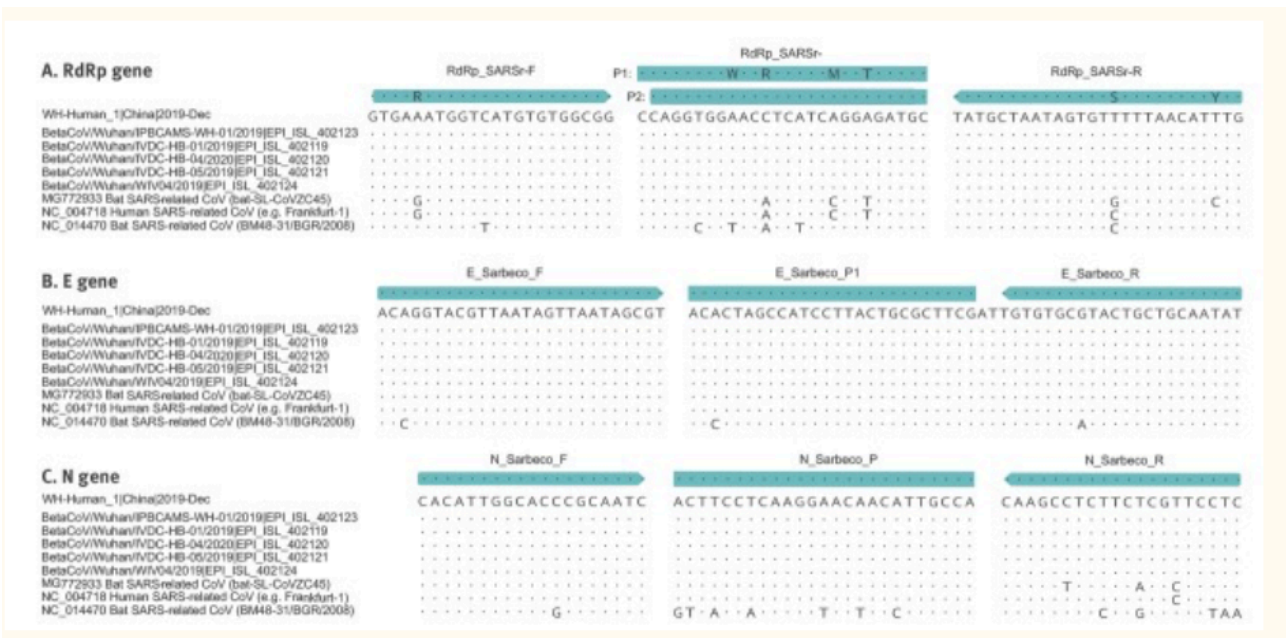
“...recommending that from now on a test can be considered positive even if only the dosage of the E gene (which probably will detect all Asian viruses!) gives a positive result ”.³⁶

This, in many places, was received as authorising that **finding even just a single gene, not necessarily the E gene, was enough to declare a person positive to the SARS-Cov2**. So, in the Covid test certificate reported also (p. 21) above it is stated that:

"From 02/04/2020, in agreement with the regional coordinating center, the detection of even a single SARS-Cov2 target gene is interpreted as a POSITIVE test".

If the original approach had been maintained, the number of positive asymptomatics we have today would have been much smaller. From April on, also thanks to the WHO, it is enough to detect only one of the 3 genes to be declared positive! This was clearly motivated by the need to keep up the numbers of positives to sustain the fake pandemic, as there is no logical or scientifically sound explanation to justify such move.

The need to detect all three genes becomes evident when one looks at the low specificity of each gene. Below, we see the gene sequences of the German team of Drosten, the one who made the test swab admittedly only based on computer sequences, without having any physical virus available. However, it is one of the most popular swab tests in Europe³⁷:



As one can see from the above table, Drosten's swab test uses all the 3 genes: E, N and RdRP. But if we compare the gene sequence of SARS-Cov 2 with that of the original SARS-Cov (at the penultimate place in the list), we see that:

- the **SARS-Cov2 gene E is 100% identical to that of SARS-Cov1** (in the penultimate line there are no variations of letters) and probably to that of all SARS coronaviruses, as the E gene "...also detects SARS-related coronaviruses."³⁸
- The **N gene has only one variation**, a C instead of a T, in the 15th place in the sequence of the Reverse Primer; while the Forward Primer is 100% identical! This is a variation of just 1/64th, or just 1.5%. The possibilities of confusion and cross-reactivity (detecting a SARS virus other than SARSCov2) it is clearly very high, especially considering the 100% sameness of the Forward Primer.
- The **RdRP gene has only two variations**, one in the Forward and one in the Reverse Primer. Again not a big difference, only one variation for each primer is a very, very small difference.

Given the above shown lack of specificity of the 3 genes tested for, at least the requirement to generate a positive result only with the presence of all 3 viruses, did constitute some guarantee against producing too many false positives.

If the virus was actually present, all 3 genes should be found, because if the virus is intact it must be whole, as this is the only case in which it could have a pathogenic role. If only one gene is found, this could mean, at best, that there is only a fragment of the virus, and given the lack of specificity of the genes used, God knows of which virus. As pointed out recently by Dr. Mike Yeadon, finding just a gene is equivalent to finding a fragment of a "dead" virus still stuck into your organism.³⁹ In reality, no virus is alive, being just a filament of RNA; so, it would be more appropriate to say that a fragmented, not whole, virus, simply cannot be pathogenic! The only role of a broken fragment of a virus is to make the test that tests for just 1 gene a positive test, capable of creating huge numbers of falsely positive, asymptomatic individuals!

But there is more to this search for only one gene, and that is the high probability of cross-reactivity, of confusing the SARS-Cov2, or whatever is defined as such, with other presumed viruses, as shown by some researchers who evaluated the cross-reactivity of the genes used in the swab tests:

"Although these genes reported as potential targets for the detection of coronavirus, we found out that only one of them (RdRP_SARSr-P2) was almost specific for the new coronavirus and the other introduced probes

³⁸ Wagginer J et al., Triplex Real-Time RT-PCR for Severe Acute Respiratory Syndrome Coronavirus 2, Research Letter, Volume 26, Number 7—July 2020.

³⁹ <http://tapnewswire.com/2020/11/pandemic-is-over-former-pfizer-chief-science-officer-says-second-wave-faked-on-false-positive-tests/>

would detect the other types of coronaviruses. In this regard, the false-positive test results may extend for COVID-19...”.⁴⁰

And let’s not forget that the Drosten test, the first produced in Europe, has been produced, by admission of the authors themselves, without any relation with an isolated virus:

“The present report describes the establishment of a diagnostic workflow for detection of an emerging virus in the absence of physical sources of viral genomic nucleic acid.”⁴¹

The question of RT-PCR cycles (Ct = Cycle threshold)

Another serious problem of swab tests, which all use the RT-PCR, or Reverse Transcriptase-Polymerase Chain Reaction, is that the reliability of this method depends on the number of cycles (replications) which are used to find the SARS-Cov2 virus. Prof. Stephen Bustin, one of the world PCR authorities, wrote in a recent article regarding the identification of the presence of SARS-Cov 2:

“...the most widely used method is quantitative fluorescence-based reverse transcription polymerase chain reaction (RT-qPCR). Despite its ubiquity, there is a significant amount of uncertainty about how this test works, potential throughput and reliability.”⁴²

Above all, this is due to the question of the cycles of PCR that are normally performed with the swab tests. In an interview with the late and valuable Canadian researcher David Crow, Bustin states:

“...the cycle number per se is not a good measure...most instruments, when you get above a cycle number of 35, then you start worrying about the reliability of your result...so, you want to be sure that your results are within the 20 to 30 cycles...”

And given that most Covid-19 swab tests use often 40 cycles, Crow asks:

“...if you get up to 40 cycles, you could get a ghost, the PCR could string bases together casually...”;

To which Bustin replies:

“I would be very unhappy about 40 cycles...”.⁴³

⁴⁰ Kakhki RK et al, COVID-19 target: A specific target for novel coronavirus detection, Gene Reports 20 (2020) 100740

⁴¹ Corman V et al, Detection of 2019 novel coronavirus (2019-nCoV) by real-time RT-PCR, Euro Surveill. 2020 Jan 23; 25(3): 2000045, p.10.

⁴² Bustin S.A, Nolan T., *RT-qPCR Testing of SARS-CoV-2: A Primer*, Int. J. Mol. Sci. 2020, 21, 3004; doi:10.3390/ijms21083004, p. 1.

⁴³ David Crow, The Infectious Myth: <https://infectiousmyth.podbean.com/e/the-infectious-myth-stephen-bustin-on-challenges-with-rt-pcr/>

Recently this has been confirmed by a series of published studies. Canadian researchers tested at what PCR Cycle Threshold the viral load could still be considered infective: they incubated SARS-Cov2 positive biological samples in vitro to see which ones could replicate, and so be considered infective:

“Ninety RT-PCR SARS-CoV-2–positive samples were incubated on Vero cells. Twenty-six samples (28.9%) demonstrated viral growth...There was no growth in samples with a Ct > 24 or STT > 8 days...SARS-CoV-2 Vero cell infectivity was only observed for RT-PCR Ct < 24...”⁴⁴

The idea was confirmed that above 25 cycles of PCR, whatever is found should be considered negative for all practical purposes. The same has been confirmed by a more recent scientific article:

“...at Ct = 25, up to 70% of patients remain positive in culture and...at Ct = 30 this value drops to 20%. At Ct = 35...<3% of cultures are positive.”⁴⁵

At 35 Cycles of PCR, true positivity to Covid-19 test is below 3%, that is 97% are false positives! And almost all Covid-19 swab tests normally use a Ct >35 :

⁴⁴ Bullar J et al, Predicting Infectious Severe Acute Respiratory Syndrome Coronavirus 2 From Diagnostic Samples, *Clinical Infectious Diseases*® 2020;XX(X):1–4

⁴⁵ Jaafar R et al, Correlation Between 3790 Quantitative Polymerase Chain Reaction–Positives Samples and Positive Cell Cultures, Including 1941 Severe Acute Respiratory Syndrome Coronavirus 2 Isolates, *Clinical Infectious Diseases, Correspondence*, 2020.

Company Prodotto Numero Gene. Ct

altona Diagnostics	RealStar® SARS-CoV-2 RT-PCR Kit 1.0	821003/ 821005	E	1–10	35.45
			S	1–10	35.99
CerTest Blotec S.L.	VIASURE SARS-CoV-2 Real Time PCR Detection Kit	VS-NC0112L VS-NC0212L	ORF1ab	10–50	35.16
			N	1–10	35.46
DAAN Gene Co. Ltd of Sun Yat-Sen University	Detection Kit for 2019 Novel Coronavirus (2019-nCoV) RNA (PCR- Fluorescence	DA0930- DA0932	ORF1	1–10	38.76
			N	1–10	36.97
BGI Health (HK) Co. Ltd	Real-time Fluorescent RT-PCR kit for	MFG030010	ORF1	1–10	32.43
Beijing Wantai Biological Pharmacy Enterprise	Wantai SARS-CoV-2 RT-PCR Kit	WS-1248	ORF1ab	1–10	36.20
			N	1–10	37.12
Bloneer Corporation	AccuPower® SARS-CoV-2 Real-Time RT-PCR Kit	SCV-2122	E	10–50	35.85
			RdRP	10–50	36.18

KH Medical Co. Ltd	RADI COVID-19 Detection Kit	RV008	S	1-10	37.94
			RdRP	10-50	36.74
bioMérieux SA	ARGENE® SARS-COV-2 R-GENE®[b]	423720 (CE-IVD) 423717 (RUO)	N	10-50	36.44
			RdRP	10-50	32.44
EUROIMMUN AG	EURORealTime SARS-CoV-2[c]	MP 2606-0425	ORF1ab/N	1-10	37.88
Boditech Med. Inc.	ExAmplicar COVID-19 real-time PCR kit (L)	UFPK-4	E	10-50	34.9
			RdRP	50-100	33.46
GeneFirst Ltd	The Novel Coronavirus (2019-nCoV) Nucleic Acid Test Kit	MPA-COVID19	ORF1	1-10	35.45
			N	1-10	36.72
PerkinElmer Inc.	PerkinElmer® SARS-CoV-2 Real-time RT-PCR Assay[c,d]	SY580	N	1-10	39.43
			ORF1	1-10	38.99
Primerdesign Ltd	Coronavirus COVID-19 genesig®	Z-Path-COVID-19-CE	RdRP	1-10	36.7
QuantumDx	QuantuMDx SARS-CoV-2 RT-PCR	Q22003	Orf1, N, S	1-10	36.8
R-Biopharm AG	RIDA®GENE SARS-CoV-2	PG6815RUO	E	1-10	37.99

This table summarises the first 16 of the 22 swab tests analysed and tested by FIND (Foundation for Innovative New Diagnostics), an organisation often referred to also by health authorities as a reliable tool for the assessment of diagnostic tools. As can be seen from the table I have selected (for the complete table see: <https://www.finndx.org/covid-19/sarscov2-eval-molecular/>), apart from a couple of cases at the limit, all the others use more than 35 cycles, sometimes even close to 40. And what is declared by companies is often not what happens in practice. I was sent by a friend the reply of a laboratory in Italy, that stated that they look for the RdRp and E gene, and that their standard for PCR is 50 cycles!

ISS of the Italian Government:

in this epidemic status, the swab test give up to 91% of false positives!

FIND emerges as relevant in relation to another critical aspect of the evaluation of the swab test's efficacy. In the document "In Vitro Diagnostic Devices for COVID-19. Part 2: market evolution and information for stakeholders", of 23 May 2020, the Istituto Superiore di Sanità makes an in-depth analysis of the swab test devices in circulation in Italy, underlining the tension existing between **sensitivity**, the ability of the tests to detect as much viral RNA as possible, and **specificity**, that is the need for this viral RNA to refer only to the virus you are looking for, in this case the SARS-Cov2. As we shall see, there is a crucial third parameter, **prevalence**, but let's focus for a moment on the first two.

In the same FIND document we have seen above, the levels of sensitivity and specificity were self-reported by the manufacturing or distributing companies for all the swab tests analysed. The scores for sensitivity average between 90% to 100%, most are reported at 100% sensitivity. The scores for specificity average between 98% to 100%. These are excellent scores, that put all the devices in the upper category, that of High Performance, and this is very relevant, as we shall see. But we shall also see that these scores are self-declared, and scarcely credible. After all, how can we have an almost perfect efficacy while declaring to use an average of above 35 cycles of PCR? Something is clearly amiss!

Going back to the ISS, they so present the tension between sensitivity and specificity:

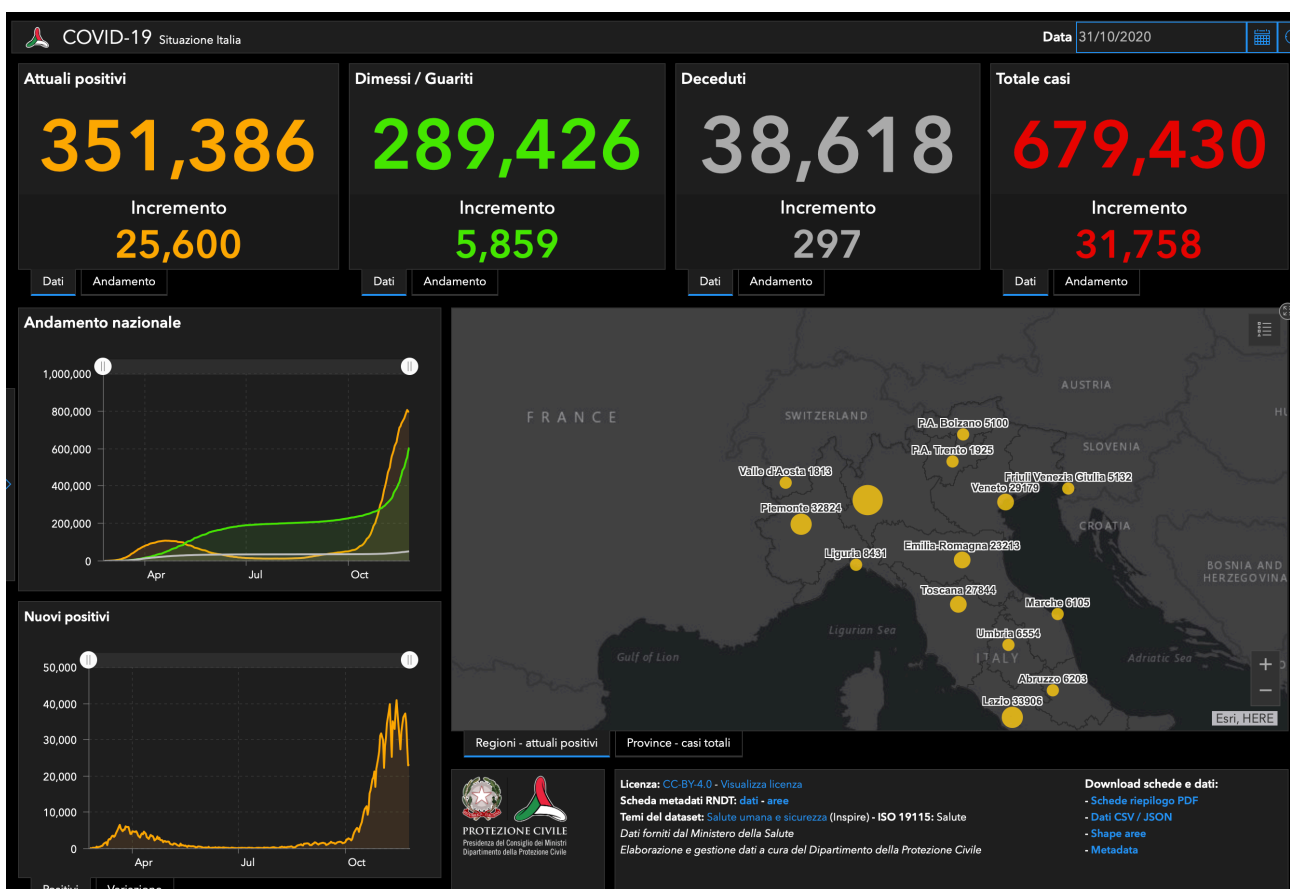
“A very sensitive test in detecting the target of interest has more likely to detect related but distinct targets than not are of interest, meaning that it may be less specific.”⁴⁶

Even the ISS knows that with a very high sensitivity it is very likely to have a low specificity, but miraculously all the self-reported scores by the swab test companies report almost 100% for both sensitivity and specificity. Be as it may, the ISS then explains that the tension between sensitivity and specificity is modulated by another factor, namely **prevalence**. In the epidemiological context, prevalence describes the percentage of the population affected by a certain pathology. In the case of a presumed viral pathology such as Covid-19, the prevalence indicates how many people are currently infected by the Covid-19 virus in relation to the total population. Why is this data important in relation to the reliability of the swab tests? Because the greater the percentage of the affected population, the greater the circulation of the virus, and therefore the greater the probability that the swab test will detect actually that virus rather than other viruses. The ISS thus adopts the FIND table that establishes the effect of prevalence over the effectiveness of swab tests. FIND is an authoritative international organisation, and its standards have an international relevance. Writes the ISS introducing the Table:

⁴⁶ Gruppo di Lavoro ISS Test Diagnostici COVID-19, Dispositivi diagnostici in vitro per COVID-19. Parte 2: evoluzione del mercato e informazioni per gli stakeholder, Rapporto ISS COVID-19 n. 46/2020, 23 Maggio 2020, p. 6.

“In the following table, taken from the Rapid diagnostic tests document for COVID-19, with a numerical example it is shown how the ability to correctly identify positives (PPV column) correlates both to the sensitivity and specificity of the test, and to the prevalence of the marker in the target population, exemplified by four cohorts of 1,000 people with **4 different prevalence values: 2%, 5%, 10%, and 30%.**”⁴⁷

Hence, the ability of the test to correctly detect the presence of the virus depends on 3 factors, all considered in the Table, namely sensitivity and specificity, but in light of the prevalence; and the Table takes into account 4 prevalence levels: 2%, 5%, 10% and 30%. Before looking at the Table itself, let's see at which of the four groups belong the Italian and European situations. The following is the Covid-19 situation in Italy as of 31 October 2020:



When talking of prevalence, things tend to get muddly. If we take the number of actual test-positives individuals, 351.386, this represents about 0.58% of the population, well below the 2% minimum considered by the FIND table. However, there are plenty of people who claim that the spread of the virus is much larger, based on hypothetical projections. For instance, currently in Italy the percentage of people testing positives is about 15%, a very high number (certainly due to the high number of false positives generated by the distorted PCR methodology). On

⁴⁷ ibid., which quotes FIND, *Rapid Diagnostic Tests for Covid-19*: https://www.finddx.org/wp-content/uploads/2020/05/FIND_COVID-19_RDTs_18.05.2020.pdf

the other hand, a recent mass testing via a rapid antibody testing on 60% of the population in the Trentino Alto Adige region gave a percentage of positives of only 1%.⁴⁸ So, let's take two values: the 2% as being the closer to the real 0,5% prevalence; and the 10% as being closer to the average between the 15% found with the very questionable PCR tests and the 1% found with the antibody tests. This is the FIND table:

Cohort	Pre-test probability (prevalence)	Sensitivity	Specificity	Cases	Non-cases	True positive (TP)	False negative (FN)	True negative (TN)	False positive (FP)	PPV	NPV
High performance											
1,000	2.0%	95%	98%	20	980	19	1	960	20	49.2%	100%
1,000	5.0%	95%	98%	50	950	48	2	931	19	71.4%	100%
1,000	10.0%	95%	98%	100	900	95	5	882	18	84.1%	99%
1,000	30.0%	95%	98%	300	700	285	15	686	14	95%	98%
Mid performance											
1,000	2.0%	85%	90%	20	980	17	3	882	98	14.8%	100%
1,000	5.0%	85%	90%	50	950	43	8	855	95	30.9%	99%
1,000	10.0%	85%	90%	100	900	85	15	810	90	48.6%	98%
1,000	30.0%	85%	90%	300	700	255	45	630	70	78%	93%
Low performance											
1,000	2.0%	75%	85%	20	980	15	5	833	147	9.3%	99%
1,000	5.0%	75%	85%	50	950	38	13	808	143	20.8%	98%
1,000	10.0%	75%	85%	100	900	75	25	765	135	35.7%	97%
1,000	30.0%	75%	85%	300	700	225	75	595	105	68%	89%

Now the question becomes: but at what level of performance can be placed the majority of the circulating swab tests? First, let it be clear, as we have seen, that all the declarations on the sensitivity and specificity of the devices are self-declarations from the manufacturers, given that no one of the circulating swab test has undergone any external valuation or validation. In fact, almost none of the circulating swab tests even report which genetic sequences (primers and probes) they use for their PCR.

If we go back to the EU Commission document cited above, that document makes also a very important statement concerning that lack of probes and primers declaration in the great majority of the swab tests in circulation:

⁴⁸ <https://www.lastampa.it/cronaca/2020/11/22/news/covid-test-di-massa-in-alto-adige-dopo-270mila-tamponi-rapidi-solo-l-1-per-cento-e-positivo-1.39570355>

“This [the lack of specification of primers and probes] makes the complementation of **performance information** from what is published in the scientific literature for an individual device conditional on the explicit mentioning of the device in the ‘materials and methods’ part of a publication.”

The consequence of this statement may not be immediately clear. The EU document is essentially saying that whatever each swab test device self-reports, in terms of sensitivity and specificity, does not have any value unless that device has been explicitly cited in a published scientific article on the topic. And as the EU document itself included all the scientific published articles on performances, and the same proportion of devices that does not report primers and probes is also not cited in any scientific article, that means that the calculations of effectiveness of the swab tests made by governmental offices, such as the ISS in Italy, are all based on the self-declaration of the swab test producers, without any scientific support! Even when checking for such studies today, all that I could find is one article from a Dutch group of researchers, which analysed only 7 out of the more than 100 circulating, and even with those they had to admit that, at least concerning the E gene, they could not guarantee the lack of cross-reactivity with tether viruses, that is they could not guarantee the lack, or very low levels, of false positive results.⁴⁹

It is thus no surprise that the EU document itself ironically stresses how the **“...manufacturer’s claims on both the diagnostic sensitivity and specificity were very optimistic...”**⁵⁰

If we add to that the fact that for the most part circulating swab tests make use of more than 35 and sometime up to 50 PCR cycles, thus generating meaningless results, we must conclude that the values of sensitivity and specificity of the swab tests will have to be taken at their lowest, not at the self-declared upper level! We may just be generous and accept the presence of both low and mid performance devices.

With this awareness, let’s look at what emerges from the ISS/FIND table:

⁴⁹ Elslande JV et al, Diagnostic performance of seven rapid IgG/IgM antibody tests and the Euroimmun IgA/IgG ELISA in COVID-19 patients, Clin Microbiol Infect, 2020 Aug; 26(8):1082-1087.

⁵⁰ European Commission, Working Document, Current performance of COVID-19 test methods and devices and proposed performance criteria, April 16 2020., p.16

Level 2%	True positives	False positives
2% Mid performance	14.8%	85.2%
2% Low performance	9.3%	90.7%
Level 10%	True positives	False positives
10% Mid performance	48.6%	51.4%
10% Low performance	35,7%	64,3%

So, in the best case scenario, with a level of 10% prevalence and a mid performance device, we **would still have 51.4% of false positives**; which goes **up to 90.7% false positives** in the worst case and more likely scenario or 2% prevalence and low quality devices.

In conclusion, my own position is that the pandemic is clearly false and there are no numbers to support its existence, as I believe I have shown. Also, given that, as we have seen, the SARS-Cov2 virus has never been isolated and so its existence is purely conjectural, the respiratory disease associated with Covid-19 cannot but be the usual pneumonias and flu-like syndrome that have always existed and that have grown progressively stringer and more widespread in the last decade.

Finally, given the lack of isolation of the virus, and thus the lack of what is called “the golden standard” for the search of the virus with the RT-PCR, the swab tests that have invaded our nations have no meaning at all, and their results are purely causal. They are, in other words, 100% false.

However, even if and when accepting the reality of the virus, and thus the fact that the swab test do actually search for something real and known, the very official documents on the issue of diagnostic performance in different level of virus prevalence, state very clearly that at these levels of prevalence and with the current quality of the swab tests, the false positives vary from 51% in the best case scenario, to 91% in the worst and most likely scenario. Isn't it time we stop this self-destructive folly of Covid-19 that is devastating our economies and societies?