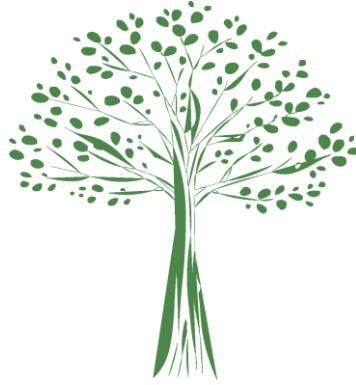


# COVID-19 SCIENTIFIC REVIEW REPORT

BIÓLOGOS POR LA VERDAD  
(Biologists for the Truth)

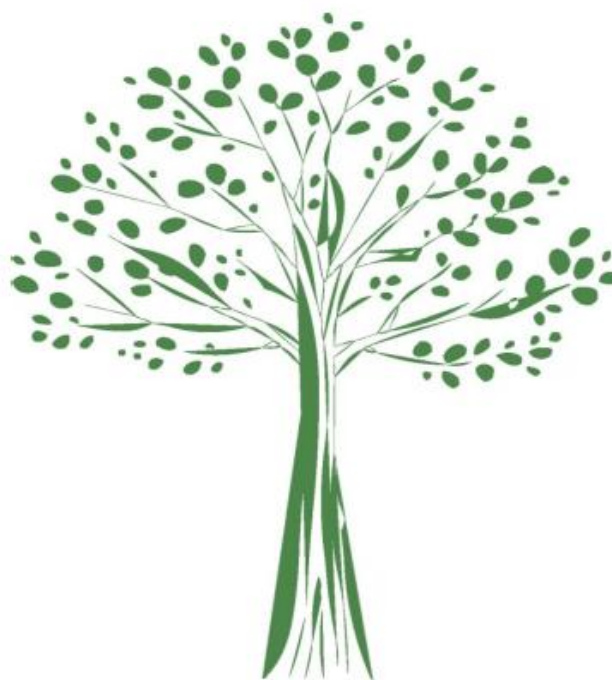


March 15th, 2021

[www.biologosporlaverdad.es](http://www.biologosporlaverdad.es)

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## 0. INTRODUCTION: THE PROFESSION OF BIOLOGY IN THE FACE OF THE PANDEMIC

Grouped under the name *Biologists for the Truth*, we are professionals in biology and the environmental sciences. We have elaborated a scientific review report with the purpose of addressing official institutions, health authorities and the general public concerning the lack of presence of collegiate representatives of our profession in the public arena, and also concerning the fact that throughout this pandemic our profession has not taken a leading role in such a critical matter, which is supposed to be of viral origin, although the pertinent scientific knowledge in this matter is essential to our profession. However, to our dismay, other kinds of collegiate professions with intrinsically less scientific knowledge of viruses are those which have led the way by setting themselves as essential professionals and leaders for the general public, using speeches which bear no hint of reasonable doubt and which offer no criticism of the official discourse.

The intention of the undersigned group of biologists is to attempt to awaken our profession to take that leadership and to critically analyse everything that has happened during this pandemic, so that as a result at least a minimum of reasonable doubt may be established in our professional sphere.

Making a compilation of what has happened with this pandemic since its beginning in the month of March 2020 to the present, we discuss what has been done in the past, what is currently being done in the present, and what will apparently be done in the future. To achieve this end, we enclose a scientific review report with 101 scientific references and arguments that allow us to affirm the following assertions:

1. Viruses are the origin of life; they are present in all living organisms, both inserted as part of their genome and also performing vital functions as part of their microbiome.
2. The theory of contagion and the struggle against biological entities, such as bacteria and viruses, is a self-destructive struggle against life itself, being in absolute opposition to biology, which is the science that studies life.
3. The SARS-CoV-2 virus is an artificial chimera virus whose origin must be a laboratory, as we know in biology that there is a species barrier which can only be crossed by virus cultures in animal cells, a situation which can only occur under controlled conditions and never in nature.
4. The purported isolation of the SARS-CoV-2 is a scientific fraud, as was the isolation of SARS-CoV, due to the fact that no viral cultures or viable viral particles have been obtained. Furthermore, viruses in genomic libraries or databases cannot be considered real pathogens without demonstrating their direct growth in human cells and without going through animal cultures. In any discussion, one should demonstrate direct growth in respiratory cells of the human respiratory tract.

5. The accepted ACE 2 receptors for the SARS-CoV-2 are not found in the lung nor in the respiratory tract; so, there is no evidence that it is a respiratory virus, and therefore facemasks are a useless measure which is causing serious disorders and pathologies in the population.
6. Human-to-human transmission of this virus has not been scientifically proven. A positive RT-PCR test cannot be considered as proof of any contagion, since PCR is a technique that should never be used to diagnose a disease.
7. The WHO-approved RT-PCR test protocol is not specific to SARS-CoV-2, as it may detect human endogenous retroviruses, such as the human coronavirus NL63 in its extracellular phase, or it may detect other components of the human transcriptome. We should remember that endogenous retroviruses have sequences that are highly conserved and homologous to the primers and probes used in the RT-PCR protocols; so, we may simply be detecting the expression of endogenous viruses related to the common cold.
8. The variation in the number of cycles used for RT-PCR testing in different laboratories is equally unacceptable due to the fact that this parameter is easy to manipulate, which can be done at the convenience of the authorities, with more cycles more positives. In fact, RT-PCR tests set at more than 20 cycles should not be accepted.
9. A positive RT-PCR test can never be considered as a contagion or a case of disease, as this test is not only non-specific, but in this case it does not even detect viable viral particles but RNA fragments. If no cell culture of the patient is performed, it is not possible to state that the patient is infected with SARS-CoV-2, and therefore, the test does not meet the gold standard.
10. Due to a lack of scientific evidence, we do not accept the association of an RT-PCR positive test with an asymptomatic capable of infecting. There are no conclusive studies on the SARS-CoV-2 virus contagion at present; so, a person without symptoms is a healthy person.
11. From a scientific point of view, we find that the injection of genetically modified organisms or fragments of genetic material into healthy people by means of substances deceptively called vaccines is not only a mistake, but it is also a dangerous practice which is in total disregard of the deontological principles of our profession, since these substances are not vaccines. In fact, people are being coerced to agree to this dangerous experiment falsely called a vaccine under the threat of losing their rights, even though these substances have been shown to cause serious side effects, as already reported to drug agencies around the world. Not a single adverse effect is worth tolerating.
12. The gene products used have serious deficiencies at the biological level, such as homology with human endogenous retroviruses and their retroviral proteins, which are of vital importance for reproduction, the immune system, and the neurological system, among other systems. These substances may cause serious pathologies of autoimmunity, infertility and neurodegeneration, among others.

13. The statistical data on positive PCR test results associated with the Cumulative Incidence has been used to establish measures that restrict fundamental freedoms and rights and has thus destroyed the socioeconomic fabric of our country, since the data has been overestimated and poorly analysed, for at no time has the data been standardised, nor has the percentage of positives been used.
14. With the exception of an unusual peak of mortality detected between March and April 2020, it is also clear from the statistics that COVID-19 has replaced influenza, without this disease having disappeared at any time, but instead it has been renamed.
15. The unusual peak of excess mortality in March-April 2020 occurred in response to the 2019-2020 influenza vaccine, for there is sufficient evidence to support the fact that it played an important role in causing that peak. Considering the flu vaccine, together with measures that caused the abandonment of groups of elderly people in nursing homes, where most deaths occurred, in conjunction with medical neglect or misdiagnosis and the possible role of recently installed networks emitting electromagnetic radiation, we have come to the conclusion that this peak of mortality was not caused by the SARS-CoV-2 virus, which was blamed without evidence. It is precisely this peak that needs to be studied in order to find out what really happened, while disregarding PCR determinations because of their total inability to diagnose disease, and also disregarding the claim that this SARS-CoV-2 artificial chimera virus is causing the usual epidemic of seasonal flu.

We, the undersigned, as professionals in biology and the environmental sciences, dissociate ourselves from the fear-based official narrative about viruses, including their criminalisation, and we also dissociate from unsubstantiated theories and anti-scientific malpractice. We openly and publicly declare ourselves in favour of respecting the rights and freedoms of the people, which have been curtailed in the name of a virus, which has never been empirically proven to be circulating in the population but whose origin should be directly investigated. It is our hope that this letter and the attached scientific report will serve to establish a debate within our profession and thus serve to express our voice to the general public.

This voice, by the way, is most qualified at the curricular level as compared to the different voices expressed by people from other health professions, who have not ceased to follow the official discourse without the slightest criticism of what has happened with this pandemic, which is why we ask for your collaboration to clarify this evidence.

Sincerely.

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*"The permanent war against the biological entities that have built, regulated and maintained life on our planet is the most serious symptom of a civilization alienated from reality that walks towards its self-destruction."*

Quoted from the article: *La guerra contra bacterias y virus: una lucha autodestructiva* (2009) (The war against bacteria and viruses: a self-destructive struggle) by Máximo Sandín, Doctor in Biology; available at <http://somosbacteriasyvirus.com/lucha.pdf>

## 1. ON VIRUSES.

Based on the latest discoveries at the planetary level, the abundance of viruses in the ecosystems is staggering, since we are talking about 10 nonillion; that is to say, if we were to send a virus from Earth to every star in the universe, we would have to do it 100 million times to send them all (1). They are responsible for the insertion of photosynthesis genes into bacteria, which made it possible for us to breathe oxygen (2). They are responsible for the nucleation of clouds and snowflakes (3), which led to the beginning of the water cycle, and we even know that they provided the genetic pool for higher lifeforms, as they are the components that make up living organisms (4) as well as essential parts of the biogeochemical and ecological cycles on our planet (5).

In human beings, the importance of the role of viruses in the maintenance of an adequate balance in the organism is vital. They are part of the cellular genetic material, as is the case of endogenous retroviruses (6), which participate in processes of great importance, such as telomere formation (7), placentation (8), fertilization (9), and even signal transduction in the cells of the central nervous system (10).

In addition to retroviruses, our genome contains sequences of the non-LTR retrotransposon type called LINE and SINE, which are also of viral origin as are other mobile elements, such as DNA transposons. Even in what is considered the dark parts of the genome, elements of the human transcriptome have been found; it is a set of RNA molecules responsible for triggering or terminating important physiological and metabolic processes, which are known to be expressed in all tissues of the body (6, 11). It has been shown that the functions of these endogenous human viruses are so important that they are considered to be responsible for innate immunity (12).



Outside our genome, in mucous membranes and skin, the number of viruses that are part of the microbiome is between 5 and 25 times higher than that of bacteria, and their role is also fundamental in maintaining our body's equilibrium. It is now known that the bacteriophages that adhere to our mucosal surfaces provide us with an innate immunity that is not host-derived but is one of the main protective barriers of an organism (13).

Their role in life being of such importance, and in light of the latest discoveries in molecular biology and genetics, we support a strong proposal to change the current definition of viruses to the following:

*"The reality is that viruses have been involved in the evolution of life from its very origin. As a consequence of their determining role [...], the genomes of living beings are for the most part made up of endogenous viruses, i.e. viruses, mainly retroviruses, which have been inserting their genetic sequences into the chromosomes and their derivatives, the mobile elements, repeated sequences, short and long "dispersed elements", introns..."*

*In normal adult individuals, the endogenous retroviruses are expressed in all tissues confirming that they are permanent components of the human transcriptome. They are also involved in the regulation of many other genes. In other words, these are genes that are involved in the functioning of tissues. And if we add to this the activities of the viruses in the colon and on the surface of mucous membranes, it turns out that the "evil" viruses are absolutely essential for our existence. In short, viruses are, in reality, "genetic information packages." They could be defined as subroutines of life processes".*

Quoted from the book:

"Trilogía del coronavirus" (Trilogy of the Coronavirus) (2020) by Máximo Sandín, Doctor in Biology.

Published by Cauac Editorial; available at <https://cauac.org/libros/trilogia-del-coronavirus/>



## 2. ON THE ORIGIN OF THE SARS-COV-2 VIRUS.

It is vital to provide a biological answer to the origin of this virus that has terrified humanity. And the answer must come from science and common sense. In biology, there is what is known as the **species barrier**, which is a biochemical language that prevents viruses of one species from interacting with the cells of other species. In fact, in the particular case of diseases induced by viruses or bacteria entering the bloodstream, such as rabies or other diseases inoculated by mosquitoes, these diseases are in no case transmitted from human to human. This is unequivocal proof that natural zoonotic diseases are caused by the entry of foreign viruses and bacteria into the bloodstream, and that at no time the species barrier is crossed. If one traces the origin of all viruses that are claimed to have crossed the species barrier, one comes to the conclusion that the reason for this is **the way they are grown in laboratories** (14).

The cultivation of human viruses in animal cell lines through so-called "passaging" generates unnatural recombinants, which are then injected with the vaccines into the healthy population, and this is a very dangerous practice (15, 16). The ultimate expression of these artificial viruses has materialised with the creation in laboratories of chimera viruses (17), which have the characteristic of having to be injected into the bloodstream in order to evade host immunity and carry an efficient cellular transduction mechanism that allows them to cross the species barrier (18).

Having sequences from human coronaviruses, from the bat *Rhinolophus affinis*, from the pangolin *Manis javanica*, and from a canine *Beta-coronavirus*, the SARS-CoV-2 is an artificial chimera virus (19) (Table 1), a situation which would never have been possible in nature, nor are there any conclusive publications to this effect. However, we do have evidence of the creation of such chimera viruses in laboratories (17) and of the cultivation of new cell-based vaccines in dog kidney cell lines, as in the example of the FlucelVax influenza vaccine that was launched in Spain last year (20).

Source	GISAID	NCBI	NCBI	NGDC	NGDC	GISAID	GISAID	GISAID
Virus	SARS-CoV-2	Other Taxa	SARS-CoV-2	Other Taxa	SARS-CoV-2	Betacoronavirus	SARS-related coronavirus	betacoronavirus
Host	Homo Sapiens	Homo Sapiens	Homo Sapiens	Homo Sapiens	Homo Sapiens	Manis javanica	Rhinolophus affinis	Canine
# Samples	8,256	20,572	32	487	96	9	1	1
CAC GTA GGA ATG TGG CAA CTT	99.73%	0.00%	100.00%	0.00%	97.92%	0.00%	100.00%	100.00%
TATTAG TGA TAT GTA CGA CCC	99.60%	0.00%	100.00%	0.00%	97.92%	0.00%	0.00%	100.00%
AAT GAA TTA TCA AGT TAA TGG	99.55%	0.00%	100.00%	0.00%	96.88%	100.00%	0.00%	100.00%
AAT AGA AGA ATT ATT CTA TTC	99.54%	0.00%	100.00%	0.00%	96.88%	0.00%	100.00%	100.00%
CAA CTT TTA ACG TAC CAA TGG	99.38%	0.00%	100.00%	0.00%	97.92%	0.00%	0.00%	100.00%
CTA AAG CAT ACA ATG TAA CAC	99.38%	0.00%	100.00%	0.00%	100.00%	0.00%	0.00%	100.00%
TAG CAC TCT CCA AGG GTG TTC	99.29%	0.00%	100.00%	0.00%	97.92%	0.00%	0.00%	100.00%
CGA TAA CAA CTT CTG TGG CCC	98.84%	0.00%	100.00%	0.00%	97.92%	0.00%	100.00%	0.00%
TGC CAC TTG GCT ATG TAA CAC	97.57%	0.00%	100.00%	0.00%	97.92%	0.00%	100.00%	100.00%
CAT CTA CTG ATT GGA CTA GCT	97.40%	0.00%	100.00%	0.00%	97.92%	0.00%	100.00%	100.00%
TGA GCA GTG CTG ACT CAA CTC	96.06%	0.00%	100.00%	0.00%	98.96%	0.00%	0.00%	100.00%
GAT GGT CAA GTA GAC TTA TTT	95.24%	0.00%	100.00%	0.00%	96.88%	0.00%	0.00%	100.00%

**Table 1.** Found in the NCBI database at <https://www.ncbi.nlm.nih.gov/>, this table shows the only 12 sequences which do not match human endogenous coronaviruses. Published in the official WHO bulletin in April 2020 (19). Most notable about one of these fragments is that it matches the pangolin (*Manis javanica*) coronavirus; 4 of them match the horseshoe bat (*Rhinolophus affinis*), and 11 of them match a canine *Beta-coronavirus*.

### 3. SARS-COV-2 VIRUS ISOLATION AND RT-PCR TESTING.

Isolation of the virus from patients has been as questionable as the story of its origins. As early as 2003, with the description of the first SARS-CoV (21), the main signatory of that description, who, interestingly, is the same main author of the current RT-PCR protocol of the SARS-CoV-2 (22), Christian Drosten, incurred a number of serious irregularities. It was his team of researchers that in 2003 linked the SARS (Severe Acute Respiratory Syndrome) disease to a presumed new virus which they named SARS-CoV. The most relevant aspect of this association between disease and virus is **that it was made with only 300 nucleotides**, which are also shared by a large part of the coronaviruses (19), which are now known to be endogenous viruses present in all mammals and birds and which are not pathogenic (23).

This coronavirus fragment found in only one sample, in which *C. pneumoniae* bacteria were also found, was cultured in monkey kidney cells (21), and on this poor basis and without taking into account the recombination that viruses undergo when cultured in animal cells, nor taking into account any other factor that could have caused the disease, the first SARS-CoV was claimed to be the culprit by associating it with a severe acute respiratory syndrome, a condition which has now been replaced by the name COVID-19, since in 99.73% of cases it is neither a respiratory disease, nor is it acute or severe (24). Very revealing.

Today, from the same journal that published both protocols for the association of **a tiny gene sequence** with a virus they called SARS-CoV, we have the purported SARS-CoV-2 isolates in the first patients (25). To explain this scientific fraud, we will focus on the first publication that claimed to have isolated the virus from patients in Wuhan in China. The first thing that is reported in the methodology is the use of the RT-PCR test to detect small fragments representing as little as 200 nucleotides out of 30,000 in the complete genome of the suspected disease causing virus.

Using the BLAST tool (<https://blast.ncbi.nlm.nih.gov/Blast.cgi>), we have compared the probes and primers of the protocol used according to Corman and Drosten, 2020 (22), and 8 of the 10 sequences from which the complete nucleotide composition is reflected are a 100% match to the endogenous human coronavirus NL63, which in its extracellular phase is encapsidated and in RNA form and is also associated with common colds and has the same receptor as the SARS virus (26).

# Primers and probes, real-time RT-PCR for 2019 novel coronavirus

Assay/use	Oligonucleotide	Sequence <sup>a</sup>	Concentration <sup>b</sup>
RdRP gene	RdRp_SARSr-F	GTGARATGGTCATGTGTGGCGG	Use 600 nM per reaction
	RdRp_SARSr-P2	FAM-CAGGTGGAACCTCATCAGAGATGC-BBQ	Specific for 2019-nCoV, will not detect SARS-CoV. Use 100 nM per reaction and mix with P1
	RdRp_SARSr-P1	FAM-CCAGGTGGWACRTCATCMGGTGATGC-BBQ	Pan Sarbeco-Probe will detect 2019-nCoV, SARS-CoV and bat-SARS-related CoVs. Use 100 nM per reaction and mix with P2
	RdRp_SARSr-R	CARATGTTAAASACACTATTAGCATA	Use 800 nM per reaction
E gene	E_Sarbeco_F	ACAGGTACGTTAATAGTAATAGCGT	Use 400 nM per reaction
	E_Sarbeco_P1	FAM-ACACTAGCCATCCTTACTGCGCTTCG-BBQ	Use 200 nM per reaction
	E_Sarbeco_R	ATATTGCAGCAGTACGCACACA	Use 400 nM per reaction
N gene	N_Sarbeco_F	CACATTGGCACCCGCAATC	Use 600 nM per reaction
	N_Sarbeco_P	FAM-ACTTCTCTCAAGGAACAACATTGCCA-BBQ	Use 200 nM per reaction
	N_Sarbeco_R	GAGGAACGAGAAGAGGCTTG	Use 800 nM per reaction

<sup>a</sup> W is A/T; R is G/A; M is A/C; S is G/C. FAM: 6-carboxyfluorescein; BBQ: blackberry quencher.

<sup>b</sup> Optimised concentrations are given in nanomol per litre (nM) based on the final reaction mix, e.g. 1.5 µL of a 10 µM primer stock solution per 25 µL total reaction volume yields a final concentration of 600 nM as indicated in the table.

**Table 2.** Shown are the primers and probes used in this protocol, which presumably detects the SARS-CoV-2, according to Corman & Drosten, et al. 2020 (22). Note that in some of the sequences the complete nucleotide composition is not reflected, but a sequence claimed to be specific to the 2019 n CoV is provided. This protocol has been approved for use by the WHO (27).

Regiones a amplificar.	Primer y sondas.	% de coincidencia con el coronavirus humano NL63.
RdRp gene	RdRp_SARSr-F	100
	RdRp_SARSr-P2	100
	RdRp_SARSr- P1	90, 91
	RdRp_SARSr-R	91, 67
E gene	E_Sarbeco_F	100
	E_Sarbeco_P1	100
	E_Sarbeco_R	100
N gene	N_Sarbeco_F	100
	N_Sarbeco_P	100
	N_Sarbeco_R	100

**Table 3.** Shown are percentage matches of probes and primers used in the RT-PCR protocol of Corman & Drosten, 2020 (22), compared with human coronavirus NL63 (complete genome) using the BLAST tool (<https://blast.ncbi.nlm.nih.gov/Blast.cgi>).

Due to the evidence shown, this RT-PCR test proves to be completely non-specific, since it is capable of detecting endogenous viruses in the extracellular phase. Therefore, this protocol should not be used to

diagnose a disease, let alone to blame a virus for it. It should be noted that RNA fragments are amplified, and complete viral particles are never isolated, nor is the test corroborated with individual viral cultures from patients from whom the samples were collected. If it were a respiratory virus, it would be as simple to prove as collecting bronchoalveolar fluid and culturing it directly on a plate with human respiratory system cells, and that's it. However, at no time are cultures being grown directly from patient samples, but only a few RNA fragments are being collected using RT-PCR and then bioinformatically matched to the SARS-CoV-2 sequence from genomic databanks, such as GenBank (25).

**Therefore, the fragments detected in the RT-PCR are most likely from the human transcriptome, and since no viral cultures are being grown from patients said to be infected with SARS-CoV-2 (or test positive), this virus cannot be considered to be the cause of the disease called COVID-19, as there is insufficient empirical evidence of its isolation.**

Furthermore, it is of vital importance to understand that the PCR test is a technique designed exclusively for amplifying DNA and for detecting the presence of an organism or its remnants, but it does not necessarily imply disease or contagion factor. The Polymerase Chain Reaction (PCR) developed in 1986 by Nobel laureate Kary Mullis is a reaction that has the sole function of multiple copying DNA molecules in order to increase their concentration to levels high enough to be studied for the following basic purposes: (1) biochemical studies, (2) legal and forensic biology, and (3) taxonomic and phylogenetic studies.

In the case at hand, the commonly known PCR test for diagnosing patients sick from coronavirus is included in the section on taxonomic studies. That is to say, the test detects, at most, the possible presence of the virus; however, the differential sequence or sequences of its RNA are supposedly known, but not known are its concentration or viability at the clinical level or its ability to cause disease. The PCR can only copy DNA strands, but not RNA, which is the virus component, so, the RNA must first be converted to DNA by a reverse transcriptase enzyme from bacteria with high temperature reaction capability.

Sampling of RNA or DNA as the case may be is complicated and **very frequently contaminated, giving erroneous results** (28). Both sample collection and preservation is complex and must be carried out by specialised personnel in a correct time frame in order **to avoid detecting traces of already inactivated virus** (29). These conditions are not usually present at sampling sites, or sampling is performed by insufficiently trained personnel, resulting in frequent contamination. Furthermore, the reaction only detects the possible presence of virus fragments, and it is necessary to know not only the differential sequence of the RNA (in this case) but also the sequence(s) that define the given taxon, which is most of the time complex and requires studies by experts in the taxonomy of the group under study (30).

On the other hand, ***the presence of the putative virus RNA does not imply disease***, since disease determination depends on the viral load or on the number of individual viruses present. Determination of disease-producing load is analogous to determining population density of pest-producing insects in crops (31). This technique consists in establishing two linear regression lines between which the population density is to be monitored; below the line, the parasite population does not produce pest or disease, but above the line it does produce pest or disease. The surveillance of population-density threshold lines, below or above which no pest or disease is produced, is performed in statistical studies by experts in the problem taxon, while for sampling (in our case, the performance of the PCR test) a thorough knowledge of the population dynamics of the causative agent is required, though these dynamics are not always known.

Unfortunately, thresholds are not always determined by real experts. Thresholds are often established on an irregular basis in response to economic interests. The upper or lower threshold stocking density is determined by the concentrations of viral RNA (DNA). But this concentration is determined by so-called cycles. Each cycle copies the molecule  $n$  times and increases its concentration logarithmically. The theory is good, and once the threshold is determined, the fewer the number of cycles needed to reach the threshold the higher the viral load and vice versa. In reality, ***PCR is open to potential frauds because the number of cycles can be set at will, so the more cycles the more DNA (RNA) molecules and thus a higher estimated viral load, resulting in as many positive PCR tests as desired***. On the other hand, the amplification efficiency is highly dependent on the pair of oligonucleotides being used and is governed by laws that are not entirely known. The one mentioned above is the main problem of the test but there may be others, such as (1) reaction temperature, which may cause erroneous readings, (2) the use of appropriate primers, (3) errors in reading and in transcription of RNA to DNA by the reverse transcriptase used, and (4) proper purification of the sample by removal of residual DNA using *DNAase* enzymes. The test results also vary with the state of the population dynamics, as the test may detect traces of inactive viruses in people who supposedly have had the disease, thus testing positive in non-infectious individuals, and more importantly, this is done without absolute certainty that the amplified and detected RNA is specific to the virus under study and does not occur in any other taxonomically close virus. For this, it is necessary to have a Gold Standard, which can be none other than a sample of isolated and purified of SARS-CoV-2 (32).

In summary, the so-called PCR test, under very precise sample collection and laboratory conditions, is suitable for detecting the presence and specific separation of an organism, especially of DNA or parts of that organism, but it is not useful for determining a population density, which could be falsified, among other intervening factors, by the number of cycles used.

#### 4. THE SARS-COV-2 ACE 2 RECEPTOR AND AIRBORNE INFECTIONS.

Classical virology calls **infection** the interaction of the capsid proteins of a virus with certain receptors on very specific cells, a concept which nowadays is clearly under debate due to the fundamental role that viruses play in the maintenance of life and in the information exchange pathways between cells in which they are involved, as explained above. In the case of the SARS-CoV-2, it is accepted that the viral receptor is the ACE 2 enzyme (Angiotensin-Converting Enzyme 2). However, this receptor is not expressed in the lung, as the team that discovered it demonstrated in 2000 in a paper published in the *Journal of Biological Chemistry*, whose first author is Doctor Sarah R. Tipnis (33). More recently, in 2020, a Swedish team corroborated this information and confirmed that the expression of this receptor is not in the lung, nor in the respiratory tract (34). The mistake or intentionality occurred in 2003, as explained in a document prepared by the Argentinian Scientific Review Board, that human reproductive tissues are the organs in which the expression of this receptor is focused and not the respiratory tract (35). It is important to know, that the SARS-CoV-2 virus cannot be cultured in cells of the pulmonary alveolus (A549), as demonstrated by a study published in *The Journal of the Royal College of Pathologist of Australia* (36), which states that in the lung it can only be cultured in metastatic cancer cells and metastatic cells, which are not lung-specific. The Astra Zeneca's vaccine label misleadingly admits this by stating that these A549 cells do not allow replication "of the vector" (37).

***Airborne transmission (droplets and aerosols) has not been scientifically proven, which can only be done by culturing and sequencing the sample under study, as the Ministry of Health itself admits.*** Quoting from the scientific and technical information provided by the State Secretariat of Health, *Centre for the Coordination of Alerts and Health Emergencies*, updated on January 15, 2021 (38):

***"In all cases the amount of RNA detected was small and the virus could not be cultured. [...] It is therefore unknown whether it could be infective".***

The characteristic pneumonia of COVID-19 is bilateral, symmetrical and interstitial, which proves that the pathogenesis is blood-borne, since the pulmonary interstitium contains blood capillaries (25).

If we accept that COVID-19 is produced by the SARS-CoV-2 and that the cellular receptor of the SARS-CoV-2 is ACE2, then, since this virus cannot be cultured in natural lung cells, and since the ACE2 receptor cannot be found in lung tissue, and since the associated disease occurs via the blood, it must necessarily be concluded that COVID-19 is not an airborne disease and that facemasks are useless to stop transmission.



## 5. EXPERIMENTAL VACCINES.

This section outlines the scientific reasons for which administration of experimental genetic substances to the population is considered dangerous.

### 5.1 DNA-RNA-Protein interaction.

People are continually bombarded with the idea that the mRNA in the vaccine will not alter the DNA of the host cell. The reality is that the different RNAs that may be found in a cell have the function of synthesising proteins and also of regulating the expression of the genes themselves, together with proteins. Proteins are essential for cellular function and structure, and for the defence and communication of an organism at the intra- and intercellular level. The cell turns the readout of protein-forming genes on or off according to its needs. This on-off switching is due to the so-called induction- or repression-regulated expression of operons caused by the presence of certain elements (e.g. foreign proteins) or conditions in the extra- or intracellular environment. Regulation may take place at the level of transcription on the mRNA or at the level of RNA translation in the ribosome. In this way, molecules present in the intracellular environment interact with regulatory proteins specific to each operon system or operon group, and in turn, the regulatory proteins interact with an area of the operon close to the promoter (a DNA sequence located upstream of the nucleotide sequence to be transcribed).

The intermediate used by DNA to read genes and translate them into proteins is messenger RNA (mRNA), such as that used in these vaccines. Retroviruses (viruses with RNA, a group to which the *Coronaviridae* family belongs) are an exception, because they have an enzyme called *reverse transcriptase* that allows reverse transcription from RNA to DNA if necessary (39). In eukaryotic cells, although the reverse transcriptase is typical of retroviruses, there are enzymes with a reverse transcriptase function, such as telomerase, which adds deoxyribonucleotides to telomeres, but this addition is directed by an RNA (7). Contrary to the “one-gene/one-protein” model of classical genetics, modern genetics and even more so epigenetics have shown that a single gene may code for different polypeptides or proteins depending on cellular needs and on the actual reading of the gene or on interactions with neighbouring genes and sequences (40).

Proteins recognise DNA and RNA by means of DNA binding domains (41); common amino acids in proteins such as *Asn*, *Gln*, *Glu*, *Lys* and *Arg* may form hydrogen bonds between their side chains and the base pairs of DNA and/or RNA. Domains are a common evolutionary structural element in regulatory proteins of DNA-to-RNA transcription.



Mutations and polymorphisms (multiple forms of the same gene) may lead to different gene readouts with unknown consequences, while proteins via the indicated domains may cause these polymorphisms, as DNA not only has the ability to present the typical double helical structure but also to adopt three different spatial organisations (A, B and Z) with internal capabilities and great plasticity and adaptability to environmental factors, including foreign proteins inside cells. Furthermore, as in social organisations, DNA may be subjected to internal and external perturbations of different types which may affect its relationships with RNA and consequently lead to changes in the fulfilment of its purpose, ***putting life itself at risk*** (42).

There is work that introduced complementary RNA with mRNA into a cell to cause ablation of a gene; this mixture of RNA and mRNA produced a hybrid RNA which inhibited gene expression, giving rise to so-called *interfering RNA* (RNAi), which opens a new dimension for RNA in the regulation of gene expression and as an experimental and therapeutic tool (43), demonstrating, contrary to prevalent belief, that in the interaction with DNA it is not only proteins that which regulates gene expression and the reading of DNA itself, since ***RNA's may also interact to regulate the expression and reading of DNA within the cell***. Thus, the RNAi shows how wrong the central dogma of molecular biology is by stating that the information encoded in our genes is transcribed into a strand of mRNA which is then translated into the corresponding final protein; thus, ***by inhibiting the expression of a gene, the production of the final protein is blocked***. In the case of RNA interference, gene inhibition takes place by selective degradation of the messenger RNA, which, as a consequence, ***cannot be translated into the corresponding protein*** (44).

On the other hand, non-coding RNA's, as used in these vaccines, are emerging as communication factors in physiological and pathological states and have been reported to act as mRNAi (interfering messenger) sponges, interacting with mRNAi and modulating mRNA availability. Importantly, RNA's *ins* (interfering nuclear short) may have a cell-type specific expression pattern. It has been proposed that RNAinc-mRNAi interactions, analogous to receptor-ligand interactions, are responsible for cell-type specific results. The specific binding of mRNAi to RNAinc may lead to cell-type specific signalling cascades and may modulate biochemical feedback loops that ultimately determine cell identity and response to stressors (45). In recent years, the importance of genomic variants which alter pre-mRNA processing or maturation and cause various diseases has been highlighted. It is estimated that approximately 15% of disease causing mutations affect the pre-mRNA maturation process (46).

Taking into account that the vaccines used are based on non-coding mRNA from coronavirus, and that the dogmatic orthodoxy of molecular biology – which stipulates that information encoded in genes is transcribed into a strand of mRNA which is then translated into the corresponding final protein – was overturned

by the discovery of RNAi, and adding the fact that the complex interactions of DNA, RNAs and proteins are yet to be fully understood, together with the fact that new discoveries keep showing that not only is there DNA-protein intercommunication but also functional and three-dimensional plasticity of DNA occurring as a function of complex DNA-RNA-protein interactions, including the presence of exogenous and endogenous self-compounds in the cell, ***all leads to the consideration that these vaccines are not safe and do not give a 100% assurance that there will be no interaction of the mRNA introduced and the proteins produced in the ribosomes with the genetic material of human host cells.***

## **5.2 Retroviruses and their implications for evolution, creation of biological structures and physiological activities of cells and organisms – in clear conflict with gene vaccines.**

As discussed in the first section, it is thought that 10% of the human genome is composed of endogenous retroviruses, i.e. viruses which have over the course of evolution inserted their gene sequences into our genome. But if we take into account the virus-derived sequences (mobile elements such as transposons and retrotransposons, short and long repeated elements, introns, etc.), we find that the vast majority of our genomes are made up of viruses and their derivatives (98.5%), which control the expression of protein-coding genes, which is what was classically considered as the genome, when it accounted for only 1.5% of the entire genome (47).

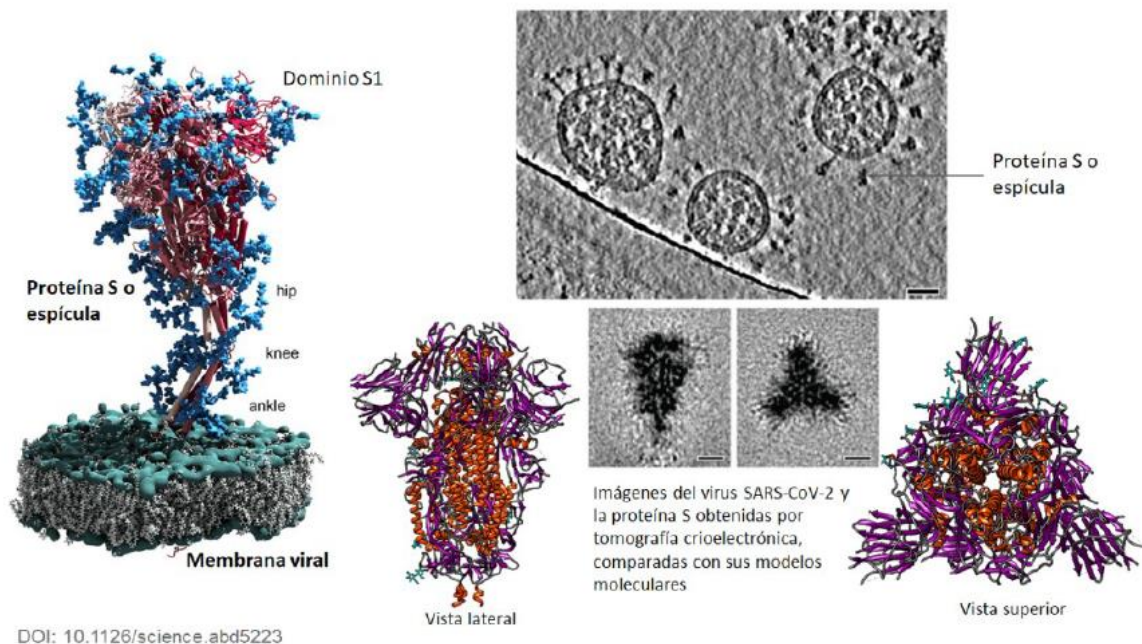
It has long been known that endogenous viruses are expressed as a constituent part of the genomes, i.e. they ***are the genome***. This fact is of great significance, since endogenous retroviruses or parts thereof are expressed in such important processes as in the production of key enzymes (48) or in the formation of the placenta during pregnancy (49). In embryonic tissues, a multitude of endogenous retroviruses are expressed (developmentally involved). As can be seen, retroviruses are expressed in placenta, adrenal cortex, kidneys, tongue, heart, liver, and central nervous system, as well as in all other tissues. And in normal adults, endogenous retroviruses are expressed in all tissues, confirming that they are permanent components of the human transcriptome (50, 51, 52).

An important function inherent to humans is the storage of memory in the brain. Several decades ago it was suggested that memory was possibly stored in the form of RNAs or proteins. Very recently, in 2017, it was found that memory and memories are stored and preserved by a mysterious protein called Staufen homolog 2 (Stau2) bound to an RNA, and it is precisely an mRNA that which is responsible for going to specific sites in the brain in order to programme the specific proteins that store that information, and this is the aforementioned protein which directs the mRNA to the synapses (53).

In 2018, the responsible gene *ARC* was discovered, and it turns out to be a gene from a retrovirus integrated into our genome. Furthermore, this gene is capable of self-assembling into capsids similar to the virus it produces, being mRNA that which mediates transfer to other neurons in a new signalling and communication pathway between neurons similar to the process by which viruses infect cells (54). For this reason, introducing foreign DNA or RNA into our bodies will cause a clear interaction with the natural expression of our genome, causing severe and unpredictable imbalances.

### 5.3 Human retroviral proteins in potential conflict with the "vaccine" coding for the spike protein of the SARS-COV-2.

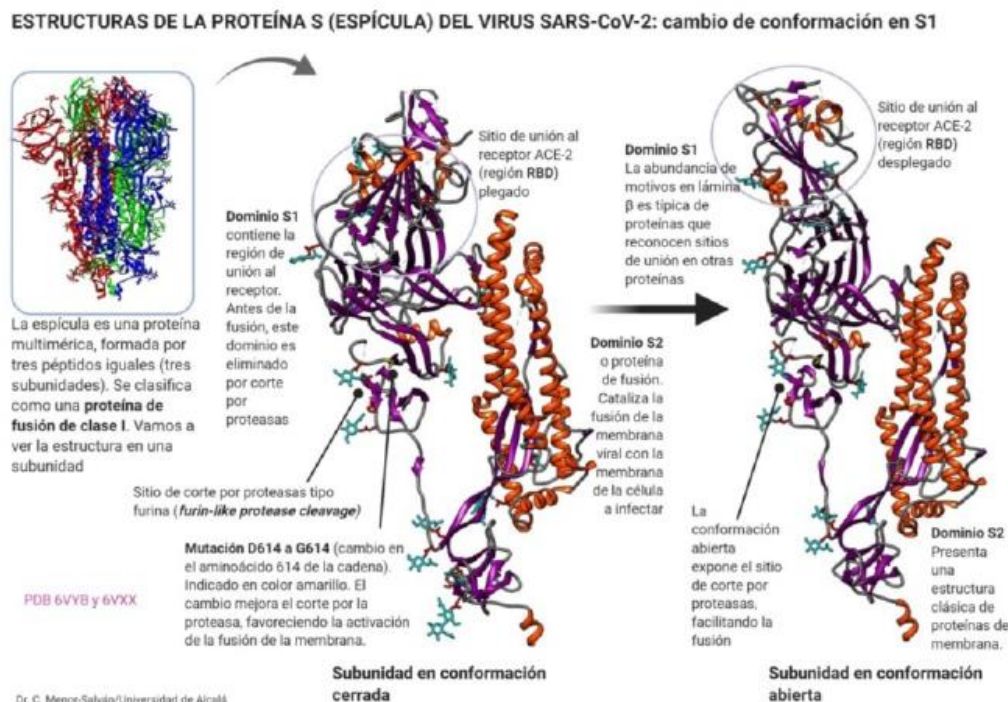
Members of the *Coronaviridae* family are viruses that possess "corona-like" projections that correspond to polypeptides called *spikes*. These, in turn, are composed of two proteins, a globular "head" of approximately 160 kilodaltons in size, called **S1**, and a fibrous foot of approximately the same size, called **S2**. Each spike protein is composed of three long polypeptides of about 1200 amino acids each, and the complete protein with its three monomers has a total of 3600 amino acids (**Image 1**) (55).



**Image 1.** Morphology of the S spike protein.

The S1/S2 association constitutes a protein complex, which is a complete protein composed of three S1/S2 monomers, so that each complete spike protein forms a trimeric (six-peptide) structure (56). The fibrous protein S2 is of the transmembrane type responsible for fusion with the cell membrane and has two repeat regions called *HR1* and *HR2*. The HR2 region is located near the membrane anchor and the HR1 region about 170 amino acids from the membrane. These HR repeat regions are found in many proteins in the human body and are one of the fundamental characteristics of the key features of Class I entry fusion proteins (57). The general characteristic of this set of proteins is that they are trimers in their pre-fusion and post-fusion states and their final states. They are characterised by having a central trimeric N-terminal  $\alpha$ -helical coil decorated by three "C-terminal" helix.

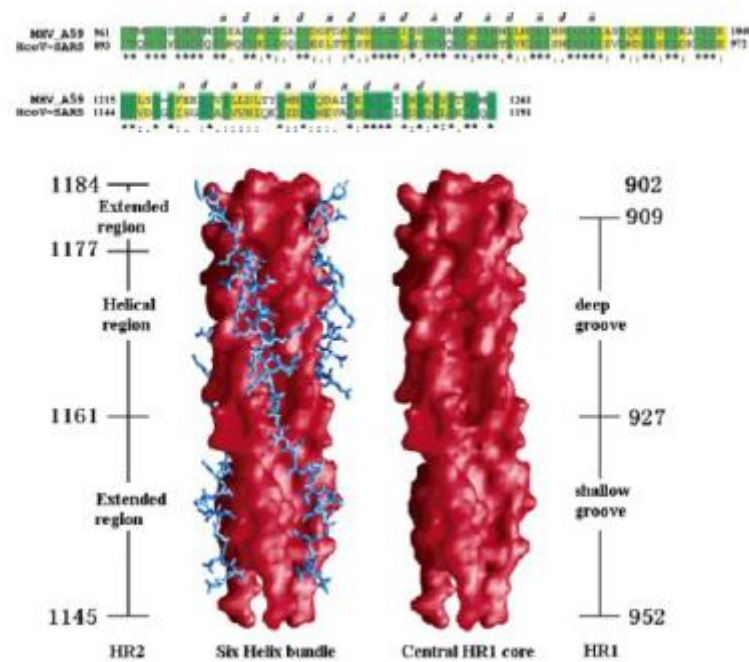
Most viral fusion proteins are expressed as precursor proteins, which are cleaved by cellular proteases, resulting in a complex consisting of a receptor binding subunit and a membrane fusion subunit. Following binding of the receptor to the cell membrane after endocytosis, the fusion proteins undergo a dramatic conformational transition (**Image 2**) (58).



**Image 2.** Conformational change undergone by the spike proteins after binding to the receptor of the cell membrane with which they fuse.

These proteins are characterised by a number of regions that may be highly conserved; in this context, the sequence of the spike protein of the SARS-CoV-2 virus ***shows a high similarity to the Class I fusion proteins of endogenous human viruses that are expressed as part of our genome*** (59).

In this fusion unit, we find the regions of the S2 domain – HR1 and HR2 – which form a central trimeric core with three copies of each; this structure is called a 6-helix coiled-coil bundle. These regions are highly conserved in the animal kingdom and are therefore homologous to endogenous human viruses, such as the ***Influenza virus and HIV*** (Image 3) (60).



**Image 3.** The HR1 and HR2 domain of the spike protein responsible for fusion with the cell membrane.

In this regard, virologist Bill Gallaher observed a direct homology relationship between the region comprising the major part of the HR1A region of the SARS-CoV-2 Wuhan and HR1 encoded in the SYN 1 region of the endogenous retrovirus *HERV-W*, showing a direct relationship between the two (61). This human syncytin 1 region, belonging to the human endogenous retrovirus *HERV W* (chromosome 7), encodes for the syncytin protein whose expression is centred in the placenta, more specifically, in the syncytiotrophoblast, a set of cells that form the outermost layer of the placenta, involved in the development of the blood supply to the embryo (62, 63, 64).

In parallel, we have checked the sequence homology between the HR1 region of SARS and syncytin 1 with the BLAST tool, obtaining a result of 87.50% homology.



## BLAST® >> blastp suite >> results for RID-1RRFG9CE016

Your search parameters were adjusted to search for a short input sequence.

Job Title [Protein Sequence...](#)  
 RID [1RRFG9CE016](#) Search expires on 02-05 18:43 pm  
 Program BLASTP  
 Database RefSeq protein  
 Query ID lcl|Query\_29804  
 Description [None...](#)  
 Molecule type amino acid  
 Query Length 16

### Descriptions

Description	Scientific Name	Max Score	Total Score	Query Cover	E value	Per. Ident	Acc. Len	Accession
<a href="#">syncytin-1 precursor (Homo sapiens)</a>	<a href="#">Homo sapiens</a>	41.4	56.8	87%	6e-06	87.50%	538	<a href="#">NP_001124397.1</a>
<a href="#">endogenous retrovirus group FC1 Env polyprotein (Homo sapiens)</a>	<a href="#">Homo sapiens</a>	32.9	32.9	87%	0.006	68.75%	584	<a href="#">XP_011529387.1</a>
<a href="#">syncytin-1-like (Homo sapiens)</a>	<a href="#">Homo sapiens</a>	31.2	31.2	81%	0.024	73.33%	251	<a href="#">XP_011530744.1</a>
<a href="#">syncytin-2 preproprotein (Homo sapiens)</a>	<a href="#">Homo sapiens</a>	29.5	29.5	87%	0.098	68.75%	538	<a href="#">NP_997465.1</a>

The HR2 region of the SARS-CoV-2 spike protein also has homologies with retroviral HR2; to check this, we have compared with the BLAST tool **RH2 REGION SARS FASTA**:

**DISGINASVVVNIQKEIDRLNEVAKNLNESLIDLQEL with syncytin-1 and the output is as follows:**

## BLAST® >> blastp suite >> results for RID-WDGVJD9K016

Your search is limited to records that include: human (taxid:9606)

Job Title [Protein Sequence...](#)  
 RID [WDGVJD9K016](#) Search expires on 12-03 05:28 am  
 Program BLASTP  
 Database nr  
 Query ID lcl|Query\_91613  
 Description [None...](#)  
 Molecule type amino acid  
 Query Length 574

### Descriptions

Description	Common Name	Max Score	Total Score	Query Cover	E value	Per. Ident	Acc. Len	Accession
<a href="#">syncytin-2 preproprotein (Homo sapiens)</a>	<a href="#">human</a>	1107	1107	93%	0.0	100.00%	538	<a href="#">NP_997465.1</a>
<a href="#">unnamed protein product (Homo sapiens)</a>	<a href="#">human</a>	294	294	25%	5e-97	100.00%	144	<a href="#">BAC11396.1</a>

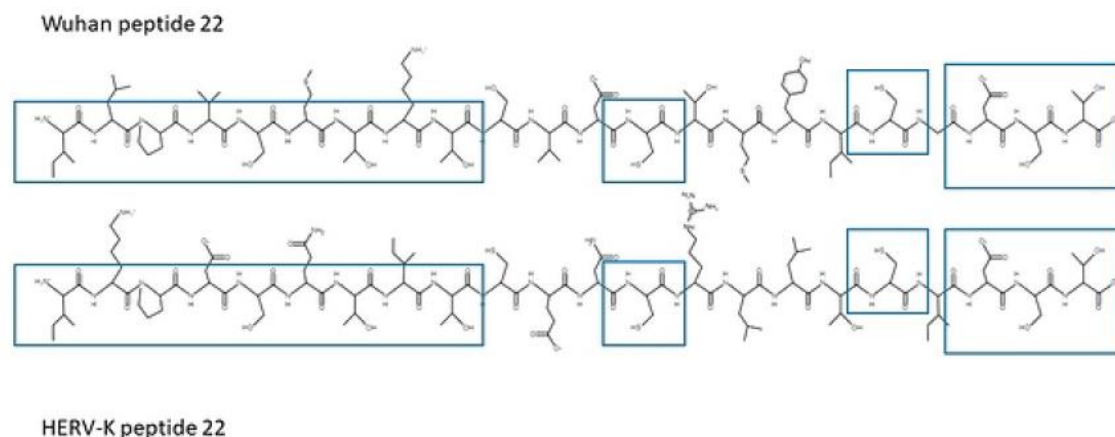
syncytin-2 preproprotein [Homo sapiens]

Sequence ID: **NP\_997465.1** Length: 538 Number of Matches: 1

Range 1: 1 to 538

Score	Expect	Method	Identities	Positives	Gaps	Frame
1107 bits(2863) 0.0()		Compositional matrix adjust.	538/538(100%)	538/538(100%)	0/538(0%)	
Query 37	MGLLLLVLILTPSLAAYRHPDFP	LLEKAQQL	LQSTGSPYSTNCWLCTSSSTETPGTAYPA	96		
Sbjct 1	MGLLLLVLILTPSLAAYRHPDFP	LLEKAQQL	LQSTGSPYSTNCWLCTSSSTETPGTAYPA	60		
Query 97	SPREWTSIEAELHISYRWDPNL	KGLMRPANSLL	STVKQDFPDIRQKPPIFGPIFTNINLM	156		
Sbjct 61	SPREWTSIEAELHISYRWDPNL	KGLMRPANSLL	STVKQDFPDIRQKPPIFGPIFTNINLM	120		
Query 157	GIAPICVMAKRKNGTNVGLP	STVCNVFT	FTVDSNQQTQYTYHNQFRHQPRFPKPPNITF	216		
Sbjct 121	GIAPICVMAKRKNGTNVGLP	STVCNVFT	FTVDSNQQTQYTYHNQFRHQPRFPKPPNITF	180		
Query 217	PQGTLLDKSSRFCQGRPSSC	STRNFWFRPADYNQCLQISNLS	SSTAEWVLLDQTRNSLFW	276		
Sbjct 181	PQGTLLDKSSRFCQGRPSSC	STRNFWFRPADYNQCLQISNLS	SSTAEWVLLDQTRNSLFW	240		
Query 277	NKTKGANQSQTPCVQVL	AGMTIATSYLGISAVSEFFG	SLTPLFHFHISTCLKTQGAFYI	336		
Sbjct 241	NKTKGANQSQTPCVQVL	AGMTIATSYLGISAVSEFFG	SLTPLFHFHISTCLKTQGAFYI	300		
Query 337	CGQSIHQCLPSNWTGTCT	IGYVTPDIFIAPGNLSLPIPIY	GN SPLPRVRRAIHFIPLLAG	396		
Sbjct 301	CGQSIHQCLPSNWTGTCT	IGYVTPDIFIAPGNLSLPIPIY	GN SPLPRVRRAIHFIPLLAG	360		
Query 397	LGILAGTGTGIAGITKAS	LTYSQLSKEIANNIDTMAKAL	TTMQEQIDS LAAVVLQNRRL	456		
Sbjct 361	LGILAGTGTGIAGITKAS	LTYSQLSKEIANNIDTMAKAL	TTMQEQIDS LAAVVLQNRRL	420		
Query 457	DMLTAAQGGICLALDEKCC	FWNQSGKVQDNIRQLLNQASS	L RERATQGWLNWEGTWKWF	516		
Sbjct 421	DMLTAAQGGICLALDEKCC	FWNQSGKVQDNIRQLLNQASS	L RERATQGWLNWEGTWKWF	480		
Query 517	SWVLPLTGPLVSLLLLLL	FGPCLLNLITQFVSSRLQAIK	LQTNLSAGRHPRNIQESPF	574		
Sbjct 481	SWVLPLTGPLVSLLLLLL	FGPCLLNLITQFVSSRLQAIK	LQTNLSAGRHPRNIQESPF	538		

Moreover, the same S2 region correctly aligns with the endogenous retrovirus *HERV K*, resulting in an almost identical peptide (**Figure 1**) (61).



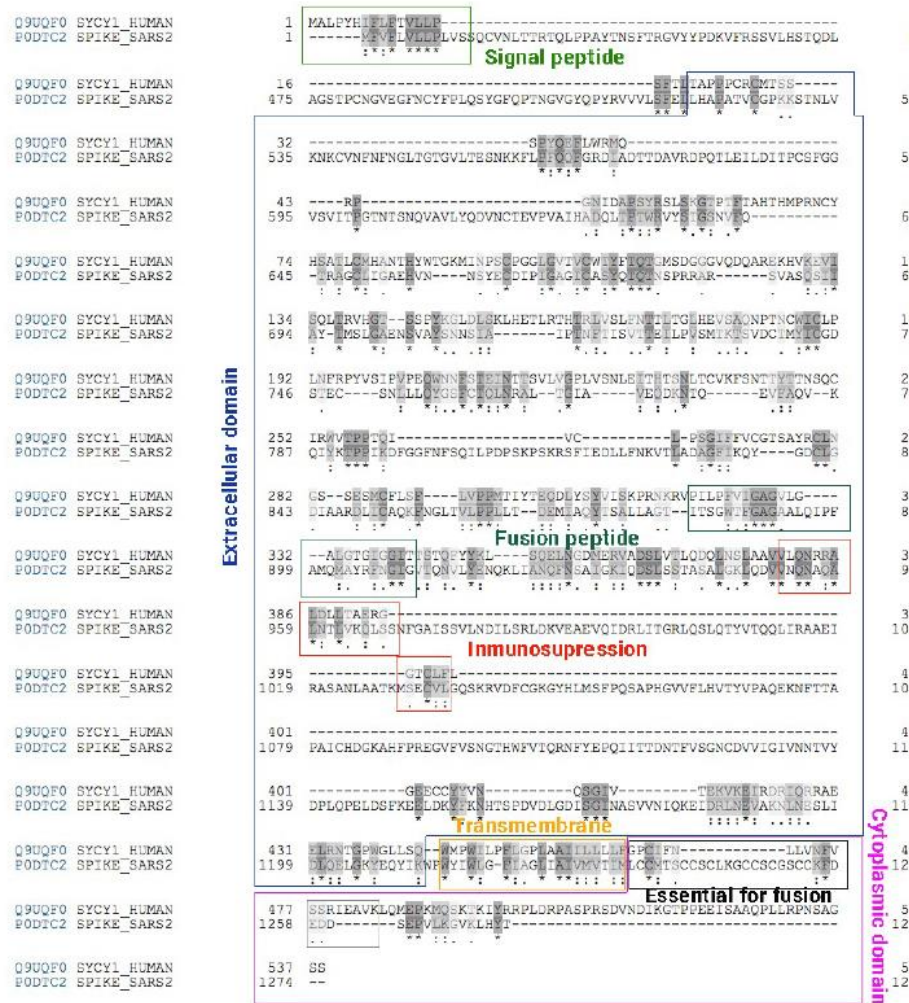
**Figure 1.** Similarity between Wuhan 22 and HERV K 22 peptides.



The Syncytin 2 (chromosome 6) is an immune-system immunosuppressant T-cell dedicated to modulating the maternal immune system during pregnancy (65). Similarly, the S1 region of the spike protein shows homology again with the endogenous retrovirus *HERV W* (61). In this respect, the contribution of Roxana Bruno, Doctor in Biochemistry and Immunology, is of great importance, as she highlighted the striking similarity between the human retroviral proteins and the spike protein S or "spike", as she herself states as follows:

*"The S protein, against which vaccine manufacturers compete to develop a vaccine, bears a high genetic and protein similarity (i.e., it is highly homologous in nucleotide and amino acid sequence) with two human proteins encoded by genes located on chromosomes 7 and 6, Sincitin-1 and Sincitin-2, respectively."* (See Image 4).

Source: <https://cienciaysaludnatural.com/las-vacunas-contr-covid-19-podrian-afectar-la-fertilidad/>



To conclude this section, it is important to emphasise that there are other proteins of vital importance, such as the synaptins (region of the human genome SYN), which are responsible for the transmission of signals between neurons and have the same ACE 2 receptor with which the synthetic SARS-CoV-2 protein interacts. The connection between these important cells of the nervous system could be disrupted, possibly resulting in problems of hypertension and neural degeneration (66, 67).

All this ***necessarily*** raises the following possibilities:

**A.** - When the metabolic proteolysis of the spike protein is forcibly induced by the gene vaccine, several fragments of the protein which are highly homologous to the syncytins, as we have checked, may behave as haptens and develop cross-immunopathology with them. It is therefore a fallacious argument to focus on the percentage of overlap between the SARS-CoV-2 spike protein and the syncytins without taking into account the different metabolic possibilities of both the vaccine RNA sequences and the proteins formed.

**B.**- These peptide similarities between syncytins and the vaccine-induced spike protein trigger autoimmune processes more easily in organisms which have already "seen" HERV proteins, that is, which, due to previous genetic or epigenetic factors, have expressed syncytin homologous proteins. Fundamentally, we are referring to autoimmune pathologies such as Type 1 Diabetes and Multiple Sclerosis.

**C.**- The function of vaccine RNA is to produce spike protein molecules, and there are quite a few studies that show that syncytins are associated with autoimmune disease, such as Multiple Sclerosis, Schizophrenia, and Type 1 Diabetes. The forced accumulation of similar molecules associated with antibodies may produce inflammatory reactions in target organs. To a greater extent, this is what could happen in the testicles and in other organs possessing ACE2 receptors, such as the heart, intestine, kidney and brain – synapsin proteins that are also encoded by HERVs have ACE2 receptors.

**D.**- We do not rule out the possibility that the encapsidated RNA behaves like a synthetic pathogen, i.e. as a "virus-like particle" which produces similar effects to the inflammatory pathology encompassed in the COVID-19 syndrome, since it is a type of M2-TH2 immunopathology, and this artificial virus could mainly affect immune cells (macrophages and lymphocytes).

**E.**-The pathogenic expression of HERV-W proteins has been proven in leukocytes, particularly in CD3 and CD4 lymphocytes, in patients with severe COVID-19, and also shown was the correlation of this expression with immune dysregulation leading towards a strongly inflammatory process with production of cytokines (Interleukin-6, 10 and 17) and chemokines (MCP1 and CXCR1) (69).

**F.**- It has also been proven that type A influenza viruses have caused the pathogenic expression of HERV-W proteins, and we ask whether the synthetic influenza A antigens (particularly H1N1) contained in influenza A vaccines may be related to the increased incidence of severe COVID-19 observed worldwide in people previously vaccinated against influenza (flu) (70).

**G.-** It must not be forgotten that the spike protein, against which these vaccines are intended to produce immunity, has an amino acid sequence similar to the Gp120 peptide of the HIV fusion protein of the Human Immunodeficiency Virus (HIV) (68) and could therefore induce lymphopenia and thus immunosuppression. This was evidenced by the halting of the vaccine project developed by the University of Queensland in Australia and CLS Pharmaceuticals when volunteers showed antibodies to HIV (71).

**H.-** We do not know how long the synthetic spike protein will remain bound to its receptor (ACE2); so, it could become a cellular entry point for new viruses.

**I.-** The fact that the S2 fusion subunit is very similar to endogenous proteins makes it a tolerogenic factor, i.e. a facilitator of entry, for example, of viruses similar to coronaviruses in immune cells, favouring a subsequent ADE syndrome (vaccine enhanced disease).

**J.-** Finally, recent research has shown that the RNA in COVID-19 vaccines may induce the formation of prions with the potential of causing neurodegenerative disease, such as Alzheimer's and ALS (72). The RNA-binding proteins TDP-43 and FUS, their misfolding and subsequent pathological deposition could explain both the loss of function suffered in some neurodegenerative diseases as well as the gain in toxicity observed in ALS (Amyotrophic Lateral Sclerosis).

The results of the mentioned study indicate that the RNA in the Pfizer COVID-19 vaccine has specific sequences that may induce the TDP-43 and FUS to be incorporated into their pathological conformations as prions. In the analysis, sixteen UG tandem repeats ( $\Psi G \Psi G$ ) and additional UG-rich sequences ( $\Psi G$ ) were identified. GG $\Psi$ A sequences were also found. G-quadruplex sequences are possibly present; so, a computer programme is needed to verify them. In addition, the spike protein formed by translation of the RNA vaccine binds to the angiotensin-converting enzyme 2 (ACE2), a zinc-containing enzyme, and this interaction has the potential to increase intracellular zinc. Zinc ions have been shown to cause the transformation of TDP-43 into its pathological prion configuration.

It is known that the folding of TDP-43 and FUS into their pathological conformations as prions causes ALS, anterior temporal lobar degeneration, Alzheimer's disease and other degenerative neurological diseases. The findings reported in the above referenced article, as well as the additional potential risks, lead the author to believe that the conditional approval of RNA-based vaccines for SARS-CoV-2 was premature and that the vaccine may do far more harm than good.

## 5.4 Epigenetics.

Cellular behaviour can be modified without altering DNA. These modifications of cell physiology and structure without changes in the genetic material are studied by the relatively recent field of epigenetics (73). Mendelian genetics could not explain all observations of phenotypic expression and inheritance of traits acquired after parental gametogenesis that do not follow the rules of basic genetics, and this led to the idea that not all information is contained in genes. The main epigenetic mechanisms are DNA methylation, modification of histone proteins and other proteins responsible for regulating the reading and transcription of DNA to mRNA and non-coding RNAs, such as RNAi produced by hybridisation of RNA's with mRNA, as discussed in section 5.1. There is evidence that there are DNA-RNAs interactions which may lead to epigenetic modifications that produce changes in the phenotype but not in the genotype, and these variations may be transmitted via mitosis and meiosis (74), which may lead to disease and to heritable modifications of a Lamarkian type, but not of a Darwinian or neo-Darwinian type. So, this perspective supports the Lamark-Sandin transformation model (75), which explains that there could be DNA-RNA interactions that produce phenotypic modifications without the need to alter the genome. Consequently, what has been said in the media and by governmental and pharmaceutical agents and health authorities – that the mRNA in the vaccine does not alter our genetic code – ***does not imply that unknown phenotypic alterations do not occur***, especially, since the interactions of RNAs and proteins with DNA are not fully understood.

## **6. ANALYSIS OF THE DATA, STATISTICAL PARAMETERS, AND ARGUMENTS USED BY THE HEALTH AUTHORITIES TO JUSTIFY THE RESTRICTIVE MEASURES TAKEN ON THE SPANISH POPULATION SINCE MARCH 2020 TO DATE.**

### **6.1 Confinements**

While making a compilation of what has happened with this pandemic since it began in March 2020 up to the current date, we discuss what has been done in the past and what is currently being done in the present. If we refer to the past, the first thing to say is that the option of strict confinement was not the solution and rather generated most of the problems. It did not serve to reduce the number of deaths, reaching figures in Spain (148 per 100,000 inhabitants) and Euskadi (174 per 100,000 inhabitants) much higher than in countries such as Sweden (127 per 100,000 inhabitants), in which no confinement was carried out; so, if we compare with Spain, Sweden would have had 16.75% of deaths, and if we compare with the Basque Country, 37.43%. (76 to 80).

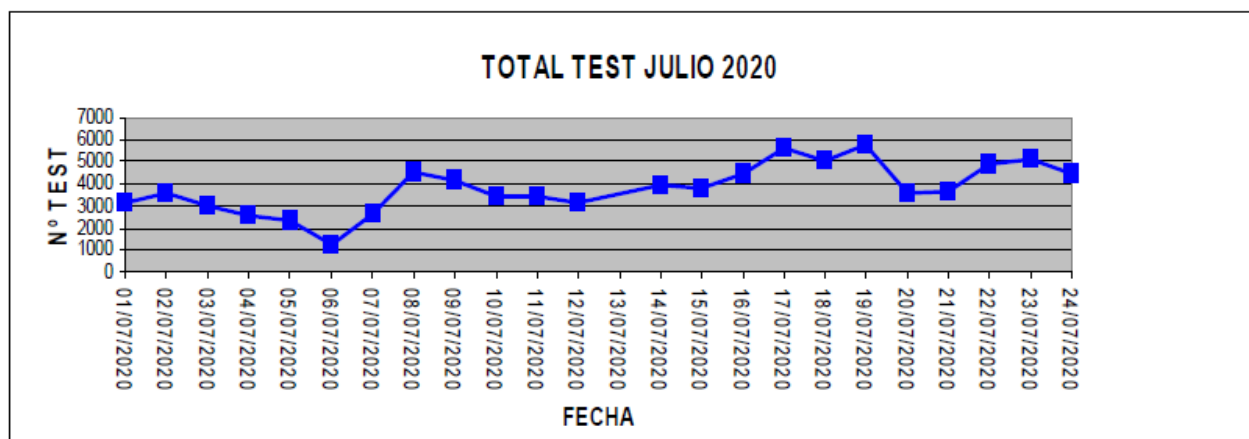
### **6.2 PCR tests and cumulative incidence as devices for restricting fundamental freedoms.**

The methodology used by the health administration takes the 14-day AI (cumulative incidence of cases per 100,000 inhabitants at 14 days) as the basic parameter to establish restrictive measures, for which the sole basis is positive test results, varying the number of tests at their convenience. Hence, by carrying out a number of tests in a large percentage who have no symptoms of illness, the health administration knows that the more tests the more positives, so the AI will be higher. The AI is the sum of the absolute number of positives in the last 14 days, with a reference value of 500 cases per 100,000 inhabitants.

Next, we need to expose PCR testing using the concept of “PCR-asymptomatic” as a device for adopting measures that restrict individual freedoms, as well as for bringing sectors of the productive and labour force into an alarming economic crisis. PCR testing, as already detailed, is a methodology with many uncertainties, and its results can be easily manipulated depending on the number of cycles performed, while at no time does it indicate the viability of the virus, i.e. whether it is infectious or not, as long as such a result is not verified with a viral culture. How many verifications with cell cultures have been carried out since March following a meticulous procedure of sample purification?

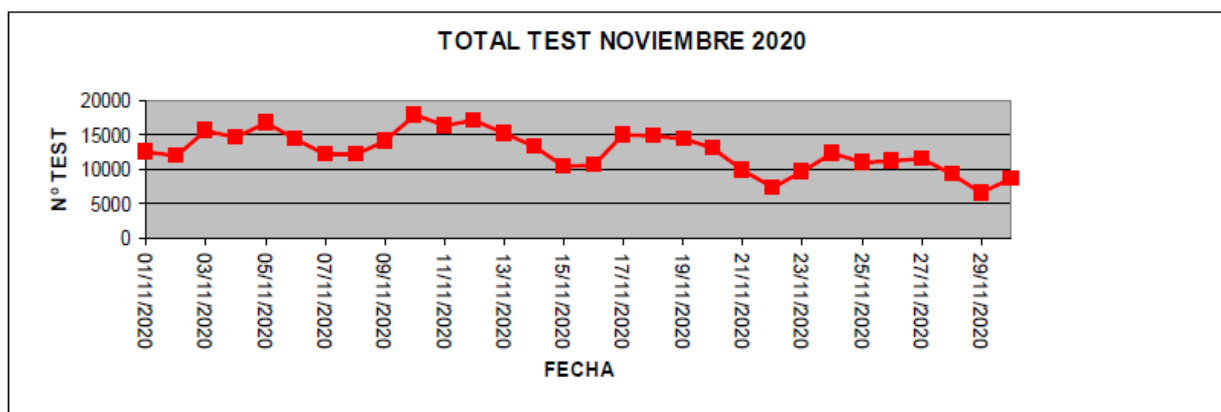
These tests do not detect infections, because in order to have an infection you have to have symptoms in the first place; so, in fact, one could say that the pandemic is not a pandemic of infection but a pandemic of positive test results and TV news.

In fact, measures based on this method are being taken because the parameters that are used are based on positive test results, varying at the convenience of the health administration. Furthermore, the more tests that are carried out the more asymptomatic positives, which raise the AI (cumulative incidence) to 14 days, using 500 cases per 100,000 inhabitants as a reference (**Graph 1, 2 and 3**) (76 to 80).



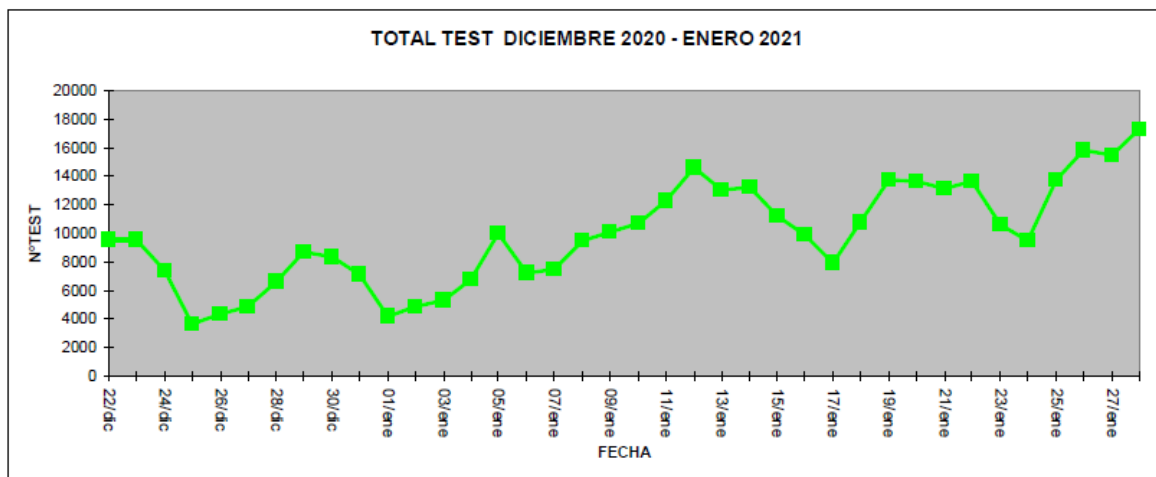
**Graph 1.** If we look at the evolution of the number of tests carried out since the return to the new normality at the end of May 2020, it can be seen that the number of tests carried out before the elections of July 12 were significantly lower than those carried out in the post-election period. The graph shows that in the first 12 days of July, 37,024 tests were carried out, while the following 12 days of July, 54,407 tests were carried out, i.e. 46.95% more, remembering that just after the elections more restrictive measures such as the obligation to wear masks were taken.

On the one hand, the percentage of positives in relation to the number of tests carried out is taken into account, and this percentage has continuously remained between 5 and 10%. This means that there was a second wave, and now the third wave is coming (76 to 80). First of all, it must be said that in order to see an evolution of this percentage of positives, the number of tests to be carried out would always have to be the same, and on the other hand, if there were really new waves, this percentage would rise exponentially and would far exceed the values that have been obtained since May.



**Graph 2.** Similarly, in the month of November, there was a decrease in the total number of tests carried out, from an average of 14,264 in the first 15 days of November to 11,011 in the last 15 days of that month, i.e. 22.81%, coinciding with a decrease of 22.81% and with the restriction measures taken during the same month.

This use of **testing on convenience** has been carried out whenever restrictive decisions have been taken. If we take into account the number of tests made to date since December 22, 2020, which is the date the measures were taken for the Christmas period, with new measures taken since January 12, we observe again, as occurred repeatedly in the past, that the average number of tests that were made since December 22, 2020, to 6 January during the Christmas period was 6.776, but since that date and in the following 15 days the average number of tests that were made was 11,562; i.e., 71% more than during the Christmas period (**Graph 3**).



**Graph 3.** Number of PCR tests performed between December and January 2020-21.

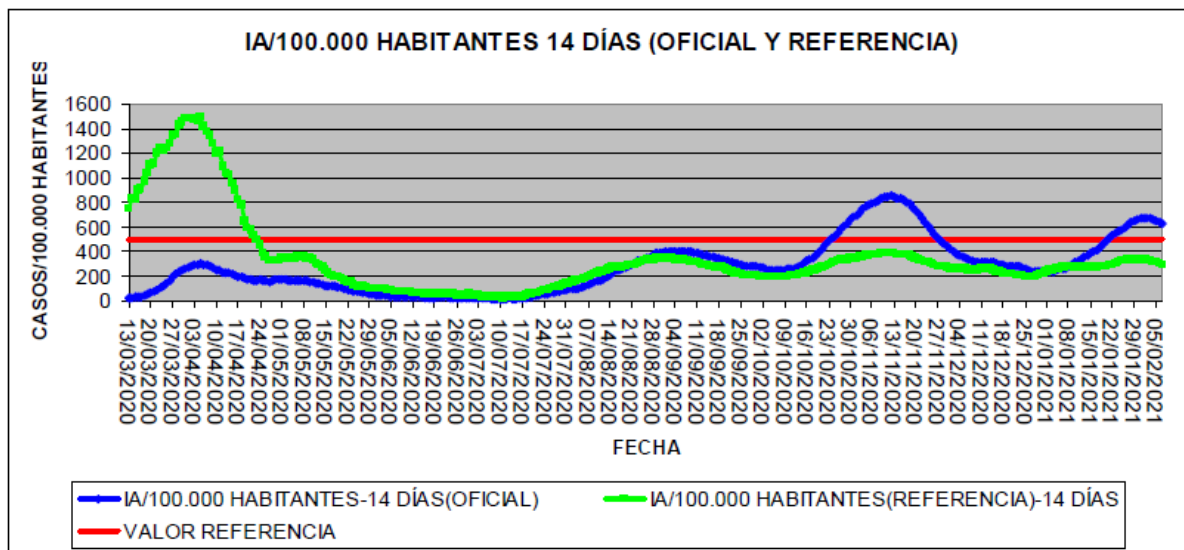
It is curious that the WHO standards are dictated in some cases and disregarded in others, such as in the case when considering if a person is ill, which is the first thing to consider. In the face of possible false positives due to the number of cycles performed, **what must be taken into account are the clinical observations**, the patient's history, and epidemiological information. And, in any case, the Ct value (number of cycles performed in each PCR test) should be indicated in the report sent to the patient. Are the cycle values reported in the analysis bulletins of the PCR tests?

What is the reason to continue with this non-sense of using such tests as an essential tool for taking measures when this has been called into question by the WHO itself? (81)



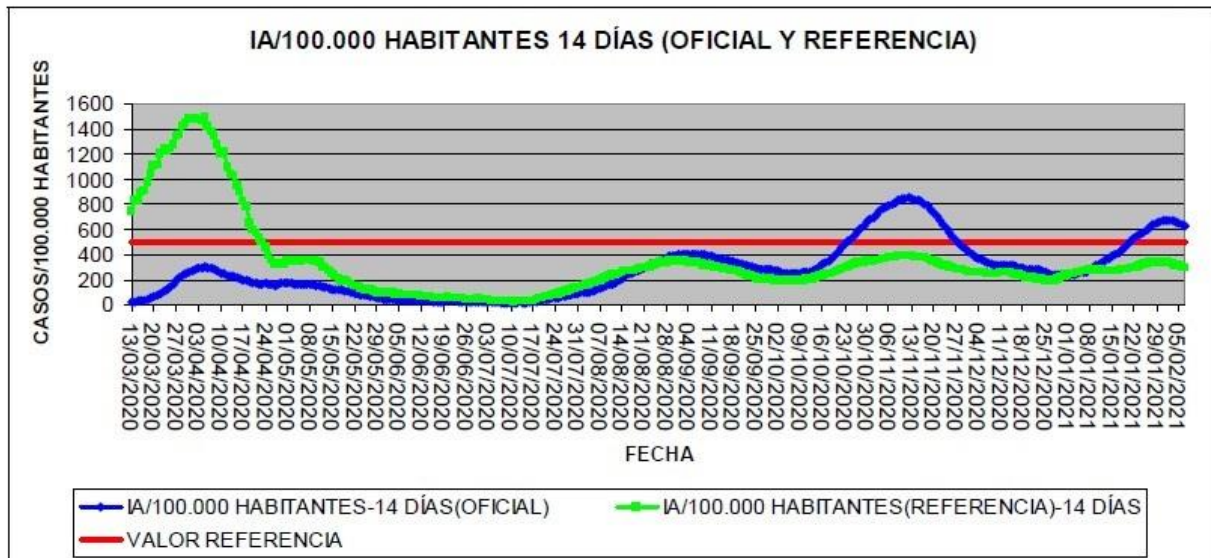
With regard to the AI (cumulative incidence) at 14 days, with data from the Basque Country but which can be perfectly extrapolated to other Autonomous Communities, it must be said that its calculation from the point of view of epidemiological statistics is not ideal, being overestimated, for the following reasons:

- The calculation is based on the absolute value of PCR positives, and as we have seen, it is subject to the dictates of the health administration.
- Given that quarantines have been in place for months now and have gone from 14 days to 10 days due to considering the contagion period to be between 7 and 10 days, it would be more logical to use AI (cumulative incidence) at 10 days instead of 14 days as has been used to date (**Graph 4**) (76, 82).



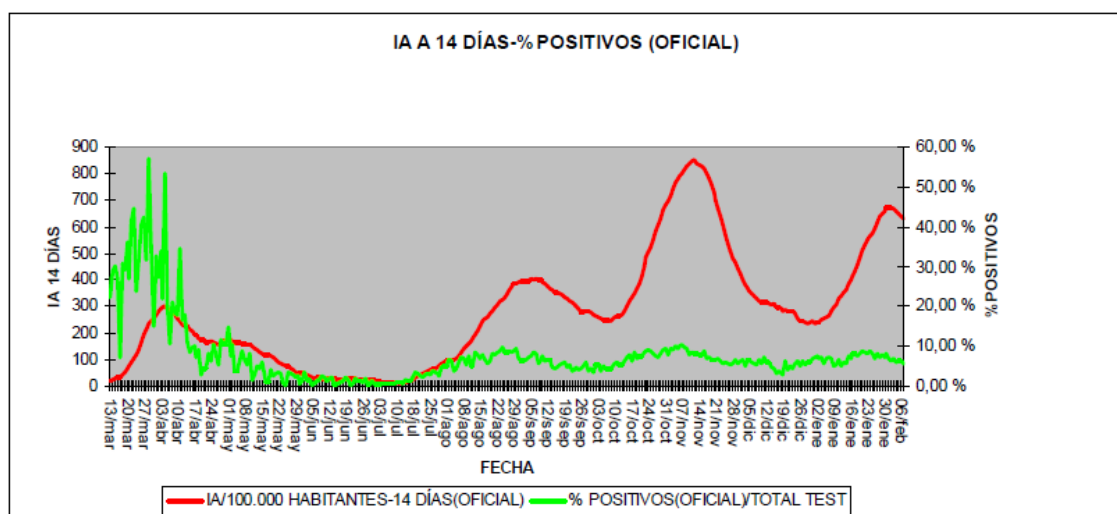
**Graph 4.** In the graph of the cumulative incidence of cases per 100,000 inhabitants in 14 days and 10 days, in the case of the AI after 14 days – in the period of March-April 2020 when the exponential growth of infections occurred – we see that the 14-day curve used from October 25 to November 28 is above 500 cases per 100,000 inhabitants, showing that already by November 13 and 14 it started to decrease, i.e. just when the last severe restrictions were put in place. However, in the case of the AI after 10 days, we see that November 3<sup>rd</sup> would be the date when the established threshold was exceeded, and on November 20<sup>th</sup> it would be below the value of 500, noting that this excess occurs only slightly in that period of time, which is to say that with these data it is not possible to ensure that the drop that occurs in neither of the two cases (14 and 10 days) is due to the measures taken. So, neither using the 14-day nor the 10-day curve indicates that measures should have been taken for the rest of the year and until the current date.

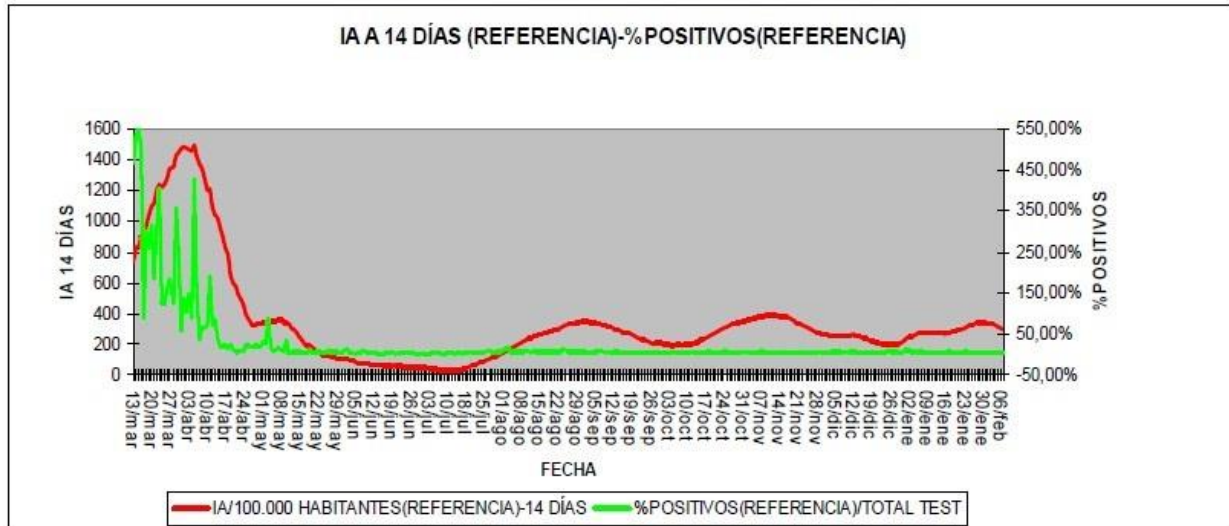
Logically, there is a good correlation between positives and number of tests and AI, but it is not significant in relation to the percentage of positives, nor to ICU occupancy (**Graph 5**) (76).



**Graph 5.** If we correlate the AI at 14 days of the official calculation with the number of positives obtained, we see that the correlation is very high ( $r = 0.90$ ), which confirms that AI as the sum of positives in 14-day is nothing more than using the number of positives, although it is not related to the percentage of positives.

From an epidemiological point of view, it would be much more realistic to perform the calculation of the AI, calculated with a normalised value, by taking into account the same number of daily tests, such as the average number of tests performed during the pandemic. If we calculate the AI on the basis of this normalised value, we would obtain a good correlation not only with the percentage of positives (**Graph 6 and 7**) but also with the R0 index (a reproduction number used to describe the intensity of an infectious disease) (**Graph 6**) (82), with the occupancy rate of ICUs, and also with the number of positives and deaths (76).





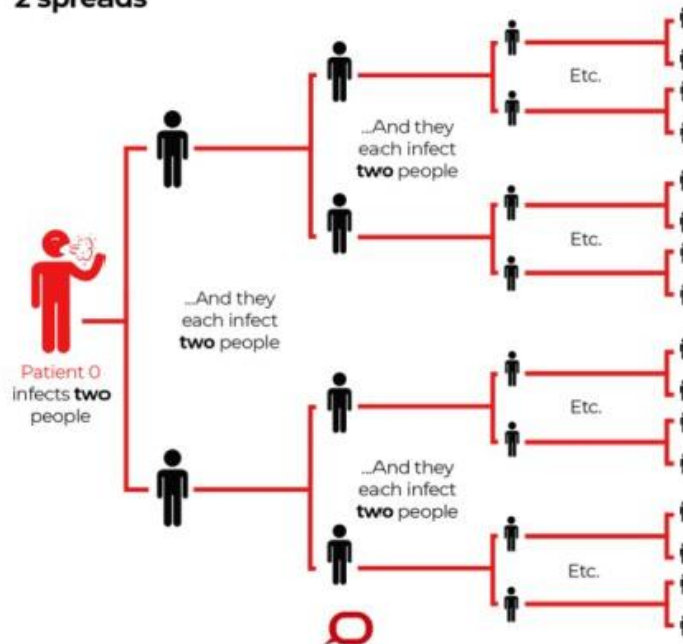
**Graph 6 and 7.** If we perform these calculations with the 14-day AI normalised to a fixed number of tests, it is clear that this AI much better corresponds to the percentage of positives in March-April; that is, the AI could have been useful as an index for making decisions, just as it could have served for not making any, especially, at the level of restriction, from May to the current date, and this has also been confirmed by the percentage of positives presenting values continuously below 10%.

A standardised 14-day AI calculation (referenced to a fixed test value) would have greater credibility, as it correlates clearly with the percentage of positives, so that the 14-day AI values above 500 are presented in the March-April period when the percentage of positives exceeded the value of 50%, without having exceeded this threshold since May and showing a clear correlation with the percentages that have been present for months, which do not practically exceed the value of 10%.

In light of this, we should ask ourselves the following question:

- What is the reason for not calculating the AI at 14 days by taking into account a reference value in order to establish a homogenisation of the data so it is truly comparable?

### How a virus with a reproduction number ( $R_0$ ) of 2 spreads



**Figure 2.**  $R_0$  is the number of people each infected individual will infect in turn. The statistic is a range because it depends on a variety of factors that change over time. Each disease has its own  $R_0$ . This parameter at present has a mean value of 1.01 with a maximum of 1.27, i.e. with values typical of influenza epidemics usually ranging between 1.2 and 1.5.

### 6.3 Facemasks.

As part of the measures already imposed in previous months, masks are another tool being used. However, based on the evidence presented in this report on the missing isolation of the virus and on the absence of the SARS virus ACE 2 receptors in the lungs, this imposition is obviously being used as a social engineering device, more than as a health providing tool, which generates mistrust and lack of empathy in people, as well as multiple pathologies. The World Health Organisation (WHO) had previously carried out studies on the flu virus and deduced that the masks did not prevent contagion. Quoting from these studies (84):

*"In the community setting, however, the usefulness of the use of facemasks has not been established with certainty, especially in open spaces, as opposed to indoors and in situations of proximity to people with influenza-like symptoms. [...] Incorrect use of a facemask may increase the risk of transmission, rather than reduce it".*

- So, what is the rationale for the continued imposition of facemasks and of the coercive methods being used?
- How is it possible that the education sector has not only failed to raise its voice but it has also been very harsh when it comes to imposing facemasks and social distancing on children? How is it possible that it has not stood up against the health administration to demand that school sports be practised?

On the basis of the preliminary studies that are being carried out on the school-age population, such as the one carried out on 25,930 German children (85), we can draw valid conclusions about the health of our children and the use of facemasks, concluding that this measure is the cause of the following symptoms observed by the parents of the participants in this study: **headaches, concentration and learning difficulties, increased sleepiness, sadness, choking sensation, dizziness, dryness of the upper respiratory tract, syncope, decreased mobility and playfulness, nausea, itchy nose, abdominal pain, rapid breathing, feelings of tiredness, itchy eyes, loss of appetite, tachycardia, hearing problems, loss of consciousness, and vomiting.** These are empirical reasons which make it an absolute aberration to have the use of facemasks be a compulsory requirement for minors.

#### 6.4 The term *asymptomatic*.

We should also mention another device used in this pandemic, the term *asymptomatic*. As in the case of the PCR test, again the WHO guidelines are not followed in this regard though they do confirm that it is rare for an asymptomatic person to transmit the virus to another person. This is specified in the section "Question & Answers" on the coronavirus COVID-19 disease, where a distinction is made between pre-symptomatic and asymptomatic patients, which specifies that: *"Even if they never have symptoms, some people can transmit the virus to others; it is not yet clear how often this happens, and further research is needed"* (86). This statement could be further qualified by the fact that ***there is no conclusive evidence of transmission from people who have no symptoms.***

Of note in this regard is a study conducted in Wuhan following the strict containment that took place from January 23<sup>rd</sup> to April 8<sup>th</sup> 2020, after PCR testing of the entire population over 6 years of age (a total of 9,899,828 people or 92.9% of the total population of the city), which concluded that:

*"Virus cultures were negative for all positive and retested positive asymptomatic cases, indicating that there is no "viable virus" in the positive cases detected in this study" (87).*

Therefore, the use of the term “PCR-asymptomatic” is a scientific error that has been made since the beginning of the pandemic to the present time, with corresponding consequences in terms of both curtailment of individual rights and socioeconomic cutbacks, in short paralysing society without taking the consequences into account. To date, the calculated use of the number of PCR tests that have been carried out continues, as was the case in July with the elections. The number of PCR tests were increased after the elections in order to impose restrictive measures, such as the mandatory mask requirement, as occurred in November 2020 when fewer tests were made in the second half of the year than in the first half in order to loosen up the measures for the Christmas period, but it is more than likely that at that time and during the flu season they will continue to use this device as they see fit (88). If we go by what is being reported by the health administration, both on the disappearance of influenza and on the COVID-19 vaccine, a comment could be made again on how the health administration, medical collegiate organisations, politicians and governments provide information which, as in the past, only serves to inculcate fear in people, which still prevails, while they try to justify the measures by alleging a lack of discipline in citizens in order to cover up for what is really happening in the healthcare and education systems and also for the lack of human and material resources that we have.

#### **6.5 The mysterious disappearance of influenza or its new name: COVID-19.**

If we consider the information we are being given about influenza, that it has practically disappeared, and if we again go back to the WHO's recent statement that this virus can be endemic like influenza (89, 90), is it really the case, as we were told, that this year had practically no influenza specifically because of the measures taken for the COVID-19, or is it that what is really being detected is simply the current seasonal influenza? We are being told that it is precisely the use of facemasks that which has caused the flu to practically disappear (89), and so once again we should ask ourselves a series of questions:

- How is it possible we are told the masks worked for the flu virus and not for the intended purpose of preventing the spread of COVID-19, when they are similar viruses and are transmitted mainly by human-to-human contact?
- How is it possible that most facemasks used, i.e. medical facemasks, by information stated on their commercial labels, are known to be valid tools for bacterial filtration > 98% but not for viruses which are considerably smaller in size than bacteria?
- How is it possible that this claim be made when the WHO, at the time, with studies carried out on the influenza virus (flu), concluded that facemasks did not prevent transmission? (86).



If we make a comparison between the Cumulative Incidence (IA proportion of people who become ill in a given period of time) at 14 days and at 10 days for both COVID-19 and influenza (74, 89, 91), we see that for influenza – with data from 2018-2019 from week 48, November 2018, until week 9, March 2019 – the rate of cases per 100,000 inhabitants exceeds with higher values than that for COVID-19 if we count these three months from October to December, reaching average values of 455 and a maximum of 853 cases per 100,000 inhabitants for COVID-19, whereas for influenza the average values are 664 with a maximum of 1765 cases per 100,000 inhabitants, which is to say that ***the cumulative index is higher for influenza than for COVID-19***, and yet no action is taken for influenza, even though there is a health breakdown in most years (89). If we consider that this data is being used for taking the restrictive measures that are being imposed on us, a question arises:

- If this ratio had been used in seasonal flu campaigns, should we not have been confined and our freedoms restricted?

If we look closely at the measures taken for the COVID-19 pandemic, which affect the population based on these rates of cases per 100,000 inhabitants, and if we compare them with our unrestricted life during the flu season of every year, a number of inferences may be drawn leading us to the conclusion that ***according to the following points there is no justification whatsoever for the measures taken:***

- An extraordinarily high number of COVID-19 tests have been carried out in comparison with the number of tests made in the flu season, and due to the uncertainties of this test, the number of positives was higher than the real number, assigning cases to asymptomatic people and even using the number of positives as if each positive corresponded to a person, when there are people who have undergone more than one test. Why is a different person counted in the calculation of the cumulative incidence?
- COVID-19 tests are based on the RT-PCR technique, which does not insure that a positive test actually indicates the presence of the disease, and as we know there is a significant percentage of asymptomatic false positives who are not contagious, since they are neither sick nor has their immune system overcome the virus, and with their viral load being minimal they are therefore incapable of infecting.
- Influenza tests are carried out using microbiological cell cultures with strains already identified in previous years and therefore with an infinitely greater reliability than that of the tests used for COVID-19, for which cell cultures are not performed, and if they are, no viable viral particles are detected.
- If the reason for implementing the measures was that influenza has a vaccine and COVID-19 did not, though now it seemingly does, this reason has very little weight, since it is known with certainty, first, that the flu vaccine is only 50% effective and, second, that the vaccination rate in the population is quite low, except in the over-65 age group (89).



What is the rationale for continuing to introduce such restrictive measures if the COVID-19 vaccine is already available? Do the health authorities even themselves not believe in the effectiveness of the COVID-19 vaccine? Are they not fully convinced that the vaccine, being of a new design and having received so little investigation and safety testing, will not produce more problems than benefits?

In this regard, it is worth noting that there are currently no specific studies on immunity generated by the experimental vaccines that are being administered, in spite of the fact that a large number of general adverse effects have been reported in the nervous and other systems, such as the gastrointestinal, musculo-skeletal, dermatological, respiratory, cardiac, vascular, immunological, microbial, psychiatric, ocular and haematological systems (92).

In influenza seasons, the highest number of cases is not detected by the tests that are carried out; it is detected however through the filter of primary care by applying the usual medical care to patients, whereas in COVID-19 this essential first filter in healthcare, which is primary care, has been reduced or even cancelled out (89).

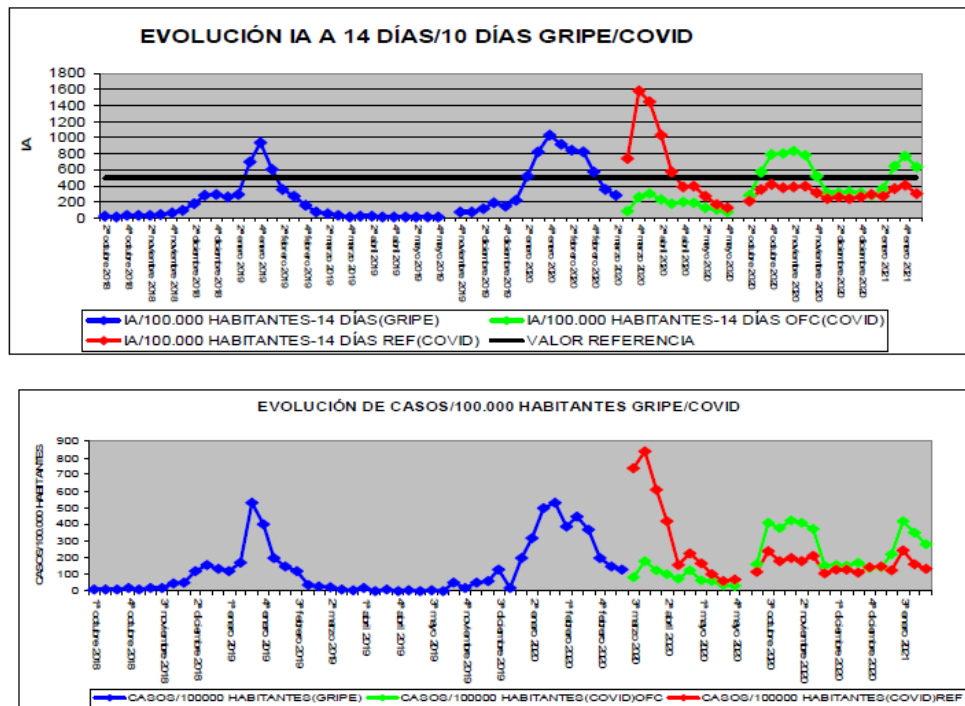
Wouldn't a better health management have been achieved if the usual protocols had been used as they are used for influenza? Wouldn't a significant part of hospital collapse have been avoided? Would we not have formed a much truer picture of the infection if all those RT-PCR tests had been replaced by more diagnosis in primary care?

If non-diseased asymptomatic positives had been removed from the equation, since asymptomatic patients would usually not attend primary care services, would the cumulative incidence not have reached the values reported? Would this not have provided us with a better picture of the scale of the epidemic, thus avoiding the need for measures, which, apart from terrifying the population and bringing about many misgivings, have generated suspicion in the population by penalising it?

- If we compare with the seasonal flu – for example, in the Basque Country, where there are around 3,005 deaths due to resistant pneumonia (79), although only a small number is attributed to the flu in people who have been tested for influenza – we can see that the case fatality rates for influenza and for COVID-19 are in the order of 5.26% for influenza and 2.38% for COVID-19 (76 to 80, 93, 94). If we count the number of positive cases for COVID-19, it is about half as many because, although each case has been counted as a different person, the reality is that more than one test has been performed in many cases, and so with this correction, the case fatality rate for COVID-19 would be 4.77%.

- If we compare mortality between influenza and COVID-19, we see that the percentages are similar if deaths from resistant pneumonia are taken into account as deaths from influenza, as was done with COVID-19 this year, in the order of 0.14% to 0.17%, taking into account that COVID 19 deaths have been overestimated.

**Therefore, both the case fatality and mortality rates are the same for influenza and COVID-19, and this explains why we are told that influenza has been virtually eradicated,** an expression which, if we look at the data, should rather mean that **influenza has been replaced or renamed COVID-19** (Graph 8 and 9).



**Graphs 8 and 9.** In this two graphs, we see that the flu ends dramatically in the second week of March 2020. From that moment on, there is a large peak in COVID-19, a disease which has similar symptoms to influenza but has some differences that make it necessary, at the very least, to reflect on the different hypotheses about what could have occurred for this peak to take place. Subsequently, the COVID-19 graph indicates that in order to draw conclusions it would be necessary to test the COVID-19 positives so as to discern whether the flu has really disappeared. In all likelihood, the COVID-19 cases will most likely be overestimated because of the significant number of positives without symptoms.

- So, since we are told that the flu has disappeared, what is the reason for not verifying that normal influenza tests are not carried out on people with symptoms and positive PCR tests for COVID-19?

If we compare the seasonal influenza of the last two years with COVID-19, we see that during 2018-2019 there was a seasonal peak of influenza, which occurred again in the 2019-2020 season with the sudden disappearance of influenza in the third week of March to be seen again in the March-April peak of COVID-19 (89, 94).

Afterwards, in the 2020-2021 seasonal period of influenza, there is an absence of influenza and a limited presence of COVID-19, which peaks because of the way in which we are monitoring it and taking action, and not as a seasonal epidemic as such (89, 94).

- What is the reason influenza is not controlled in this seasonal period using the tools that have always been used in healthcare, such as primary care and influenza tests?

## 6. 7 Statistical explanations for mortality attributed to COVID-19.

In most countries, it seems to be an established yet unexplained practice, at least on strictly scientific or medical reasons, to include patients with all kinds of clinical conditions or comorbidities in the lists of deaths from COVID-19 just because they are PCR-positive. It is necessary to contrast the official data on COVID-19 mortality with objective data on all-cause mortality. The most complete data is found in the 22 states participating in the EuroMoMo.eu. The information provided by these countries is particularly interesting, especially, because several of them are among the countries most affected by COVID-19 in the world, according to official data.

First of all, we note that during the so-called "first wave" in March-April 2020, only 6 countries show an extraordinary excess mortality: Spain, England, Belgium, France, the Netherlands and Italy. All of them, with the exception of the Netherlands, were under severe containment when it happened, and as of June 22<sup>nd</sup> they were among the 10 countries most affected by COVID-19 in the world in terms of cumulative mortality per 100,000 inhabitants (**Table 4**). In fact, if we exclude the microstates of San Marino and Andorra, which were at that time ranked first and third, respectively, and which do not participate in the EuroMoMoMo, the Netherlands would also enter the group of the 10 most affected in the first wave.

Most of the EuroMoMoMo countries either did not experience substantial excess mortality, or if they did, they had periods of excess mortality of shorter duration, intensity and importance than others experienced by European countries in recent years, which did not raise alarms or impose social, cultural, economic or mobility restrictions on the population. The excess mortality peaks in Spain and Portugal during the winter of 2017 were higher than those of most countries in Europe during the first COVID-19 wave. This helps us to size up the overall situation.

Ireland and Sweden, which ranked 10th and 6th respectively in the world ranking for COVID mortality at the end of the first wave, did not even reach a mortality peak of an intensity comparable to that in Spain in the winter of 2017 (80).

#### TOP 10 PAÍSES CON MAYOR MORTALIDAD POR COVID19 DEL MUNDO

	Muertes covid/100.000 habitantes a 22 de junio de 2020
1. San Marino	125,16
2. Bélgica	84,32
3. Andorra	67,57
4. Reino Unido	63,97
5. España	61,04
6. Italia	58,37
7. Suecia	50,62
8. Francia	45,43
9. EEUU	37,33
10. Irlanda	35,70

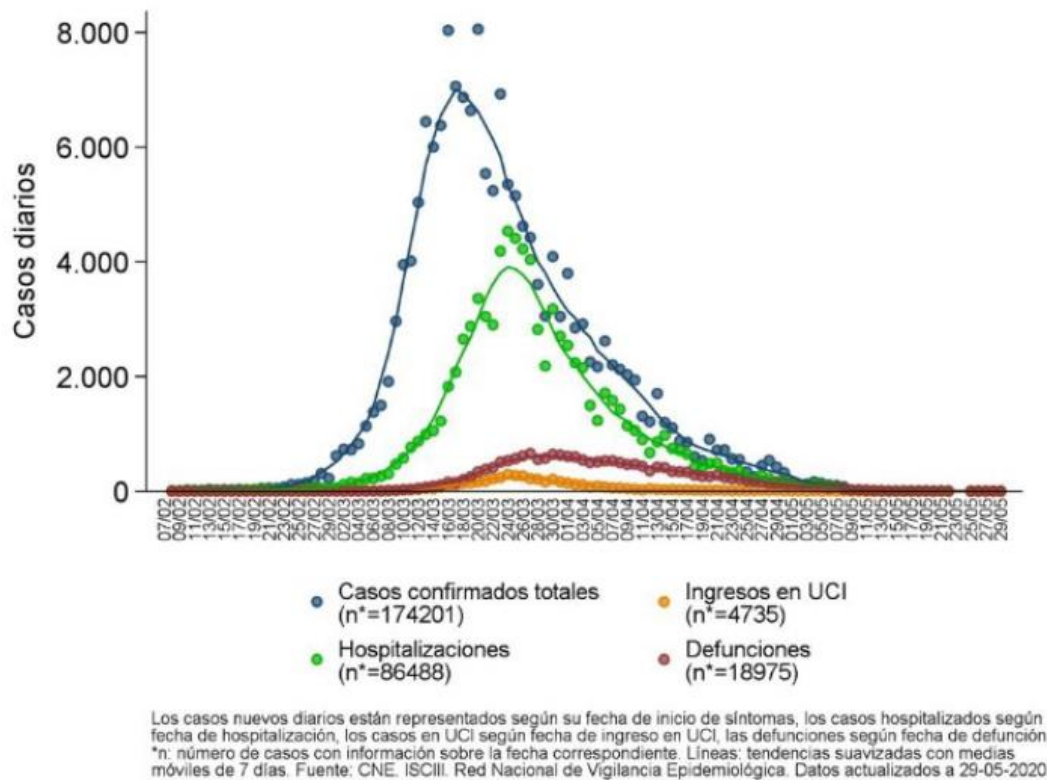
Estos 10 países acumulan cerca de dos tercios de las muertes registradas mundialmente por Covid-19

##### Algunas características comunes:

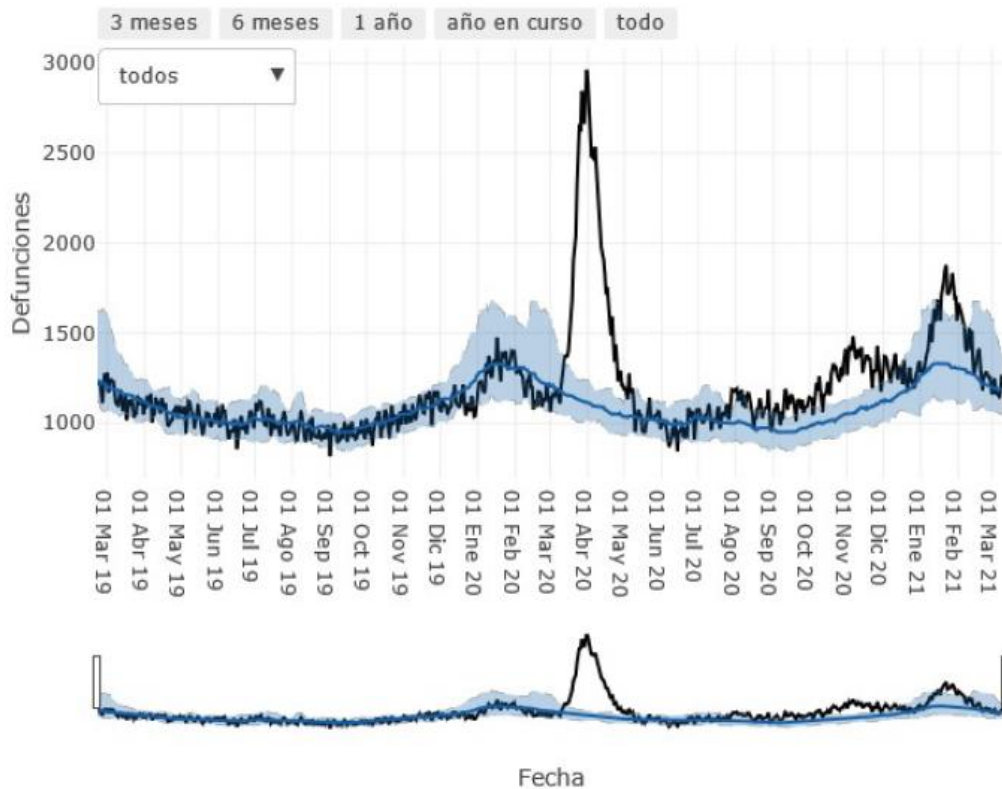
- Pertenecen al 20% más rico del planeta. Elevada cobertura tecnológica y radiación electromagnética.
- Población envejecida por baja natalidad. Elevado número de residencias de ancianos.
- Amplia cobertura sanitaria. Elevado número de hospitales e infraestructuras sanitarias, elevada cobertura vacunal.

**Table 4.** Shown are the 10 countries that account for two thirds of the world's COVID-19 deaths. These countries belong to the 20% richest countries on the planet, with high technological coverage and electromagnetic radiation, an ageing population, low birth rate, a high number of elderly homes, extensive healthcare coverage, a high number of hospitals and health infrastructures and high vaccination coverage.

From the evolution of deaths in the 2010-2020 period by week, we see that the curves are practically similar except for 2020 in weeks 11 to 18, i.e. in March and April, where there is a differentiated peak compared to the rest of the years studied (**Graph 10** and **11**) (76, 80, 94).



**Graph 10.** Epidemic curve of COVID-19 cases according to severity. COVID-19 Report No. 33. May 29, 2020.



**Graph 11.** Deaths from all causes in Spain, from Dec. 28, 2020, to Feb. 13, 2021.  
 MoMo ([https://momo.isciii.es/public/momo/dashboard/momo\\_dashboard.html](https://momo.isciii.es/public/momo/dashboard/momo_dashboard.html)).

In Spain, the number of deaths in 2020 was 463,807 with a mortality rate per 1,000 inhabitants of 9.92, of which 69,142 deaths are assigned to COVID-19, representing a mortality rate per 1,000 inhabitants of 1.48 of the total for 2020. That is, without COVID-19, the rate would have been 8.44, considerably lower than the trend observed in recent years (76, 77).

- If we compare these figures with the average mortality rate per 1,000 inhabitants over the last five years, we see that this would be 9.13; so, taking into account that the rate for 2020 is 9.92, ***the differential that could be awarded to COVID-19 would be 0.79, which would mean 32,217 fewer deaths attributed to this disease.***

- Using the same exercise with the data for the Basque Country, we see that in 2020 the number of deaths is 24,386 with a mortality rate per 1,000 inhabitants of 11.19, of which 3,794 deaths are assigned to COVID-19, which represents a mortality rate per 1,000 inhabitants of 1.74 of the 2020 total. That is, without COVID-19, the rate would have been 9.45, considerably lower than the trend observed in the previous years (78, 79).

- If we compare these figures with the average mortality rate per 1,000 inhabitants over the last five years, we see that it would be 10.14; so, taking into account that the 2020 rate is 11.19, the differential that could be awarded to COVID-19 would be 1.05, which would mean 1,505 fewer deaths attributed to this disease (78, 79).

Having established doubts concerning what is happening with the 2020-2021 seasonal flu, where cases are being counted as COVID-19, which, due to their symptomatology and treatment, are typical of influenza, the peak caused in March-April, just when the 2019-2020 flu season ends, remains to be explained.

This unusual peak in hospitalisations and deaths in Spain, as well as in Europe and the rest of the world, may be explained by analysing the following statistical data on ***influenza vaccines, applied hospital protocols, and 5G electromagnetic networks.***

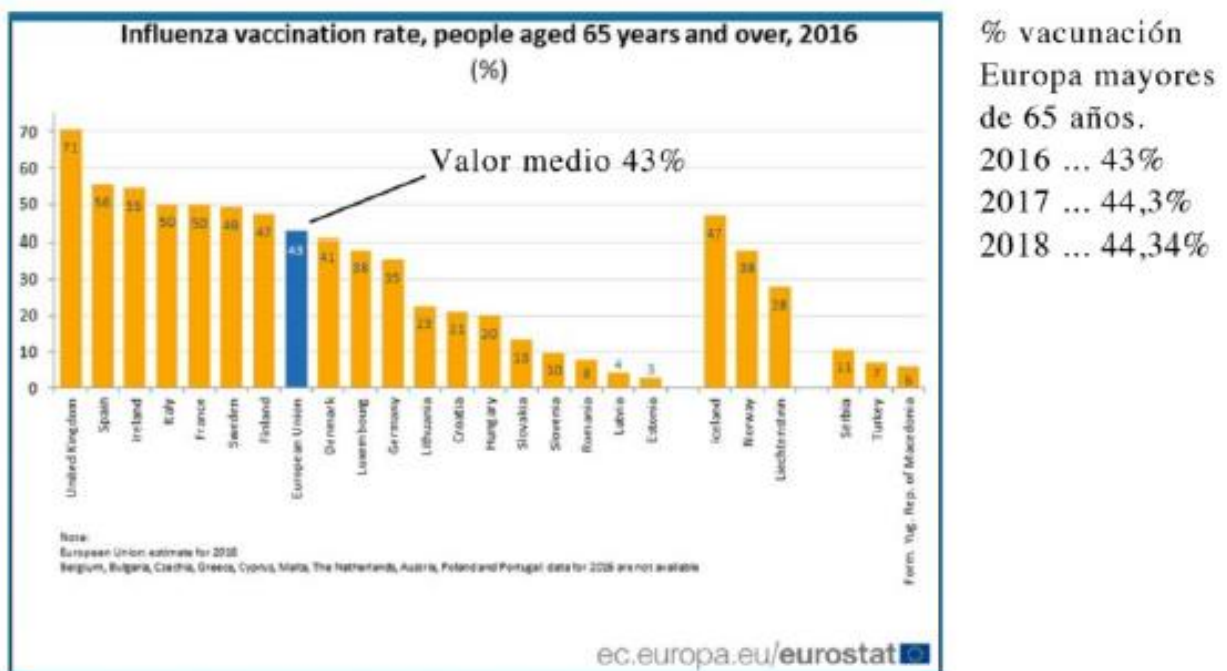
***The disease called COVID-19*** was initially described as a bilateral interstitial pneumonia (25). Then, deaths due to an hyperacute inflammatory syndrome or "cytokine storm" were diagnosed, and finally, when a group of Italian pathologists began performing autopsies in defiance of WHO guidelines, we learned that the endothelium of various blood vessels, including arteries, were damaged and thrombosed, while NETs (neutrophil chromatin traps or networks of apoptotic neutrophils) were accumulating in them. Doctor Schmied of the University of Ulm, using photos of bronchial lavage from patients diagnosed with COVID-19 and then culturing the cells, found some viral particles only in immunocompromised patients with pulmonary symptomatology, of which the least abundant were coronaviruses.



Above all, he found *Staphylococci*, *Streptococci*, *Adenoviruses* and, surprisingly, very frequently, *Borrelia*, which is indeed known to occur in immunosuppression. We also know that the bacterium *Prevotella* spp. (of the oral flora) has been frequently associated with severe COVID-19 (95).

**Indeed, in COVID-19, there are clearly differentiated symptoms characteristic of a reaction to external agents, such as toxicants, pesticides or vaccines and their adjuvants, such as polysorbate-80 (96), a substance which may cause an autoimmune syndrome due to the fact that it enters the body through the blood and not through the mucous membranes, as a respiratory virus would.**

On the basis of this statement, a relationship may be established between mortality rate per 100,000 inhabitants and the flu vaccination rates in people over 65 years of age, in order to establish a reasonable doubt as to whether the influenza vaccination of 2019-2020 has been more harmful than beneficial (97, 98). In Europe, countries with the highest influenza vaccination rates have about 6.4 times higher rates of COVID-19 mortality rates than the least vaccinated (**Graph 12**) (99, 100).

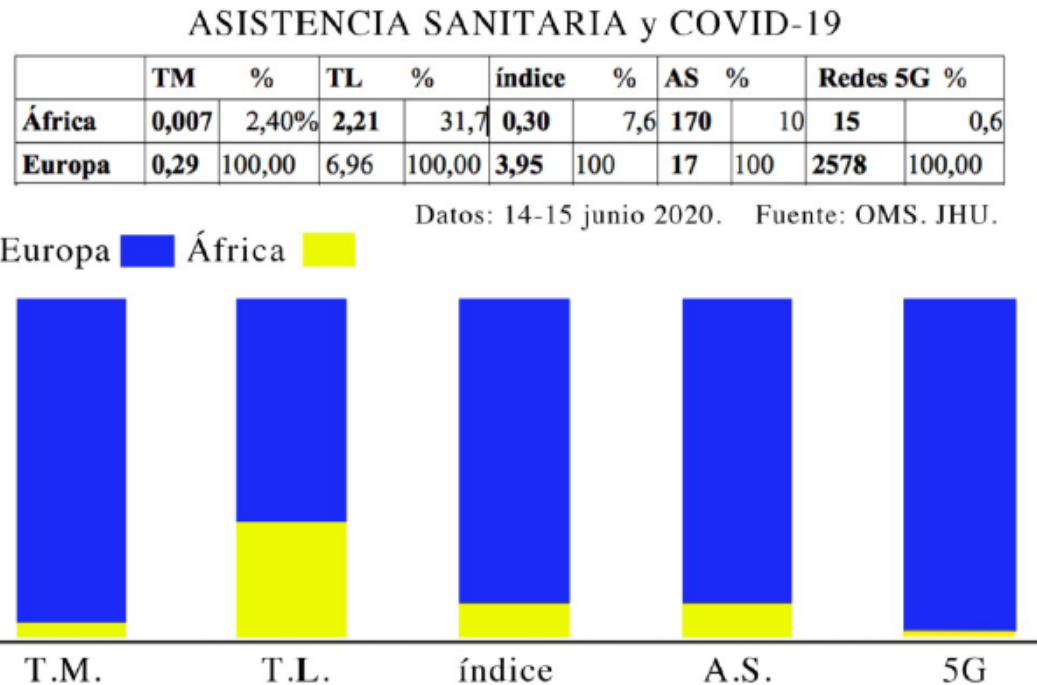


**Graph 12.** In Europe, countries with higher rates of influenza vaccination have 6.4 times higher Covid-19 mortality rates than those with the lowest vaccination rates.

The second cause, which may be attributed to a higher than expected mortality for the March 2020 peak, refers to poorly implemented hospital protocols, which caused unnecessary deaths as well as the overcrowding of healthcare facilities due to the closure of primary care and to the isolation measures for PCR-positive patients.



In that respect, the statistical data also supports this claim (**Graph 12**), for in the epidemiological study of 90 countries we found that countries with better healthcare have between a 37 to 41 times higher COVID-19 mortality rate than countries with poorer healthcare (**Graph 13**) (100).



**Graph 13.** Relationship between healthcare and deaths attributed to COVID-19. We also introduce data on the locations of 5G electromagnetic networks and their relationship with increased mortality attributed to COVID-19.

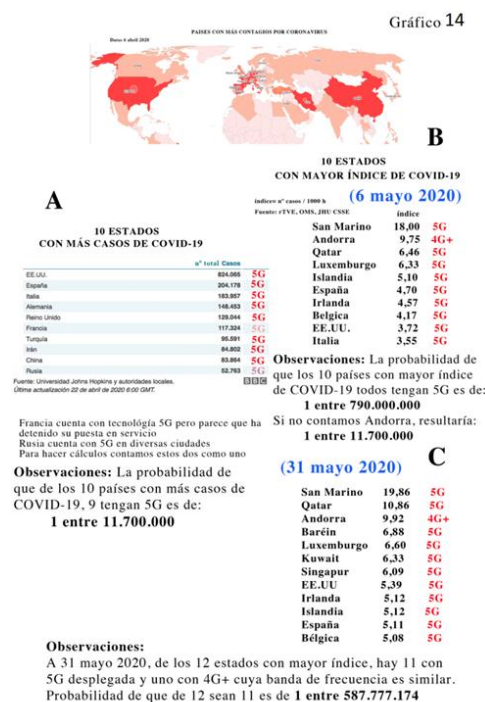
### Observations

- The Mortality Rate in Europe is 41 times higher than in Africa.
- The case fatality rate in Europe is 3 times higher than in Africa.
- The case rate in Europe is 13 times higher than in Africa.
- Number of 5G networks in Europe 172 times higher than in Africa
- Healthcare is 10 times higher in Europe than in Africa (in number of rankings)

Without a doubt, we cannot fail to mention electromagnetic networks and their potential consequences at the biological level, since mathematical calculations do show a clear and strong link between the rate of COVID-19 cases and the relationship between the COVID-19 case rate and the location of 5G networks (extendable to 4G+ as the 1st phase of the 5G NSA) (**Graph 14**) (100).

Therefore, this data confirms *that the so-called pandemic is caused by seasonal influenza, pneumonia and coronavirus-type colds that have always existed, autoimmune syndromes caused by toxins and vaccines, and environmental agents that disrupt biological systems, such as electromagnetic networks,* among others. Consequently, we must return to our normal lives, protecting our elders and especially those with pathologies, as we have always done in the past, while reinforcing primary care, diagnosing disease as medical science has always done, and also of course giving our children an education while letting them socialise and strengthen their immune system by enriching their microbiome through contact with their peers (101), and conversely, we must refuse the false medical science which is currently being implanted during these times.

Indeed, it is the duty of the authorities, on the basis of this data, to improve the level of care in residences for the elderly and to stop using this vulnerable population as test subjects for untested substances which have not yet reached the end of their testing phase (98). In short, we need to act as we have always done in response to seasonal respiratory disease epidemics, improving everything that has deteriorated in recent years, while preventing citizens from losing their fundamental rights, which are freedoms that are enshrined in our Constitution and which we should all be able to enjoy, such as the freedom of mobility throughout the Spanish territory, freedom from discrimination, freedom of enterprise, and freedom to make one's own decisions.



Graph 14.

## 7. BIBLIOGRAPHICAL REFERENCES.

1. (2020) Katherine, J. Wu. *There are more viruses than stars in the universe. Why do only some infect us?* National Geographic. <https://www.nationalgeographic.co.uk/science-and-technology/2020/04/there-are-more-viruses-stars-universe-why-do-only-some-infect-us>
2. (2005) Lindell, Debbie, et al. *Photosynthesis in marine viruses yield proteins during hosting infection.* Nature. [https://www.nature.com/articles/nature04111?error=cookies\\_not\\_supported&code=1664fe46-2360-4f82-8c81-ed2ef5a6f999](https://www.nature.com/articles/nature04111?error=cookies_not_supported&code=1664fe46-2360-4f82-8c81-ed2ef5a6f999)
3. (2010) Hoose, C. et al. *How important is biological ice nucleation in clouds on a global scale?* Biogeosciences. <https://iopscience.iop.org/article/10.1088/1748-9326/5/2/024009>
4. (2009) Villareal, Luis P. & Witzany G. *Viruses are essential agents within the roots and stem of the tree of life.* Journal of Theoretical Biology. <https://www.sciencedirect.com/science/article/abs/pii/S0022519309004895?via%3Dihub>
5. (1999) Fuhrman, Jed. A *Marine virus and their biogeochemical and ecological effects.* Nature. <https://www.nature.com/articles/21119>
6. (2017) Blinov, V. M. et al. *Viral component of the human genome.* Molecular Biology. <https://link.springer.com/article/10.1134/S0026893317020066>
7. (2008) Witzany, G. *The viral origins of telomers and telomerases and their important role in eukaryogenesis and genome maintenance.* Biosemiotics. <https://link.springer.com/article/10.1007/s12304-008-9018-0>
8. (2006) Dunlap, K. A. et al. *Endogenous retroviruses regulate periimplantation placental growth and differentiation.* PNAS. <https://www.pnas.org/content/103/39/14390>
9. (2014) Bjerregaard, B. et al. *Syncytin – 1 and its receptor is present in human gametes.* Journal of Assisted Reproduction and Genetics. <https://link.springer.com/article/10.1007%2Fs10815-014-0224-1>
10. (2018) Wang, X. et al. *Syncytin – 1, an endogenous retroviral protein, triggers the activation of CRP via TLR3 signal cascade in glial cells.* Brain, Behavior and Immunity. <https://www.sciencedirect.com/science/article/abs/pii/S0889159117304245>
11. (2015) Mele, M. et al *The human transcriptome across tissues and individuals.* Science. <https://science.sciencemag.org/content/348/6235/660.full>
12. (2020) Chuong, E. B. *Regulatory evolution of innate immunity through co-option of endogenous retroviruses.* Science. <https://science.sciencemag.org/content/351/6277/1083.full>
13. (2015) Barr, J. J. et al *Bacteriophage adhering to mucus provide a non–host-derived immunity.* PNAS. <https://www.pnas.org/content/110/26/10771>

14. (2011) Jaime, J. et al *Cell culture as an alternative for isolation and production of biologics against influenza virus*. NOVA.  
<https://hemeroteca.unad.edu.co/index.php/nova/article/view/491>
15. (2020) Huang, M. et al. *A highly pathogenic recombinant infectious bronchitis virus with adaptability in cultured cells*. Virus Research. <https://pubmed.ncbi.nlm.nih.gov/33207263/>
16. (2008) Becker, M. M. et al. *Synthetic recombinant bat SARS like coronavirus is infectious in cultured cells and in mice*. <https://www.pnas.org/content/105/50/19944> PNAS
17. (2015) Butler, B. *Engineered bat virus stirs debate over risky research*. Nature.  
<https://www.nature.com/news/engineered-bat-virus-stirs-debate-over-risky-research-1.18787#:~:text=Engineered%20bat%20virus%20stirs%20debate%20over%20risky%20research,that%20the%20novel%20coronavirus%20causing%20COVID-19%20was%20engineered.>
18. (2012) Johanna K. Kaufmann & Dirk M. Nettelbeck. *Virus chimeras for gene therapy, vaccination, and oncolysis: Adenoviruses and beyond*. Trends in Molecular Medicine. <https://www.sciencedirect.com/science/article/abs/pii/S1471491412000718>
19. (2020) Lopez-Rincon A, et al. *Specific Primer Design for Accurate Detection of SARS-CoV-2 Using Deep Learning*. [Preprint]. Bull World Health Organ. E-pub: 27 April 2020. doi:  
<http://dx.doi.org/10.2471/BLT.20.261842>
20. (2019) Lloret, Toni. *Seqirus lanza la nueva vacuna antigripal de cultivo celular en España*.  
<http://www.pmfarma.es/noticias/27553-seqirus-lanza-la-nueva-vacuna-antigripal-de-cultivo-celular-en-espana.html>
21. (2003) Drosten et al. *Identification of a Novel Coronavirus in Patients with Severe Acute Respiratory Syndrome*. New England Journal of Medicine.  
<https://www.nejm.org/doi/full/10.1056/NEJMoa030747>
22. (2020) Corman et al. *Detection of 2019 novel coronavirus (2019-nCoV) by real-time RT-PCR*. Eurosurveillance. <https://www.eurosurveillance.org/content/10.2807/1560-7917.ES.2020.25.3.2000045>
23. (2011) Woo, P. C. Y. et al. *Discovery of Seven Novel Mammalian and Avian Coronaviruses in the Genus Deltacoronavirus Supports Bat Coronaviruses as the Gene Source of Alphacoronavirus and Betacoronavirus and Avian Coronaviruses as the Gene Source of Gammacoronavirus and Deltacoronavirus*. Journal of Virology. <https://jvi.asm.org/content/86/7/3995>
24. (2020) Ioannidis, J. P. A. *Tasa de letalidad por la infección de la COVID – 19 calculada a partir de los datos de seroprevalencia*. Boletín WHO. <https://www.who.int/bulletin/volumes/99/1/20-265892-ab/es/>

25. (2020) Zhu, N. et al *A Novel Coronavirus from Patients with Pneumonia in China, 2019*. New England Journal of Medicine. N Engl J Med 2020; 382:727-733  
DOI: 10.1056/NEJMoa2001017 <https://www.nejm.org/doi/full/10.1056/nejmoa2001017>
26. (2005) Hofmann, H. *Human coronavirus NL63 employs the severe acute respiratory syndrome coronavirus receptor for cellular entry*. PNAS. <https://www.pnas.org/content/102/22/7988>
27. (2020) Protocol RT PCR – WHO.  
<https://www.who.int/docs/default-source/coronaviruse/protocol-v2-1.pdf>
28. (2000) Crespo, M. P. *El diagnóstico viral por el laboratorio*. Colombia Médica 31(3): 135 - 150.
29. (2017) Corrales Morales, M. et al. *Identificación y caracterización molecular de cianobacterias tropicales de los géneros Nostoc, Calothrix, Tolypothrix y Scytonema (Nostocales: Nostocaceae), con posible potencial biotecnológico*. UNED Research Journal 9(2): 280-288
30. (2007) Lanteri, A.A., 2007. *Código de barras del ADN y sus posibles aplicaciones en el campo de la entomología*. Rev. Soc. Entomol. Argent. 66(3-4); 15 -- 25.
31. (1996) F.A.O. *Determinación de la situación de una plaga en un área*. Normativas Internacionales para Medidas Fitosanitarias 8. Secretaría de la Convención Internacional de Protección Fitosanitaria.
32. (2020) Martínez Albarracín, M. J. *Estudio de las pruebas analíticas para la detección del SARS CoV 2*. <https://medicosporlaverdad.es/wp-content/uploads/2020/11/Dossier-PCRs-2.0.pdf>
33. (2000) Tipnis, S. R. et al. *A Human Homolog of Angiotensin-converting Enzyme*. Journal of Biological Chemistry. [https://www.jbc.org/article/S0021-9258\(20\)89003-6/fulltext](https://www.jbc.org/article/S0021-9258(20)89003-6/fulltext)
34. Hikmet, F. et al. *"The protein expression profile of ACE2 in human tissues"*. Molecular Systems Biology. <https://www.embopress.org/doi/pdf/10.15252/msb.20209610>
35. (2020) Junta Argentina de Revisión Científica. *Cronología Target Vacuna Covid-19*. [https://angelalonso.files.wordpress.com/2020/11/4\\_5879649008036612675.pdf](https://angelalonso.files.wordpress.com/2020/11/4_5879649008036612675.pdf)
36. (2020) Stelzer-Braid, S. et al. *Virus isolation of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) for diagnostic and research purposes*. The Journal of the Royal College of Pathologist of Australia. <https://linkinghub.elsevier.com/retrieve/pii/S0031302520309399>
37. (2020) COVID – 19 Vaccine AstraZeneca. Product Information.  
[https://cima.aemps.es/cima/pdfs/es/ft/1211529001/FT\\_1211529001.pdf](https://cima.aemps.es/cima/pdfs/es/ft/1211529001/FT_1211529001.pdf)
38. (2021) *Información científico – técnico. Enfermedad COVID 19*. Actualización del 15 de enero de 2021. Secretaría de Estado de Sanidad. Centro de Coordinación de Alertas y Emergencias Sanitarias.  
<https://www.mscbs.gob.es/profesionales/saludPublica/ccayes/alertasActual/nCov/documentos/ITCoronavirus.pdf>

39. (2020) Malajovich, M.A. *ADN, ARN e información*. Biotecnología: enseñanza y divulgación. <http://bteduc.com>
40. (2007) Slotkin, R. K. & Martienssen, R. *Transposable elements and the epigenetic regulation of the genome*. Nature. <https://www.nature.com/articles/nrg2072>
41. (1991) Harrison, S. *A structural taxonomy of DNA-binding domains*. Nature: 353: 715 – 719.
42. (2009) Ostos Ortiz, O.L. *La molécula de la vida en su dimensión hipercompleja: diálogo entre saberes de sistemas complejos e hipercomplejos*. NOVA publicación Científica en Ciencias Biomédicas 7(12): 111 – 174.
43. (2006) Gómez, L.A. Nobel Prize in Physiology, Medicine and Chemistry, 2006. *Una nueva dimensión del ARN en la regulación de la expresión genética y como herramienta experimental y terapéutica*. Biomédica, 26: 475 – 484.
44. (2010) Somoza, A. *Modificaciones químicas en ARN interferente: de la investigación básica a las aplicaciones terapéuticas*. An. Quim. 106(3); 215 – 222.
45. (2018) Ramón y Cajal, S. & Hümmel, S. *Más allá de los genes. Cómo podemos entender el DNA no codificante*. Anales de la Real Academia Nacional de Medicina de España, 135(3): 230 – 236.
46. (2011) González Paredes, F.J. *Alteraciones en el procesamiento del pre-ARN m de los genes pkd1 y pkd2 debidas a mutaciones exónicas relacionadas con la enfermedad poliquística renal autosómica dominante*. Tesis Doctoral. Universidad de La Laguna.
47. (2004) Britten, R.J. *Coding sequences of functioning human genes derived entirely from mobile element sequences*. Proc. Natl. Acad. Sci. USA, 101(48): 16825 – 16930.
48. (2003) Dunn, C.A., Medstrand, P. and Mager, D.L. *An endogenous retroviral long terminal repeat is the dominant promoter for human  $\beta$ 1,3-galactosyltransferase 5 in the colon*. PNAS, 100(22): 12841 – 12846.
49. (2006) Dunlap, K.A., Palmarini, M., Varela, M., Burghardt, R.C., Hayashi, K., Farmer, J.L. and Spencer, T.E. *Endogenous retroviruses regulate periimplantation placental growth and differentiation*. PNAS, 103(39): 14390 – 14395.
50. (2002) Anderson, A.C., Venables, P.J.W., Tönjes, R.R., Scherer, J., Eriksson, L. and Larsson, E. 2002. *Developmental expression of HERV-R (ERV3) and HERV-K in human tissue*. Virology, 297(2): 220 – 225.
51. (2005) Seifharth, W., Frank, O., Zeilfelder, U., Spiess, B., Greenwood, A.D., Hehlmann R and Leib-Mösch, C. 2005. *Comprehensive analysis of human endogenous retrovirus transcriptional activity in human tissues with a retrovirus-specific microarray*. J. Virol. 79(1): 341 – 352.
52. (2008) Saito, M., Sato-Bigee, C and Yu, R.K. *Neuraminidase activities in oligodendroglial cells in rat brain*. Journal of Neurochemistry, 58(1): 78 – 82.



53. (2017) Berger, S.M. et al. *Forebrain-specific, conditional silencing of Staufien2 alters synaptic plasticity, learning, and memory in rats*. *GenomeBiol* **18**, 222 (2017).  
<https://doi.org/10.1186/s13059-017-1350-8>
54. (2018) Pastuzyn, E.D et al. *The neuronal gene Arc encodes, a repurposed retrotransposon gag protein mediates intercellular RNA transfer*. *Cell*, 172: 275 – 288.
55. (2020) Turanova et al. *In situ structural analysis of SARS-CoV-2 spike reveals flexibility mediated by three hinges*. <https://science.sciencemag.org/content/370/6513/203>
56. (2018) Song W, Gui M, Wang X, Xiang Y. 2018. *Cryo-EM structure of the SARS coronavirus spike glycoprotein in complex with its host cell receptor ACE2*. *PLoS Pathog.* 14(8):e1007236.
57. (2008) Judith M. White, Sue E. Delos, Matthew Brecher & Kathryn Schornberg. *Structures and Mechanisms of Viral Membrane Fusion Proteins: Multiple Variations on a Common Theme*.
58. (2008) White, J. M. et al. *Structures and Mechanisms of Viral Membrane Fusion Proteins: Multiple Variations on a Common Theme*. *Critical Reviews in Biochemistry and Molecular Biology*. <https://doi.org/10.1080/10409230802058320>
59. (2003) Berend Jan Bosch, Ruurd van der Zee, Cornelis A. M. de Haan, Peter J. M. Rottier. *The Coronavirus Spike Protein Is a Class I Virus Fusion Protein: Structural and Functional Characterization of the Fusion Core Complex*. DOI: 10.1128/JVI.77.16.8801-8811.2003
60. (2004) Xu et al. *Characterization of the Heptad Repeat Regions, HR1 and HR2, and Design of a Fusion Core Structure Model of the Spike Protein from Severe Acute Respiratory Syndrome (SARS) Coronavirus*. *Biochemistry*.
61. (2020) Gallaher, G. *Response to nCoV2019 Against Backdrop of Endogenous Retroviruses*.  
<https://virological.org/t/response-to-ncov2019-against-backdrop-of-endogenous-retroviruses/396>
62. (2003) Frendo, J. L. et al *Direct involvement of HERV-W Env glycoprotein in human trophoblast cell fusion and differentiation*. *Molecular Cell Biology*.
63. (2000) Mi, S., Lee, X., Li, Xp. et al. *Syncytin is a captive retroviral envelope protein involved in human placental morphogenesis*. *Nature*, 403, 785–789 <https://doi.org/10.1038/35001608>
64. UniProtDataBase. Syncytin – 1 ERVW – 1 <https://www.uniprot.org/uniprot/Q9UQF0>
65. (2019) Adjimon et al. *Endogenous retrovirus - encoded Syncytin - 2 contributes to exosome - mediated immunosuppression of T cells*. *Biology of Reproduction*.
66. (2010) Feng, Y. et al. *Brain – selective overexpression of human angiotensin – converting enzyme type 2 attenuates neurogenic hypertension*. *Circulation Research*.  
<https://www.ahajournals.org/doi/10.1161/CIRCRESAHA.109.208645>

67. (2015) Janas, A. M. et al. *Exosomes and other extracellular vesicles un neural cells and neurodegenerative diseases*. Elsevier.
68. (2020) Pradhan, et al. *Uncanny similarity of unique inserts in the 2019-nCoV spike protein to HIV-1 gp120 and Gag*. <https://www.biorxiv.org/content/10.1101/2020.01.30.927871v1>
69. (2021) Emanuela Balestrieri, PhD, et al. First evidence of pathogenic HERV-W envelope expression in T lymphocytes in association with the respiratory outcome of COVID-19 patients. Europe PCM. <https://europepmc.org/article/ppr/ppr280380>
70. (2014) Li, F. et al. *Transcriptional derepression of the ERVWE1 locus following influenza A virus infection*. Journal of Virology. <https://pubmed.ncbi.nlm.nih.gov/24478419/>
71. (2020) Taylor, A. *Why the Queensland University COVID-19 Vaccine Failed*. Science The Wire. <https://science.thewire.in/health/queensland-covid-19-vaccine-hiv-protein/>
72. (2021) Classen, et al. *COVID-19 RNA Based Vaccines and the Risk of Prion Disease*. Microbiology & Infectious Diseases. <https://scivisionpub.com/pdfs/covid19-rna-based-vaccines-and-the-risk-of-prion-disease-1503.pdf>
73. (2010) Bollati V. & Bacarelli, A. *Enviromental epigenetics*. Heredity, 105: 105 – 112. <https://www.nature.com/articles/hdy20102>
74. (2011) Delgado Coello, B. *¿Qué es la epigenética?* Project Science Communication. <https://www.researchgate.net/publication/235324187>
75. (2010) Sandín, M. *Pensando la evolución, pensando la vida*. Cauac editorial nativa.
76. (2021) Etxebarria Garate, J. A. Utilización de los test de PCR, % positivos y la incidencia acumulada (IA) a 14 días. <http://www.biologosporlaverdad.es/incidenciacumulada.pdf>
77. Indicadores de Mortalidad. Instituto Nacional de Estadística. <https://www.ine.es/jaxiT3/Datos.htm?t=1414>
78. Tasas de mortalidad. Instituto Nacional de Estadística. [https://www.ine.es/dyngs/INEbase/es/operacion.htm?c=Estadistica\\_C&cid=1254736177003&menu=resultados&idp=1254735573002](https://www.ine.es/dyngs/INEbase/es/operacion.htm?c=Estadistica_C&cid=1254736177003&menu=resultados&idp=1254735573002)
79. Defunciones. Instituto Nacional de Estadística. <https://www.ine.es/jaxiT3/Tabla.htm?t=35177>
80. Periodos de exceso de mortalidad. MoMo. [https://momo.isciii.es/public/momo/dashboard/momo\\_dashboard.html](https://momo.isciii.es/public/momo/dashboard/momo_dashboard.html)
81. (2021) *Técnicas de amplificación de ácidos nucleicos en las que se utiliza la reacción en cadena de la polimerasa (PCR) para detectar el SARS-CoV-2*. [https://www.who.int/es/news/item/20-01-2021-who-information-notice-for-ivd-users-2020-05?fbclid=IwAR1ktLE7O6SsxiNliLq\\_HINZUjC4RIkBBqQEw0yx7PDJqNwvuX9TK8MTDxs](https://www.who.int/es/news/item/20-01-2021-who-information-notice-for-ivd-users-2020-05?fbclid=IwAR1ktLE7O6SsxiNliLq_HINZUjC4RIkBBqQEw0yx7PDJqNwvuX9TK8MTDxs)

82. (2020) Eisenberg, J. *Qué es el R0, el número que siguen los científicos para ver la intensidad del coronavirus*. The Conversation. <https://theconversation.com/que-es-el-r0-el-numero-que-siguen-los-cientificos-para-ver-la-intensidad-del-coronavirus-137744>
83. (2020) *Science Brief: Options to Reduce Quarantine for Contacts of Persons with SARS-CoV-2 Infection Using Symptom Monitoring and Diagnostic Testing*. CDC. <https://www.cdc.gov/coronavirus/2019-ncov/more/scientific-brief-options-to-reduce-quarantine.html>
84. (2009) *Consejos sobre la utilización de mascarillas en el entorno comunitario ante la aparición de brotes de gripe por A (H1N1)*. [https://www.who.int/csr/resources/publications/swineflu/masks\\_community/es/](https://www.who.int/csr/resources/publications/swineflu/masks_community/es/)
85. (2020) Schwarz, S. et al. *Corona children studies "Co-Ki": First results of a Germany-wide registry on mouth and nose covering (mask) in children*. Research Square. [10.21203/rs.3.rs-124394/v3](https://doi.org/10.21203/rs.3.rs-124394/v3)
86. (2020) Preguntas y respuestas sobre la transmisión de la COVID-19. OMS. <https://www.who.int/es/news-room/q-a-detail/coronavirus-disease-covid-19-how-is-it-transmitted>
87. (2020) Cao, S. et al. *Post - lockdown SARS-CoV-2 nucleic acid screening in nearly ten million residents of Wuhan, China*. Nature. <https://www.nature.com/articles/s41467-020-19802-w>
88. (2021) Mina, M. J. et al. *Clarifying the evidence on SARS-CoV-2 antigen rapid tests in public health responses to COVID-19*. The Lancet. [https://doi.org/10.1016/S0140-6736\(21\)00425-6](https://doi.org/10.1016/S0140-6736(21)00425-6)
89. (2020) *La gripe en el contexto de la pandemia de COVID 19*. Sistema de vigilancia de la gripe en España. <https://vgripe.isciii.es/PresentarNoticia.do?idNoticia=147&idtemp=20202021>
90. (2020) *El nuevo coronavirus podría "no irse nunca": la advertencia de la OMS sobre la posibilidad de que el SARS-CoV-2 se vuelva endémico*. BBC News. <https://www.bbc.com/mundo/noticias-52657184>
91. (2020) Mapa de Incidencias Acumuladas por 7 días en España. Instituto Carlos III. <https://cnecovid.isciii.es/covid19/>
92. (2020) Acontecimientos adversos notificados en España tras la vacunación frente a COVID – 19. Periodo 27/12/2020 – 12/01/2020. Agencia Española del Medicamento y Productos Sanitarios. <https://app.powerbi.com/view?r=eyJrIjoizWFIMzU1OGU1MDM1YS00YzBILWEyM2EtOTBhNjRiNDQyZjU1IiwidCI6IjJkM2I1MGUwLTZlZjQ0NGViYy05MjQ2LTdkMWwNiYjc3MDg5YyIsImMiOiJh9>
93. (2020) Ioannidis, J. P. A. *Tasa de letalidad por la infección de la COVID – 19 calculada a partir de los datos de seroprevalencia*. Boletín WHO. <https://www.who.int/bulletin/volumes/99/1/20-265892-ab/es/>

94. (2020) *Informes de situación de COVID – 19 en España*. Instituto Carlos III.  
<https://www.isciii.es/QueHacemos/Servicios/VigilanciaSaludPublicaRENAVE/EnfermedadesTransmisibles/Paginas/InformesCOVID-19.aspx>
95. (2020) Martínez Albarracín, M. J. *La Covid-19 es un síndrome de inmunodeficiencia mediada por tóxicos y/o por vacunas*. [https://cauac.org/articulos/covid19-y-autoinmunidad/?fbclid=IwAR13\\_IMGJlicAM2mTlycE1JgWdvRfr29ozawo0HsYQ\\_cr-DLgClqWkKtduA](https://cauac.org/articulos/covid19-y-autoinmunidad/?fbclid=IwAR13_IMGJlicAM2mTlycE1JgWdvRfr29ozawo0HsYQ_cr-DLgClqWkKtduA)
96. (2020) Gastón Añaños, J. F. et al. *Posible causa de la pandemia por coronavirus: Interferencia inmunológica entre el POLISORBATO 80 de la vacuna antigripal adyuvada y el SARS-CoV-2*. <https://vacunasaep.org/sites/vacunasaep.org/files/gaston-ananos-hipotesis-vacuna-gripe-covid-19-version1.pdf>
97. (2020) Wehenkel, C. *Positive association between COVID-19 deaths and influenza vaccination rates in elderly people worldwide*. Peer J. <https://peerj.com/articles/10112/>
98. (2020) *Estrategia de vacunación frente a COVID 19 en España*. Grupo de Trabajo Técnico de Vacunación COVID-19, de la Ponencia de Programa y Registro de Vacunaciones. Consejo Interterritorial Sistema Nacional de Salud. Informes del 18 de diciembre de 2020, 21 de enero de 2021 y 9 de febrero de 2021.  
<https://www.mscbs.gob.es/profesionales/saludPublica/prevPromocion/vacunaciones/covid19/>
99. (2017) Eurostat. *Influenza vaccination rate*.  
<https://ec.europa.eu/eurostat/en/web/products-eurostat-news/-/DDN-20191209-2>
100. (2020) Payeras i Cifre, T. *La distribución asimétrica de casos COVID-19 y su relación con las redes 5G. Estudio de los mecanismos causales*. Teoría Ambiental.  
<https://drive.google.com/file/d/17JGCWwGugKN5nzR0EZl-d-qv9IoNlssl/view>
101. (2020) Zheng, D. Liwinski, T. & Elinav E. *Interaction between microbiota and immunity in health and disease*. Cell Research 30, pages 492–506 (2020).  
<https://www.nature.com/articles/s41422-020-0332-7>