The Faulty Science Surrounding COVID-19

A Critical Examination of the Studies Claiming to Have Isolated SARS-CoV-2 as the Causative Agent of COVID-19

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Introduction

In this paper, we show that the science surrounding SARS-CoV-2 (i.e., the virus said to cause COVID-19) is very faulty. In fact, it is so faulty that the entire existence of SARS-CoV-2 as a pathogenic virus is highly questionable. While such a claim may sound absurd at first, we will show that our claim is sound by critically examining published studies to demonstrate that SARS-CoV-2 has not been purified according to established industry standards.

Before we do this, it should also be noted that the existence of COVID-19 as a new illness cannot be proved through mere clinical diagnosis. In particular, COVID-19 produces no symptoms that differ from the common cold or seasonal flu [1], produces no symptoms at all in nearly 80% of infected people [2], and has a cumulative hospitalization rate basically on par with the seasonal flu [3]. In other words, COVID-19 has no distinguishing traits and would have probably been identified as the seasonal flu if were not for all of the extensive testing [4].

Since COVID-19 cannot be identified through clinical diagnosis, the use of RT-PCR (reverse transcription polymerase chain reaction) molecular testing is being used extensively to detect what is claimed to be the viral RNA of SARS-CoV-2. However, this creates two additional problems: (1) RT-PCR testing has been shown to be unreliable and isn't even validated for use on asymptomatic people [5]; and (2) it is impossible to determine if the detected RNA is viral without first isolating the alleged virus. In other words, the RNA being detected through RT-PCR testing may not be viral at all. It may be endogenous to the cells.

Some scientists have claimed that they have isolated SARS-CoV-2 and identified it as the causing agent of the disease being called COVID-19. However, when examining their studies, it becomes quickly apparent that they have not isolated it. For example, the authors of one paper published in the New England Journal of Medicine claim that they have isolated SARS-CoV-2 in the introductory summary of their paper, but then admit in the body of the paper that their study does not satisfy the industry "gold standard" for determining the cause of an infectious disease [6]. Misleading summaries can create rumors that have dire repercussions in the real world. This appears to be exactly what has happened in the case of COVID-19 and is one of the reasons for the drastic measures taken all over the world by governments.

To establish our claim that there is still no scientific proof that SARS-CoV-2 exists as a pathogenic virus, we shall do the following:

1. Identify the "gold standard" for determining the cause of an infectious disease
2. Identify how to isolate (i.e., purify) the virus according to the "gold standard" and describe some shortcomings of this method
3. Examine claims of satisfying the standard for other coronaviruses
4. Examine claims of satisfying the standard for SARS-CoV-2

Section 1

What is the “gold standard” for determining the cause of an infectious disease?

The recognized “gold standard” for determining the cause of an infectious disease is Koch’s Postulates [7].

The postulates were formulated by Robert Koch in an 1884 paper entitled “ Die Aetiologie der Tuberkulose” and slightly revised in 1890. They are as follows:

(1) That the parasite occurs in every case of the disease in question, and under circumstances which can account for the pathological changes and clinical course of the disease;
(2) That it occurs in no other disease as a fortuitous and non-pathogenic parasite;
(3) That it, after being fully isolated from the body and repeatedly grown in pure culture, can induce the disease anew; [8]

Did Robert Koch ever fulfill his own postulates?

This may sound like a strange question to ask. After all, why would Koch create postulates that even he himself could not fulfill? Well, he did claim to fulfill the postulates in a number of diseases including anthrax, tuberculosis, tetanus, and many animal diseases, but he was not without his opponents, among whom was Louis Pasteur. [8]

Criticism of his work on tuberculosis was so great that it prompted Koch to publish a response in 1883. [8]

Likewise, Pasteur also criticized Koch’s work on anthrax, calling it “inconclusive.” [9]

With regard to anthrax, K. Codell Carter sums up the situation as follows:

“If one asks whether Koch or Pasteur ultimately proved that the anthrax bacillus caused anthrax, perhaps the best answer is neither alone, but, at least to some extent, both together.” [9]

Carter’s conclusion of the situation reminds us of the German supreme court ruling in favor of biologist Stephan Lanka’s claim that the existence of measles was never proven. In that court case, six proofs were furnished in proof of measles, but the court ruled that none of the six proofs individually proved measles.

Professor Andreas Podbielski, head of the Department of Medical Microbiology, Virology and Hygiene at the University Hospital in Rostock, who was one of the appointed experts at the trial, stated that even though the existence of the measles virus could be concluded from the summary of the six papers submitted by Dr. Bardens, none of the authors had conducted any controlled experiments in accordance with internationally defined rules and principles of good scientific practice. He concluded that a publication about the existence of the measles virus that stands the test of good science has yet to be delivered [10], [11].

Although the Lanka case is not directly relevant to SARS-CoV-2, it does show that proposed proofs are often not good science and fail to prove what they claim.

As far as Robert Koch’s work, we are not sure to what extent he succeeded in fulfilling his postulates, but it should be clear that some people did not acknowledge his proofs.

Can Koch’s postulates be applied to viruses?

Virologist Thomas Rivers stated in a 1936 speech that “It is obvious that Koch's postulates have not been satisfied in viral diseases” [12].

As a result, he proposed his own list of conditions as follows:

(1) A specific virus must be found associated with a disease with a degree of regularity.
(2) The virus must be shown to occur in the sick individual not as an incidental or accidental finding but as the cause of the disease under investigation.

Rivers conditions are quite condensed. With regard to association, he changed "always present" to "present with a degree of regularity." In particular, he said, "In the first place, it is not obligatory to demonstrate the presence of a virus in every case of the disease produced by it" [12]. This is really a curious statement because it is hard to imagine a virus causing a disease even if it is not present.

The condition to prove causality still remains.

An older version of the Dictionary of Virology defined Rivers' modification of Koch's postulates as follows:

(1) Isolation of virus from diseased hosts.
(2) Cultivation in experimental hosts or host cells.
(3) Proof of filterability (to exclude larger pathogens).
(4) Production of a comparable disease in the original host's species or in related ones.
(5) Reisolation of the virus.
(6) Detection of a specific immune response to the virus. [13]

There are couple major differences. As mentioned above, Rivers does not include the need for the virus to be present in every manifestation of the disease. That definitely weakens association. As for causation, Koch's original postulates state that the microbe must be grown in pure culture. However, it is said that viruses are not able to live on their own due to their inability to metabolize. They need a host cell. Therefore, Rivers' modified postulates state that the microbe be cultivated in host cells. Since they are cultivated in host cells, the need for filterability also becomes necessary. Finally, he adds that it is necessary to re-isolate the virus and detect an immune response to it in previously infected individuals.

Are there any shortcomings to Koch's postulates?

Yes, there are many. In Section 2 we identify several shortcomings that have the potential to invalidate the whole process. It is important to notice that Koch's postulates are logical postulates based on the idea that a cause must come before a consequence, and that the cause must be present to cause a consequence. They are not a bulletproof scientific method for determining the cause of infectious disease.

Thomas Rivers' acknowledged this when he said, "If the inoculated animals become sick or die in a characteristic manner, and, if the disease in them can be transmitted from animal to animal by means of inoculations with blood or emulsions of involved tissues free from ordinary microbes or rickettsiae, one is fairly confident that the malady in the experimental animals is induced by a virus." Notice that he doesn't say "confident," "very confident," or "certain." He says "fairly confident." He knew very well that there were many shortcomings to the postulates that could invalidate the whole process.

Are Koch's postulates still the gold standard today?

Yes, they are. In fact, some of the studies we will look at directly acknowledge Koch's postulates as the standard for determining the cause of infectious disease.

According to the editors of the journal Nature Reviews Microbiology, “More than 120 years after they were first proposed, Koch’s postulates still remain the gold standard for any investigation that sets out to prove the etiology (origin or cause) of an infectious disease.” [14]

Are there any other standards being applied today?

Some scientists have suggested that nucleic acid sequencing-based methods for identifying microbes are more applicable than Koch’s postulates. In particular, PCR is being used to associate viruses with diseases.

However, as mentioned above, PCR testing is prone to reliability problems [5] and cannot determine if the detected genetic material is viral or endogenous to the cells. In other words, it cannot determine the cause of infectious disease. It can only be used to try to find association. There is a big difference between association and causality. For example, firefighters are often associated with fires, but not many of us would dare say that firefighters were the ones who caused the fires. Also, flies are associated with garbage, but none of use would say that flies caused the garbage.

These sequence-based methods attempt to provide evidence of causality through detection of genetic material (e.g., what they think is viral RNA, etc.) at different stages of disease. For example, if genetic material can also be found before the onset of a disease, the association between the genetic sequence and disease is said to be stronger. However, this is still is an associative relationship and not a causal relationship. Would it be okay to say that flies caused garbage because some were present before the garbage arrived? Of course not. In addition, it is not very practical because most people do not undergo testing before the onset of symptoms.

This method is also subject to many of the shortcomings of Koch’s postulates. We will discuss some of these shortcomings in Section 2.

For more information on sequencing based methods, we refer the reader to the work of Fredricks and Relman [15].

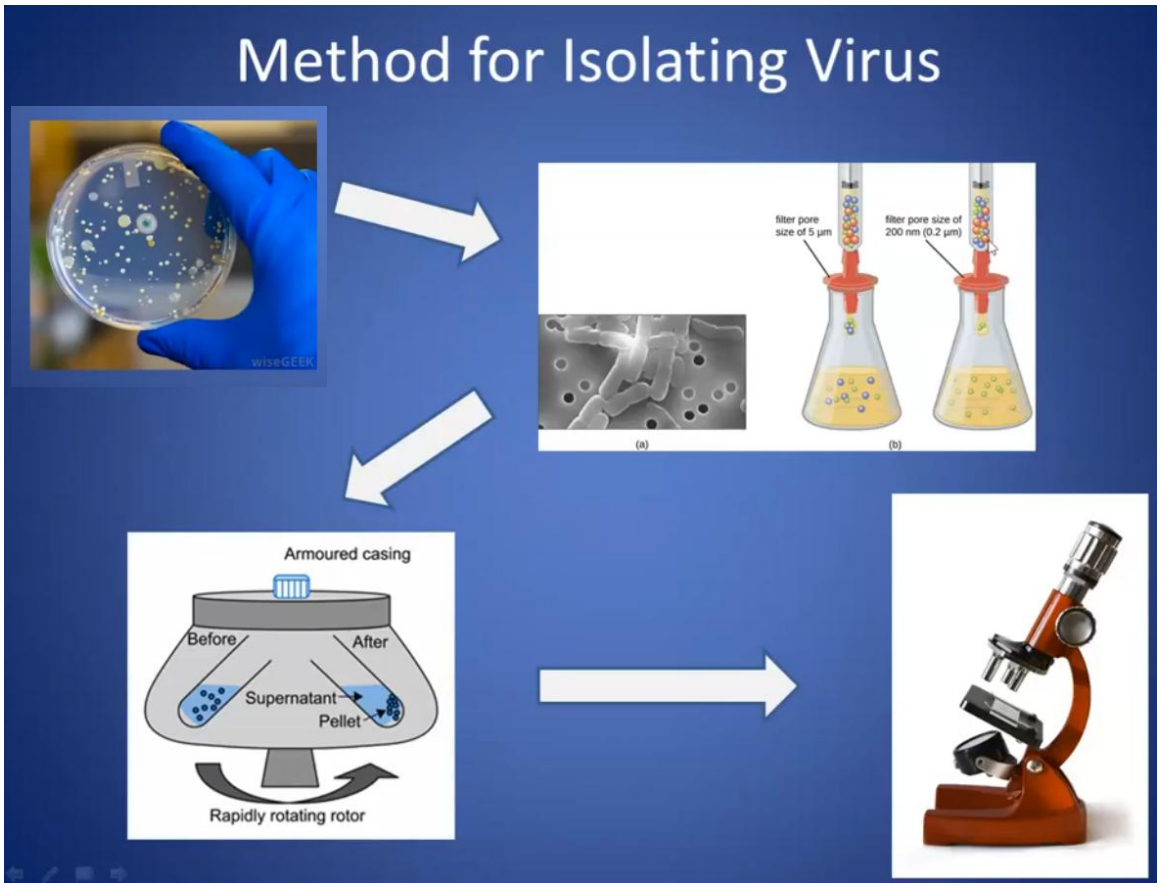
Section 2

Some of the studies we will be looking at refer to Koch's postulates. However, as we shall see, they are actually referring to Koch's postulates as modified by Thomas Rivers. This is because, according to Rivers, the modified postulates better suit viruses. Therefore, for the remainder of this paper, the "gold standard" for determining the cause of infectious disease will refer to Koch's postulates as modified by Thomas Rivers.

This section is important because it will show us how to fulfill postulates 1 to 3.

How can a virus be isolated according to Koch's postulates?

*Most of the content for this sub-section was taken from researcher David Crowe's article on isolation and purification [16].



Isolation clearly means separating the virus from all other organic materials. Logically, this requires the following steps:

1. Obtaining a specimen (such as sputum or lung fluid) from a patient.
2. Culturing materials believed to contain a virus in other cells (e.g. the Vero cells), since viruses are not believed to replicate outside target cells.
3. Purifying virus particles by removing the liquid on top of the culture (supernatant) believed to contain the free viral particles by filtering (to eliminate particles larger than a virus) and by centrifugation (to separate particles by density).
4. Putting a portion of the material under an electron microscope to verify that almost all that can be seen are particles of the same size and shape.
5. Breaking down the proteins and genetic material (RNA or DNA, depending on the virus) in the rest of the sample and analyzing them (e.g. sequencing the RNA or DNA).

It should be noted that steps 1 to 4 of the method for isolating viruses correspond to steps 1 to 3 of Koch's postulates. Step 5 does not correspond to any of Koch's postulates, but it is an important step in acquiring the proteins and genetic material of the virus. It also enables scientists to analyze the virus to sequence its genome. This is very useful in creating tests, such as the RT-PCR test kits currently being used to detect SARS-CoV-2 in specimens.

The figure shows steps 2 to 4 of the method for isolating viruses. Step 2 corresponds to the top-left picture. Step 3 corresponds to the top-right and bottom-left pictures. Step 4 corresponds to the bottom-right picture. Steps 1 and 5 are not shown in the figure.

Are there any shortcomings to this method for isolating viruses?

Yes, there are many. We shall list a few of them here.[1]

Problems with Steps 1 and 2

The method of culturing materials in other cells is problematic for many reasons. In order to do this, a sample specimen, such as sputum, needs to be extracted from the body. However, the extraction process alone could induce variables, mainly stressor variables, that could influence the specimen. Furthermore, after it is extracted, the specimen is often further stressed through the addition of antibiotics and/or other additives such as serums.

The human body is full of viruses, most of them inactivated and harmless. However, the induced stress of extracting and preparing the specimen for culturing in other cells has the potential to activate a response that appears to be a pathogenic virus, but is actually just a defense mechanism. Also, the addition of antibiotics and/or serums to the specimen could produce a response since those additives are seen as invaders. In other words, there would probably be no activation at all without the induced stress and additives. Likewise, the host cells (usually Vero cells) also see the impure materials (i.e., the sample extracted and prepared for culturing) as foreign invaders, creating even more stress. And remember, this is all taking place outside the body, in the unnatural environment of a laboratory.

Furthermore, this induced stress could potentially cause the specimen to change its genetic sequences. Any alteration to the genetic material of the specimen would be devastating to attempts to sequence and analyze RNA and DNA. With regard to this, the research work of geneticist Barbara McClintock is a milestone. In her Nobel Prize paper from 1983, she reports that the genetic material of living beings can constantly alter, by being hit by "shocks." These shocks can be toxins, but also other materials that produced stress in the test-tube. This in turn can lead to the formation of new genetic sequences, which were unverifiable (in vivo and in vitro) before [17][18].

Problems with Step 3

The method of purifying the virus through centrifugation and filtering is also problematic due to physical similarities between the target virus and other viruses.

It is said that there are about 10 nonillion (10 to the 31st power) individual viruses existing in our world [19] and millions of viruses living in the human body [20]. Of these countless number of viruses, it is said that only over 6,000 of them have been described in detail [21]. Looking for SARS-CoV-2 among all of these viruses is literally like looking for a needle in a haystack.

[1] Some of the content for this subsection was adapted from posts and comments on the Piece of Mindful blog (pieceofmindful.com).

Density gradient centrifugation is a process where particles and cells are separated based on their density. The density of SARS-CoV-2 is about 1 g/ml [22]. This type of centrifugation tends to be difficult to use and is susceptible to contamination [23][24].

Filtration separates particles and cells based on their size. SARS-CoV-2 has a diameter between 60-140 nm [48]. In order to isolate SARS-CoV-2, it must be separated from all other viruses based on its density and size. This may sound good in theory, but in practice, there could be thousands or even millions of other viruses with approximately the same density and size. It would be as if we were looking for one specific person in the whole world, but could only give a description that he was between 170 to 180 cm and weighed about 70 kg. There would literally be thousands upon thousands of people who fit our description.

Postulate 3 indicates this problem when it says "Proof of filterability (to exclude larger pathogens)" [13]. In other words, filtering can exclude larger pathogens, but it cannot deal with pathogens that are the same size or smaller. Even if smaller particles are separated out through other methods, particles of the same size would still be left behind.

Also keep in mind that Step 3 induces more stress on the specimen.

Problems with Step 4

The method of verifying that all particles are the same size and shape with an electron microscope is also problematic for the same reasons mentioned in "Problems with Step 3." Even if all particles were approximately the same size and shape, that doesn't mean that all particles are the same virus. What is being looked at might not even be a virus at all.

For example, cells produce exosomes, which are 30-100 nm in diameter and typically shaped like a cup. Exosomes are involved in immune response and signal transduction, and despite their potential applications in eliciting a "positive" immune response, exosomes might induce some "unwanted" immune responses, such as immune tolerance and immune evasion [25].

Based on the role of exosomes, it is not unimaginable that the induced stress of Step 1 and Step 2 could potentially cause a response that involves exosomes. Now, the interesting thing is that exosomes and coronaviruses are physically similar and this makes it difficult to distinguish between them when looking at them under an electron microscope.

John Ziebuhr, editor of Coronavirus Volume 96, indirectly suggested this very thing when commenting on a study on coronaviruses. He said, "Careful measurement of coronavirus particles in cryoelectron micrographs and tomograms reveals that most coronavirus particles are slightly prolate spheroids that differ from the shape of more spherical exosomal vesicles that appear in the same images." Reading between the lines, coronavirus particles and exosomal vesicles are very difficult to distinguish between. It requires careful measurement and does not exclude some having the exact same shape [26].

Therefore, even if the specimen maintains its integrity during extraction and preparation for culturing in cells and is centrifuged and filtered perfectly, it would still be nearly impossible to look at the particles under an electron microscope and say with certainty that it is SARS-CoV-2. In other words, it could be SARS-CoV-2, or it could be other viruses, or it could be a mix of SARS-CoV-2 and other viruses, or it could be exosomes and other viruses, etc.

This problem was revealed in March 1997 concerning HIV, when two papers published in the same issue of the journal "Virology" revealed that the vast majority of what had previously been called "pure HIV" was impurities that were clearly not HIV. What was being observed included micro-vesicles that look very similar to HIV under an electron microscope, but are of cellular origin [27][28].

And all of this is just to fulfill steps 1 to 3 of Koch's postulates. Even if the first 4 steps of isolating viruses is done carefully, it still doesn't prove causation of disease. But causation is really a different issue altogether.

Problems with Step 5 and Discovery of SARS-CoV-2

Breaking down the proteins and genetic material (RNA or DNA, depending on the virus) in the rest of the sample and analyzing them is a very important step. If it is done successfully, the genome can be sequenced and parts of the genome can then be used in research that uses PCR technology to help identify the virus in other specimens.

However, this step is also problematic for several reasons. In order to analyze genetic material to sequence its genome, it is first necessary to actually isolate the material according to the gold standard. The problem here is that it even if the gold standard is followed perfectly, it is still very difficult to ensure the integrity of the specimen for the reasons described above. Problems simply magnify themselves. For example, a problem in Step 1 will carry over into Step 2, which could create more problems, and then carry over into Step 3, and so on.

In the case of trying to identify a new virus such as SARS-CoV-2, an even greater problem exists. Where should we start? How can we go about trying to detect a new virus? The interesting thing about SARS-CoV-2 is that its whole genome was sequenced in record time. The Lancet reported that "The whole-genome sequence of SARS-CoV-2 had been obtained and shared widely by mid-January, a feat not possible at such speed in previous infectious disease outbreaks" [29]. This is truly amazing, but at the same time, it makes us suspicious. How did they do it so quickly? We will explain how they did it and show how problematic their method was.

In order to obtain genome of virus, it would be necessary to isolate and purify the virus, and then verify that there is no contamination. After this, the structure of the viral particles would need to be chemically characterized. This would include extracting the genetic material and performing a full sequence of the genetic material to obtain the entire genome.

As we shall see in Section 4, this procedure has not been done for SARS-CoV-2. Many studies allege to have isolated it, but not according to the gold standard. Instead, what scientists did was take an impure sample, such as lung fluid, and used PCR primers and probes to look for specific sequences that they already identified as being related to viruses (although these identified viruses were also not purified appropriately, as we shall see later in the case of SARS-CoV-1 and MERS-CoV). They then used the results of the PCR tests to reconstruct bits of DNA and combine them into a model that they say represents the genome or the entire set of genetic material of this virus.

However, since they didn't obtain SARS-CoV-2 from a purified viral particle, there is no way to know exactly what the source of that genetic material is. Most of the genetic material that would be present in the lung fluid of an ill individual would be the individual's own genetic material, as well as other microorganisms that normally live in the body or were involved in the infectious processes of such people. As mentioned before, there are literally millions of unknown viruses living harmlessly in the human body at all time. So, the genetic material from the sample specimen could easily include a whole slew of sources other than the alleged virus SARS-CoV-2. We just don't really know because it was never obtained from something that was obtained directly from a purified virus. Therefore, the PCR test is measuring these RNA strands of unknown significance.

We will now give an example of just how problematic this is. One of the WHO's primer sequences in the PCR test for SARS-CoV-2 is found in all human DNA. The sequence "CTCCCTTTGTTGTGTTGT" is an 18-character primer sequence found in the WHO coronavirus PCR testing protocol document [30]. The primer sequences are what get amplified by the PCR process in order to be detected and designated a "positive" test result. It just so happens this exact same 18-character sequence is also found on Homo sapiens chromosome 8 [31]. Therefore, the genetic material testing positive in the tests is actually found in all people naturally. Just think about all of the genetic material in the body that is unknown. How about those millions of unidentified harmless viruses in the human body? Isn't it possible that they might be detected, too? Of course, some people would probably say that it doesn't matter because DNA is zipped unlike RNA. But DNA is known to unzip during the PCR replication process. All it would take would be one exposed section of unzipped DNA to cause the PCR probes to connect. The possibility for bad results just seems nearly innumerable. This is a serious problem.

Finally, scientists allege that SARS-CoV-2 is a coronavirus from the same subgenus as SARS coronavirus (SARS-CoV-1) and other bat coronaviruses [32]. However, as we shall see in Section 3, SARS-CoV-1 is a

virus for which they used the same faulty procedures on (i.e., not purifying it and not verifying its integrity) to say that it was responsible for the SARS outbreak that took place in 2002 and 2003. So, they basically looked at this SARS-CoV-2 sequence that they reconstructed using this faulty method and compared it to the sequence that they reconstructed using the same faulty method for the SARS-CoV-1 virus and said that they are almost 80% identical, and therefore, they are part of the same coronavirus subgenus.

It should be clear now why even Thomas Rivers was not completely confident about the whole process [12]. We can only imagine that he would be even less confident if he were still alive to witness these genome sequencing methods.

Can accurate tests be developed if the virus is not isolated according to the Koch's postulates?

No, they cannot. As mentioned above, Step 5 of the Method for Isolating Viruses is an important step for developing accurate tests. As we shall see in Section 4, SARS-CoV-2 has not yet been isolated according to the gold standard. As a result, the accuracy of the tests for detecting virus is highly questionable [5].

For example, manufacturers of SARS-CoV-2 antibody test kits indirectly admit that the SARS-CoV-2 has not been isolated according to the gold standard by their use of recombinant antigen proteins instead of purified viral antigen proteins [73].

To sum up, accurate tests can only be developed after acquiring pure proteins, RNA, or DNA from the isolated virus. This ensures that the tests are actually testing for the viral materials. Furthermore, isolation according to the gold standard is the only way to validate tests once they are developed.

Don't virologists often claim to have isolated viruses?

*The content for this sub-section was taken from researcher David Crowe's article on isolation and purification [16].

Yes, they do. But their meaning of isolation is quite different from what Robert Koch had in mind when he formulated his postulates. Isolation according to Koch's postulates actually refers to purification. Virologists almost never use the word purification.

Virologists must know that the common definition of isolation and purification are virtually identical. For example, according to the Oxford English Dictionary:

Isolation: "The action of isolating; the fact or condition of being isolated or standing alone; separation from other things or persons; solitariness."

Purification: "Freeing from dirt or defilement; cleansing; separation of dross, dregs, refuse, or other debasing or deteriorating matter, so as to obtain the substance in a pure condition."

One can argue about subtleties, but if you took some ore and isolated gold, it would be the same as purifying gold. But with viruses, virologists have completely debased the word "isolation" while rarely using the word "purification."

We shall see how far they have debased the word isolation in the upcoming two sections.

Are there any general criticisms of the work being done by virologists and the papers they publish?

Yes, there are. All virologists adhere to what is called germ theory, a theory that states that microorganisms known as pathogens or “germs” can lead to disease. This adherence to germ theory greatly influences their work and often causes them to lose their objectivity. In other words, they have a bias and they perform their experiments and write their papers with the intent of confirming that bias.

Here are few general criticisms of the whole experiment and paper publishing process:

1. The authors of the papers are the ones responsible for providing the data and materials. They know there is no point in submitting data that would significantly contradict their claims. This means that they can spend time adjusting their experiments and perform their analysis in a way that ensures that their papers achieve their goal. All of the peer reviewers hold the same fundamental biases as the authors of the papers. In other words, the peer reviewers are only objective critics within their common framework of scientific principles. They are unable to think outside that common framework.

2. There is no way for the readers of the papers to validate the work of the authors. Readers are forced to trust the authors and peer reviewers.

3. The photos included in the papers will certainly be the “best” photos. These photos are usually the ones with the greatest number of viral particles or the ones that show the most cytopathic effects (i.e., cell death or structural change alleged to be caused by an invading virus). Lab technicians can literally spend hours looking around to find the most photogenic images, while ignoring photos that are disadvantage to achieving their goal.

4. Most papers seem to regard the occurrence of cytopathic effects (CPE) in cells cultured with impure materials (rather than with purified virus obtained through the method for isolating viruses described above) to be equivalent to virus isolation. However, observing CPE in cells cultured with impure materials proves nothing. The CPE could have been caused for various reasons. For example, Thomas Rivers, in his 1936 speech, stated that “Viruses, regardless of whether they are parasites or the fabrications of autocatalytic processes, are intimately associated with host cells and, therefore, should always be found at the proper time in specific lesions” [12]. This is basically an acknowledgement that CPE could potentially be the result of internal processes, such as apoptosis, rather than invasion of foreign microbes. Likewise, CPE could simply be due to induced stress and the impure and artificial environment created to culture the cells. Also, when reading the papers, it is often the case that CPE is only present for a portion of the cultured cells. As mentioned above, lab technicians can literally spend as much time as they want searching for CPE that best supports their hypothesis, while ignoring other cultured cells altogether.

5. Simply asserting that there is a new virus because CPE was observed in cells cultured with impure materials does not make it true. Furthermore, simply publishing the genome of that alleged new virus does not make it a valuable contribution to the world. Virologists seem to think that if they just assert something, it becomes true based on their authority as so-called experts, despite their experiments not being scientifically or logically sound. As mentioned before, there are countless unknown microorganisms in the body that are capable of altering their genetic sequences and forming new sequences due to shocks or stress. Certain expressions of illness can potentially produce certain types of new genetic sequences in people’s bodies. Some of these genetic sequences might even appear to be related to each other. We suppose with the right equipment and enough time, countless numbers of new so-called virus families could be found and new PCR tests could be developed based on the genomes of the viruses. But none of them would be caused by invading microbes that adhere to the germ theory. The moral of the story is this: Do not believe virologists simply because they assert things and create databases of gene sequences.

Section 3

In this section, we will examine studies claiming to have isolated SARS-CoV-1 (i.e., Hong Kong's SARS epidemic) and MERS-CoV. Both of these viruses are coronaviruses. These studies will provide us with important precedents for when we examine the studies on SARS-CoV-2.

Study 1: Koch's postulates fulfilled for SARS virus [31]

*Nearly all of the content for Study 1 was taken from researcher David Crowe's book on SARS [34].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Aetiology

Koch's postulates fulfilled for SARS virus

Ron A. M. Fouchier, Thijs Kuiken, Martin Schutten, Geert van Amerongen, Gerard J. J. van Doornum, Bernadette G. van den Hoogen, Malik Peiris, Wilina Lim, Klaus Stöhr & Albert D. M. E. Osterhaus

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https://www.nature.com/articles/423240a

In May 2003, the journal Nature allowed Dutch researchers to claim "Koch's postulates fulfilled for SARS virus" by allowing the phrase in the title. We will see that this was clearly not true and provides simply amazing evidence that the claims of virologists are not examined critically, but simply accepted on the basis of faith or consensus.

In the paper, the researchers say the following:

"According to Koch's postulates, as modified by Rivers for viral diseases, six criteria are required to establish a virus as the cause of a disease. The first three criteria — isolation of virus from diseased hosts, cultivation in host cells, and proof of filterability — have been met for SCV [SARS-associated coronavirus] by several groups."

In particular, the researchers refer to four groups who claim to have met the first three postulates. We shall now examine these four studies to see if they indeed met the first three postulates.

#1 – Carlo Urbani Paper [35]


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ORIGINAL ARTICLE

A Novel Coronavirus Associated with Severe Acute Respiratory Syndrome

Thomas G. Ksiazek, D.V.M., Ph.D., Dean Erdman, Dr.P.H., Cynthia S. Goldsmith, M.S., Sherif R. Zaki, M.D., Ph.D., Teresa Peret, Ph.D., Shannon Emery, B.S., Suxiang Tong, Ph.D., Carlo Urbani, M.D., James A. Comer, Ph.D., M.P.H., Wilina Lim, M.D., Pierre E. Rollin, M.D., Scott F. Dowell, M.D., M.P.H., et al.

Article Figures/Media

33 References 1823 Citing Articles Letters

May 15, 2003

N Engl J Med 2003; 348:1953-1966

DOI: 10.1056/NEJMoa030781

Abstract

Related Articles

BACKGROUND

A worldwide outbreak of severe acute respiratory syndrome (SARS) has been associated with exposures originating from a single ill health care worker from Guangdong Province, China. We conducted studies to identify the etiologic agent of this outbreak.

METHODS

PERSPECTIVE MAY 15, 2003

SARS-Associated Coronavirus

K.V. Holmes

PERSPECTIVE MAY 15, 2003

Managing SARS amidst Uncertainty

https://www.nejm.org/doi/10.1056/NEJMoa030781

One of the papers from the May 15, 2003 issue of the New England Journal of Medicine included the deceased Carlo Urbani as a co-author although he likely was not alive to have written a word of it. The authors claim in their abstract that, “a novel coronavirus was isolated from patients who met the case definition of SARS” and, in addition, that the virus was shown to kill cells because, “Cytopathological features [evidence of diseased cells] were noted in Vero E6 cells inoculated with a throat-swab specimen”. They also claim that electron microscope pictures show particles typical of coronaviruses and that a stable portion of the “consensus” coronavirus genome was used to extract the RNA of this novel virus. Based on this they claimed that, “the evidence indicates that this virus has an etiologic [causative] role in SARS”.

These authors must have hoped that nobody would ever critically read their research and discover just how thin the evidence was, and how flawed their experiments. They imply that they isolated this coronavirus from many of the nineteen people they had samples from. Actually they only achieved isolation in samples from four of those people, but isolation was according to their own definition and not according to Koch’s postulates. Basically, they simply took a throat swab, added the impure material to cell culture, noticed cell death, and called it isolation. Again, this is not isolation according to the gold standard set forth in Section 2 of this paper. Furthermore, in two of the cases where they had multiple samples from different parts of the body, only one from each of the cases resulted in so-called isolation.

Antibody tests were not much more successful, only 7 of 17 people tested produced positive results. Their conclusions relied heavily on the genetic PCR (Polymerase Chain Reaction) test for the coronavirus genome even though this is not, by their standards, isolation. PCR was, however, positive in 15 out of 17 samples tested.

They achieved 100 percent correlation by lumping the three tests together. The two people for which PCR was negative and the two for which PCR was not done were also negative by isolation but did have positive antibody tests. Bingo! Everyone with SARS was now positive, although nobody was positive on all tests.

Remember this trick - you will soon come across it again.

#2 - German Study [36]

https://www.nejm.org/doi/full/10.1056/NEJMoa030747

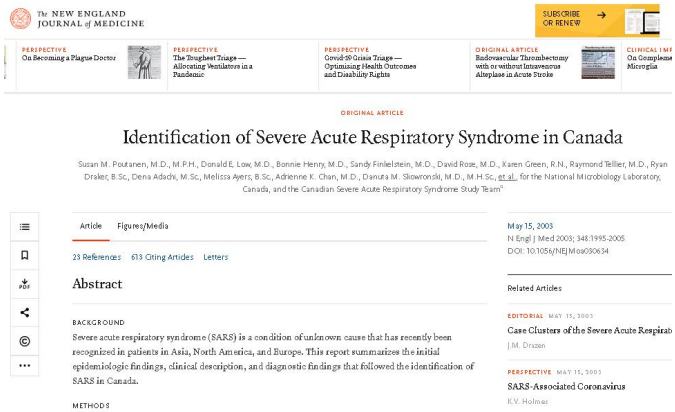
Another New England Journal of Medicine by a group of German scientists made the modest claim that “The novel coronavirus might have a role in causing SARS.” After developing a genetic test based on material fished out of cell cultures (not from purified virus) they claimed that all five patients with probable SARS tested positive on samples mostly taken from their lower respiratory tracts. So did three of thirteen with probable SARS, but for these people they used samples of mucus from their noses. They also found that none of 21 healthy contacts were positive, but for these people they used fecal samples.

The accuracy on a very small number of people with or without SARS is impressive, but if the test is accurate for “probable SARS” it indicates that doctors had been severely over-diagnosing the condition. This is a common problem that occurs with diagnostic tests, when there is a disagreement between two tests or between a test and clinical diagnosis, there is no ‘gold standard’ to sort it out, no completely accurate yardstick by which to compare the two competing methods. Often diagnostic tests win out over diagnosis

because a test seems more scientific and less biased, but that does not mean that they are necessarily more accurate.

A clearer problem occurred when they studied a number of samples from the patient from whom the test was developed and less than half of the samples were positive. Of the two cases blamed on close contact with him, one person was negative on all samples that were genetically tested, and the other was negative on 16 out of 21 tests.

#3 - The Canadians [37]



https://www.nejm.org/doi/full/10.1056/NEJMoa030634

Canada, which had sacrificed the economy of Toronto to SARS, obviously did not want to get left out of the scramble to claim the discovery of the cause of this disease. In the third New England Journal of Medicine paper, scientists from Toronto and Vancouver studied samples from the first ten cases diagnosed in Canada. They were unable to detect any virus by viral culture, electron microscopy and a number of specific viral tests, but they did detect genetic material that they claimed matched a human metapneumovirus and a new coronavirus. The coronavirus was detected using probes from scientists in Hong Kong and at the US Centers for Disease Control although only five out of the nine patient samples were positive on this test. At another laboratory, tests were performed on samples from four of the patients and two of those were positive. It is hard to understand how this paper was described as meeting any reasonable criteria for "isolation of virus from diseased hosts, cultivation in host cells, and proof of filterability".

#4 - The Hong Kong Paper [38]

https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(03)13077-2/fulltext

The last paper referenced by the Dutch researchers in Nature was published in the prestigious British medical journal Lancet whose editors claimed that it provided, "strong evidence that SARS is associated with a novel coronavirus" which, if read carefully, is clearly not a claim that the coronavirus is the cause of SARS, just a claim that they often associate together.

Unfortunately, this paper's authors, all from Hong Kong hospitals, also used tricks to try to boost a weak association to the level of a causative association. They found the RNA that they believed was from a new coronavirus in only half the nose swab specimens tests (22 out of 44) and in just over half (10 of 18) of the stool samples from fifty Hong Kong patients. They also found antibodies in 35 out of 50 samples (but

because at least 32 of the 50 patients had at least two antibody tests, this actually only represents about half of the total number of patients).

Even after lumping all three tests together, and taking a leap of faith, they only claimed hesitantly that, "If seropositivity to human pneumonia-associated coronavirus in one serum sample or viral RNA detection in the nasopharyngeal aspirates or stools is deemed evidence of infection with the coronavirus, 45 of the 50 patients have evidence of infection."

#5 – The Last Three Postulates [33]

The Dutch scientists in Nature claimed to fulfill the last three of the six conditions to prove causality using two (2) macaques. The monkeys were inoculated with impure materials taken from a cell culture believed to contain the SARS coronavirus. The paper does not indicate whether the materials were put into the macaques directly into the brain (as is often done), under the skin or, more realistically, into their noses, and did not respond to requests for clarification. Another limitation is that the animals were only monitored for six days before being killed.

One of the two macaques suffered "respiratory distress" but the other only suffered from a skin rash and tiredness. The scientists claimed to have detected viral RNA in their nose and throat two to six days after the injection. Antibodies were found in two other macaques who were allowed to live for 16 days, although no information on the health of these monkeys was provided. An autopsy of the two macaques found severe lung disease in one of them (presumably the one suffering from respiratory distress) and unusual large cells (synctitia) that they claimed were indistinguishable from those found in lung tissue of human SARS victims.

The scientists claimed that the virus cultured from the monkeys was visually identical to the virus inoculated into them, but only provided the 'after' electron microscope photograph, not the 'before' picture. This data was used to support the claim that they had produced a comparable disease to SARS (although they only claimed that they had done this in one of the four macaques), that they had re-isolated the virus, and that they had detected a "specific immune response" (but not in the one monkey that had the SARS-like disease).

There were no controls in this study, no macaques injected with materials produced in the same way differing only in the absence of the virus. There was no evidence provided that the monkeys were exposed to the virus in the same way that humans must be. And there was no consideration that injecting foreign cells and the chemicals used to culture them into a monkey could cause allergic or other toxic reactions, perhaps the source of the rash that both monkeys experienced.

Their claim of 'isolation' is based on a debased definition used by virologists, bearing no resemblance to the dictionary or chemical definitions which mean to separate one thing from everything else, derived from the Latin/Italian word 'isola' meaning island. They claimed to have pictures of the virus, but without purification there is no way to tell if the particles shown under the electron microscope are actually the source of the RNA that is claimed to be from a coronavirus. There is no way to know, in fact, just from electron micrographs, that the particles are even a virus at all.

The sloppiness of the coronavirus science is hard to understand until you realize that the search never really was for the cause of SARS, it was for the virus most closely associated with SARS, even if the association wasn't very good.

Significance of this study

It should be obvious now from the above analysis that SARS coronavirus was not isolated (i.e., purified) according the gold standard. SARS coronavirus was not shown to be the causative agent of disease. Despite this, scientists performed gene sequencing on the RNA of the alleged SARS virus. This basically enabled them to acquire the genome of an impure piece of genetic material that tends to be associated with disease. It was never proved to be a pathogenic virus. Yet, this is the genome that is used to create the RT-PCR test kits to determine if someone is infected with the virus. It should be strikingly clear how problematic this is.

Furthermore, SARS-CoV-2 (i.e., the virus said to cause COVID-19) was identified as a coronavirus from the same subgenus as SARS coronavirus and other bat coronaviruses [35]. In other words, SARS-CoV-2 is being

grouped together with SARS coronavirus, despite SARS coronavirus having never been purified. We shall soon seen that SARS-CoV-2 has also never been purified according to the gold standard. So, basically, we have unscientific experiments becoming the basis for more unscientific experiments. We hope the reader can see how dangerous this is.

Study 2: Isolation of a Novel Coronavirus from a Man with Pneumonia in Saudi Arabia [40]

https://www.nejm.org/doi/full/10.1056/NEJMoa1211721

The authors of this study begin by saying that "[HCoV-EMC (i.e. MERS-CoV)] was remarkably similar to that of the severe acute respiratory syndrome (SARS) outbreak in 2003 and reminds us that animal coronaviruses can cause severe disease in humans." So, here they are implying that HCoV-EMC causes severe disease in humans, but at the end of their paper, they contradict themselves by saying, "It will be equally important to test whether HCoV-EMC fulfills Koch's postulates as the causative agent of severe respiratory disease." In other words, they admit that they did not prove causality, but still imply that HCoV-EMC can cause severe disease. This is unacceptable, especially given the fact that this was one of the first attempts to isolate the virus. Their study influenced other studies, such as the Korean study that we will be examining shortly. Furthermore, by not being careful with the words they choose, they are liable to create rumors. These kind of rumors can lead to drastic public policy that turns society upside down. We shall see in Section 4 that this is exactly what has happened with SARS-CoV-2.

Looking at the content of the study, they did centrifuge the sample before inoculating Vero and LLC-MK2 cells. As mentioned in Section 2, antibiotics and/or serums are often added to the cell cultures, and this study was no exception. The researchers mention that they added 2% fetal bovine serum. After this, they incubated the flasks and observed them daily for 15 days for cytopathic changes (i.e., cell changes allegedly caused by a pathogenic virus) while changing the medium every 3 days. Note here that they changed the medium with the serum every 3 days. All of this increases the amount of induced stress on the specimen and can be a contributing factor to the cytopathic effects.

After detecting some cytopathic effects in some of the cell cultures, they were prepared for RT-PCR testing. During this process, they do mention that they centrifuged and filtered the virus, but add the important admission that this was "to minimize bacterial background." This is further proof of the shortcoming we mentioned in Section 2. Centrifuge and filtering, even if done carefully, are not bulletproof methods for isolating a virus. As noted before, centrifugation is susceptible to contamination and filtering cannot exclude other viruses the same size or smaller. Furthermore, there are no descriptions or photos at all to indicate that the researchers used electron microscopy to verify their work after filtering.

After this, the authors proceeded to sequence the genome. The interesting thing is how they did this. Since they did not have purified virus, they were not able to chemically characterize the structure of the viral particles to extract its genetic material and sequence its genome. Instead, they used a method called arbitrarily primed PCR (AP-PCR), despite this method not even being designed for this type of work [72]. Regardless, they found sequences of DNA that they later assembled into contig maps (a set of overlapping DNA segments). They said that they were able to obtain approximately 90% of the virus genome sequence.

In summary, the authors admit that they did not fulfill Koch's postulates, despite using the very suggestive word "cause." Furthermore, their isolation procedures did not satisfy the method for isolating viruses in

Section 2. They did do centrifugation and filtering, but it is clear that they were not able to overcome the shortcomings mentioned in earlier.

Study 3: Infectious MERS-CoV Isolated From a Mildly Ill Patient, Saudi Arabia [41]

Volume 5, Issue 6
June 2018

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Infectious MERS-CoV Isolated From a Mildly Ill Patient, Saudi Arabia

Hail M Al-Abdely, Claire M Midgley, Abdulrahim M Alkhamis, Glen R Abedi, Azaibi Tamin, Alison M Binder, Khalid Alanazi, Xiaoyan Lu, Osman Abdalla, Senthilkumar K Sakthivel ...
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Open Forum Infectious Diseases, Volume 5, Issue 6, June 2018, ofy111,
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Published: 15 May 2018 **Article history**

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Abstract

Middle East respiratory syndrome coronavirus (MERS-CoV) is associated with a wide range of clinical presentations, from asymptomatic or mildly ill to severe respiratory illness including death. We describe isolation of infectious MERS-CoV from the upper respiratory tract of a mildly ill 27-year-old female in Saudi Arabia 15 days after illness onset.

Keywords: asymptomatic, MERS, mild, prolonged detection, virus isolation
Topic: saudi arabia, middle east respiratory syndrome coronavirus, middle east respiratory syndrome

https://academic.oup.com/ofid/article/5/6/ofy111/4996435

Saudi Arabian researchers in collaboration with the United States CDC claim to have isolated MERS-CoV from a female patient.

In the “Clinical Testing in Respiratory Specimens” section of the study, they try to correlate RT-PCR cycle count with viral load. They say, “Cycle threshold (Ct) values of MERS-CoV upE rRT-PCR were available for 11 of the 12 positive specimens and ranged from 25 to 37 (Figure 1B). On day 10 after onset, the upE Ct value was 37, indicating a low viral load; by day 14, the Ct value was much lower, at 25, indicating a notable rise in viral load.”

However, there are several studies available that show the cycle count is not indicative of viral load. For example, one study concludes by saying “Although correlations between CT values and clinical parameters in patients with single and multiple viral infection were found, the clinical importance of these findings is limited because individual differences in host-, viral and laboratory factors complicate the interpretation of statistically significant findings. In multiple infections, viral load cannot be used to differentiate between disease causing virus and innocent bystanders” [42]. Another study on this topic says that “Ct values from the qRT-PCR of upper respiratory tract specimens were associated with clinical severity for some respiratory viruses” [43]. This sounds good until one reads the study and realizes that association was only found for one out of five studied viruses. In one instance, outpatients had lower Ct values than inpatients, a result that is completely the opposite to the the researchers’ hypothesis.

As far as Ct values being used to indicate viral load for SARS-CoV-2, researcher David Crowe has analyzed a study from Singapore where scientists found that the sickest patients had Ct values that were entirely comparable with patients who only had mild symptoms [44]. Also, in a survey of RNA-positive people in Guangdong, China, scientists examined the ‘viral load’ (quantity of RNA) and concluded that “the viral load that was detected in the asymptomatic patients was similar to that in the symptomatic patients” [45].

From these studies, it seems apparent that Ct values cannot be used to estimate viral load, at least not consistently.

Getting back to the MERS-CoV study, researchers proceeded to isolate the virus. They say, “ The 2 MERS-CoV RNA-positive NP specimens submitted to the CDC (collected on days 13 and 15) were serially diluted 10-fold in DMEM in a 96-well plate, and subsequently used to inoculate Vero cell suspensions. The cells were observed daily between days 3 and 7 postinoculation; cytopathic effect (CPE) was observed under inverted scope 3 days postinoculation. Any wells that exhibited CPE were harvested and passaged in a 24-well plate.”

There are some points to mention here. First, they used Vero cells (i.e., monkey kidney epithelial cells) instead of host cells. Thomas Rivers said that the cells should be obtained from patients with the natural disease [12]. Second, it is unclear whether they added antibiotics to the culture, but this seems to be common practice, so it is likely that they did. This would have increased the amount of induced stress described in Section 2. Third, the authors make no mention of negative controls. However, negative controls are necessary to help show that it is not the lab procedures, such as preparing the culture and adding the serum, that are responsible for any cytopathic changes that may occur.

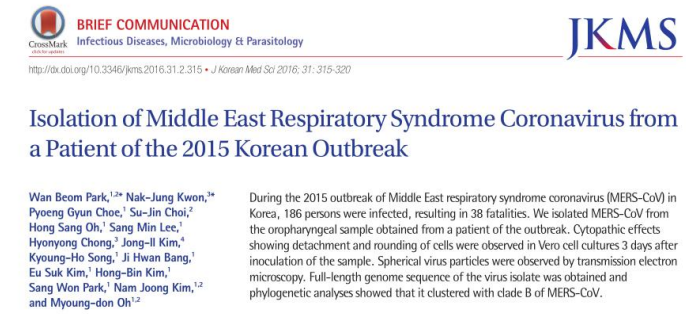
In general, their method of isolating a virus sounds very similar to the Dutch researchers method who claimed to have isolated SARS. Their method does not comply with Koch's postulates.

Basically, they added the impure material (i.e., not purified virus) to cell culture, noticed cell death, and called it isolation. But since impure materials were added to the cell culture, it is impossible to know exactly what caused the cell death. It could have been the virus, but it could have been something else. Without first isolating (i.e. purifying) the virus according to the gold standard, there is no way to know for sure what caused the cell death.

Next, they proceeded to sequence the RNA and found that the 2 isolated viruses had identical sequences as the clinical specimens. Basically, they are saying that the same RNA was obtained at the end of the process that they had put in at the beginning. But what exactly does this prove? Since they cultured the Vero cells with samples that contained the RNA, it isn't very surprising that they would find the same RNA later. The presence of the RNA can provide no more than evidence of association. It cannot prove causation. Again, without purifying the virus, it is unclear whether the RNA is viral or simply endogenous to the cells.

In conclusion, the authors of the study claimed to have isolated the MERS-CoV, but on examining their work, it is clear that they did no such thing.

Study 4: Isolation of Middle East Respiratory Syndrome Coronavirus from a Patient of the 2015 Korean Outbreak [46]



BRIEF COMMUNICATION
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Isolation of Middle East Respiratory Syndrome Coronavirus from a Patient of the 2015 Korean Outbreak

Wan Beom Park,[1,2*] Nak-Jung Kwon,[3*] Pyoeng Gyun Choe,[1] Su-Jin Choi,[2] Hong Sang Oh,[1] Sang Min Lee,[1] Hyonyong Chong,[3] Jong-Il Kim,[4] Kyoung-Ho Song,[1] Ji Hwan Bang,[1] Eu Suk Kim,[1] Hong-Bin Kim,[1] Sang Won Park,[1] Nam Joong Kim,[1,2] and Myoung-don Oh[1,2]

During the 2015 outbreak of Middle East respiratory syndrome coronavirus (MERS-CoV) in Korea, 186 persons were infected, resulting in 38 fatalities. We isolated MERS-CoV from the oropharyngeal sample obtained from a patient of the outbreak. Cytopathic effects showing detachment and rounding of cells were observed in Vero cell cultures 3 days after inoculation of the sample. Spherical virus particles were observed by transmission electron microscopy. Full-length genome sequence of the virus isolate was obtained and phylogenetic analyses showed that it clustered with clade B of MERS-CoV.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4729515/pdf/jkms-31-315.pdf

The Korean authors of this study start out by saying "[MERS-CoV] was first isolated from the sputum of a patient with severe pneumonia in Saudi Arabia in 2012 (3)." Checking footnote 3, we are referred to the study "Isolation of a Novel Coronavirus from a Man with Pneumonia in Saudi Arabia" [40], which we examined in Study 2. Remember what we said about rumors starting from imprecise use of terminology? The authors of that study used the word "cause" even though they admitted to not fulfilling Koch's postulates. We are not sure to what extent the Korean researchers examined that study, but it surely influenced them.

As for allegedly isolating the virus, they simply took the patient's oropharyngeal samples, inoculated monolayers of Vero cells with the samples, and waited to see if there was any cytopathic effects. In the electron microscope photos, they do show photos of Vero cell cultures of negative control, but there is no information regarding how the cultures were prepared. It is unclear whether the negative control was exactly the same as the specimen, lacking only the oropharyngeal sample. For example, did the specimen and negative control both receive antibiotics and/or additives such as serums?

After 3 days of inoculation, some cytopathic effects were observed. This was basically the extent of what they called isolation. It seems that they completely skipped the centrifugation and filtration.

As for the cytopathic effects, we mentioned before in Section 2 that it could be due to a number of reasons other than an alleged virus. It could be due to the addition of impure materials, the laboratory procedures, induced stress, etc. Just to assume it is a pathogenic virus is not good science.

After this, they seemed to have used RT-PCR testing to find some RNA that apparently corresponded to the upE gene and ORF1a gene of MERS-CoV. But as mentioned before, MERS-CoV was never isolated properly to begin with, so it is impossible to say with any certainty what they detected.

And this was the extent of their isolation work.

Following this, they examined what they called viral particles using an electron microscope. They said, "spherical particles ranging 77 to 131 nm in diameter were observed within the cytoplasm of infected cells." That is quite a broad range of particles. Were they really all the same, just different sizes? Are we just to assume that they are viral? Is it impossible that they could be endogenous to the cells? None of these questions were considered.

The study makes much of the scientist's full-length genome sequencing of the virus. But their method simply follows the same faulty procedures discussed in the "Problems with Step 5 and Discovery of SARS-CoV-2" of Section 2. What they basically did was use PCR primers and probes on the specimen to look for specific sequences that were already identify as being related to MERS-CoV. They then used the results of the PCR tests to reconstruct bits of DNA and combine them into a model that they say represents the genome or the entire set of genetic material of this virus. The primer pairs that they used are shown in Table 1 of their study.

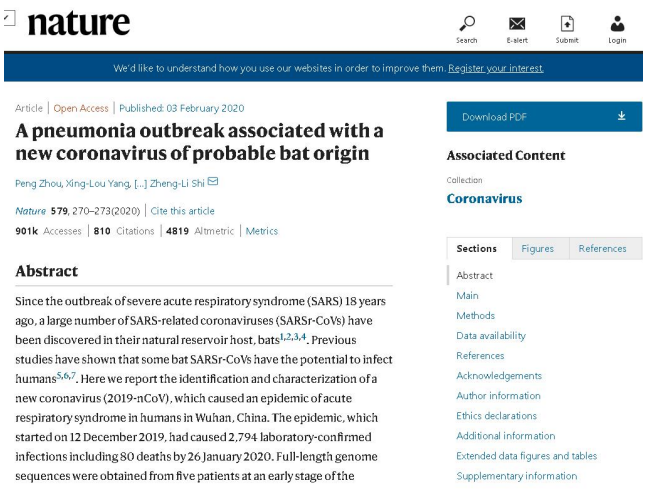
Remarks

We have now finished laying the groundwork that we needed before examining the studies on SARS-CoV-2. Just to sum up, we have (1) identified the gold standard for determining the cause of infectious disease; (2) learned the method for isolating viruses; (3) discussed some of the shortcomings and general criticisms of the method for isolating viruses; and (4) have examined several studies related to SARS-CoV-1 and MERS-CoV. As we take a look at the studies on SARS-CoV-2, we suggest that the reader review the previous material as necessary to help facilitate understanding.

Section 4

In this section, we will examine studies claiming to have isolated SARS-CoV-2 (i.e., the virus alleged to cause COVID-19).

Study 1: A pneumonia outbreak associated with a new coronavirus of probable bat origin [47]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A pneumonia outbreak associated with a new coronavirus of probable bat origin

Peng Zhou, Xing-Lou Yang, [...] Zheng-Li Shi

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Abstract

Since the outbreak of severe acute respiratory syndrome (SARS) 18 years ago, a large number of SARS-related coronaviruses (SARSr-CoVs) have been discovered in their natural reservoir host, bats[1,2,3,4]. Previous studies have shown that some bat SARSr-CoVs have the potential to infect humans[5,6,7]. Here we report the identification and characterization of a new coronavirus (2019-nCoV), which caused an epidemic of acute respiratory syndrome in humans in Wuhan, China. The epidemic, which started on 12 December 2019, had caused 2,794 laboratory-confirmed infections including 80 deaths by 26 January 2020. Full-length genome sequences were obtained from five patients at an early stage of the

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https://www.nature.com/articles/s41586-020-2012-7

The authors of this study begin by saying open summary, "Here we report the identification and characterization of a new coronavirus (2019-nCoV) [SARS-CoV-2], which caused an epidemic of acute respiratory syndrome in humans in Wuhan, China." This is quite a claim! To cause an epidemic goes beyond simply being the causative agent of disease. Let's hope they have good proof of this.

Reading through their paper, it becomes apparent quickly that they have no proof of their claim. In fact, they contradict it by later saying, "The study provides a detailed report on 2019-nCoV, the likely aetiological [causative] agent responsible for the ongoing epidemic of acute respiratory syndrome in China and other countries." So, they went from "caused an epidemic" to "likely aetiological agent." That is quite a reversal. However, they are not finished. They go on to say that "The association between 2019-nCoV and the disease has not been verified by animal experiments to fulfil the Koch's postulates to establish a causative relationship between a microorganism and a disease." Here, they plainly state that they did not prove that SARS-CoV-2 is the causative agent of disease.

By saying "caused the epidemic" in the Abstract of their paper, the authors completely mislead their readers. It is outright carelessness. This was one of the first attempts to isolate the alleged virus, so their study influenced other studies, such as the Korean study that we will be examining shortly. Furthermore, by not being careful with the words they choose, they are facilitating the spread of rumors, and in the case of SARS-CoV-2, it is likely that these rumors were part of the cause of the drastic public policy that has turned society upside down.

Since the authors admit that they didn't fulfill Koch's postulates, this study cannot be used to prove that SARS-CoV-2 is the causative agent of a disease. Therefore, we can simply move onto the next study. However, for the sake of learning, let's examine the content of this study a little more.

The paper says, "qPCR analysis showed that the viral load [of sample specimens] increased from day 1 to day 3." But as we showed in Study 3 of Section 3, PCR tests are unable to consistently use their Ct value estimate viral load.

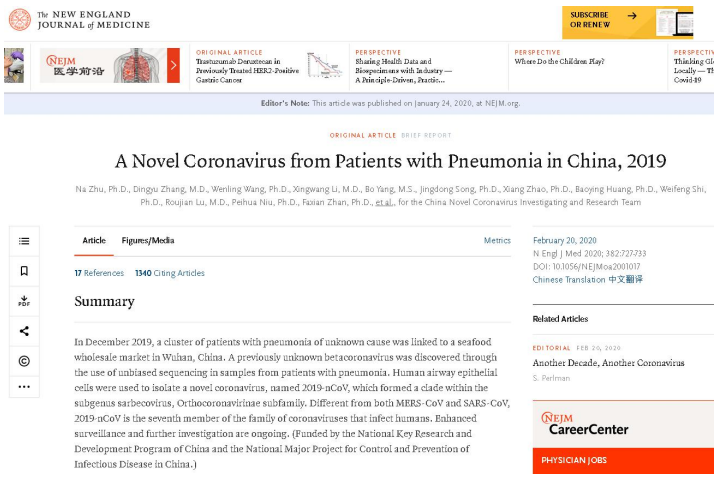
The study makes much of the authors' full-length genome sequencing of the virus. But their method simply follows the same faulty procedures discussed in the "Problems with Step 5 and Discovery of SARS-CoV-2" of Section 2. At one point they say, "We rapidly developed a qPCR-based detection method on the basis of

the sequence of the receptor-binding domain of the S gene, which was the most variable region of the genome." They are basically saying that this S gene is unique to SARS-CoV-2. But then they curiously make the admission, "Of the samples obtained from the seven patients, we found that six BALF [bronchoalveolar lavage fluid] and five oral swab samples were positive for 2019-nCoV during the first sampling, as assessed by qPCR and conventional PCR. However, we could no longer detect virus-positive samples in oral swabs, anal swabs and blood samples taken from these patients during the second sampling." In other words, their PCR test didn't prove to be very useful. To deal with this, they say, "we recommend that other qPCR targets, including the RdRp or envelope (E) genes are used for the routine detection of 2019-nCoV." The problem here is that the RdRp and E genes are not unique to the SARS-CoV-2. Their unique SARS-CoV-2 sequence proved to be useless, but that did not deter these scientists! Instead, they just recommended relying on non-unique sequences to find a unique virus. We are dumbfounded!

As far as trying to isolate the virus, they curiously only used samples from one of the seven patients. Basically, they simply took the patient's BALF samples, added antibiotics and serums to the samples, used the samples to inoculate Vero E6 and Huh7 cells, and waited to see if there were any cytopathic effects. Please refer to the shortcomings and general criticisms in Section 2 to learn why this method is so problematic.

The study also provides a disclaimer: " No statistical methods were used to predetermine sample size. The experiments were not randomized and the investigators were not blinded to allocation during experiments and outcome assessment." This is quite disappointing indeed, considering how much public policy was based on this paper.

Study 2: A Novel Coronavirus from Patients with Pneumonia in China, 2019 [48]



https://www.nejm.org/doi/full/10.1056/NEJMoa2001017

The authors of this study state clearly that "Although our study does not fulfill Koch's postulates, our analyses provide evidence implicating 2019-nCoV in the Wuhan outbreak." Since the authors admit that they didn't fulfill Koch's postulates, this study cannot be used to prove that SARS-CoV-2 is the causative agent of a disease. Therefore, we can simply move onto the next study. However, for the sake of learning, let's examine the content of this study a little more.

As far as trying to isolate a virus, the authors basically followed the same procedures as the previously studies we have examined. Basically, they simply took the patient's bronchoalveolar-lavage fluid (BALF) samples, added antibiotics and/or serums to the samples (i.e., added the viral transport medium), centrifuged the samples, inoculated human airway epithelial cells, and waited to see if there were any cytopathic effects. It is noteworthy that they didn't use Vero cells this time, but instead used epithelial cells taken from patients with lung cancer. This is significant because cancer cells are known to produce exosomes [49] that would look very similar to a coronavirus when examined with an electron microscope [26]. Refer to "Problems with Step 4" in Section 2 for more information on exosomes.

The authors did centrifuge the samples, but they did not filter them. They mention seeing coronavirus-like particles when performing electron microscopy, but since they skipped filtering and were using samples already susceptible to exosomal vesicles, it is impossible to say for sure what they were observing. Furthermore, bear in mind that all of the other shortcomings and general criticisms mentioned in Section 2 still apply to this process.

Like the previous study, the authors make much of sequencing the genome. But without virus purification, the sequenced genome is of limited use.

Study 3: Identification of Coronavirus Isolated from a Patient in Korea with COVID-19 [50]

Osong Public Health Res Perspect 2020;11(1):3-7

Osong Public Health and Research Perspectives

Journal homepage: http://www.kcdcphrp.org

Original Article

Identification of Coronavirus Isolated from a Patient in Korea with COVID-19

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ABSTRACT

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Objectives: Following reports of patients with unexplained pneumonia at the end of December 2019 in Wuhan, China, the causative agent was identified as coronavirus (SARS-CoV-2), and the 2019 novel coronavirus disease was named COVID-19 by the World Health Organization. Putative patients with COVID-19 have been identified in South Korea, and attempts have been made to isolate the pathogen from these patients.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7045880/pdf/ophrp-11-3.pdf

Researchers from the Korean CDC start off this study by stating, “Following the first outbreaks of unexplained pneumonia in Wuhan, China, in late 2019, a new coronavirus was identified as the causative agent in January 2020.” To back this claim, they provide a reference to the study “Full-genome evolutionary analysis of the novel corona virus (2019-nCoV) rejects the hypothesis of emergence as a result of a recent recombination event” [51]. However, in this paper, the authors do not deal with the topic causation at all. In fact, they clearly state in the conclusion of their paper, “The unique genetic features of 2019-nCoV and their potential association with virus characteristics and virulence in humans remain to be elucidated.” In other words, these authors did not even commit to association. In this respect, causation is the farthest thing from their minds. How could the Korean CDC have read this paper and concluded that a new coronavirus (i.e., SARS-CoV-2, which was tentatively named 2019-nCoV at the time of the study) was the causative agent of unexplained pneumonia. There are only three options:

(1) The Korean CDC used the wrong reference. But this seems unlikely for the following reasons:

a) The authors rather purposefully use the exact words “causative agent” four times in their study, as if this is the main point they want to emphasize.

b) The authors also refer to the works of Zhou P et al. and Zhu N et al. who both deny fulfillment of Koch’s postulates by stating respectively, “The association between 2019-nCoV and the disease has not been verified by animal experiments to fulfil the Koch’s postulates to establish a causative relationship between a microorganism and a disease” [47] and “Although our study does not fulfill Koch’s postulates, our analyses provide evidence implicating 2019-nCoV in the Wuhan outbreak” [48].

(2) The Korean CDC didn’t read the papers and just assumed that the papers supported their claim.

(3) The Korean CDC purposely set out to deceive their readers, thinking that nobody would check their work.

It is difficult to imagine that there could be any other options.

The study by the Korean CDC has completely disqualified itself right from the beginning. However, for the sake of learning, let’s examine the content of this study a little more.

As far as trying to isolate a virus, the authors simply acquired impure nasopharyngeal and oropharyngeal samples, added antibiotics and serums, inoculated Vero cells, and waited to see cytopathic effects. They apparently did no filtering. Please see the shortcomings provided in Section 2 for some of the reasons why this is so problematic.

They also state that “Virus replication was confirmed using real-time RT-PCR with RNA extracted from the cell culture medium. The Ct values were 14.40 and 18.26 for the nasopharyngeal and oropharyngeal samples, respectively, which were lower than the cycle threshold (Ct) values of 20.85 and 21.85 in the pre-inoculated samples.” However, as mentioned before, RT-PCR is not reliable for quantitative assessment. Ct values cannot be used to estimate viral load [42][43][44][45].

After this, they performed genome sequencing “using the synthesized cDNA and primers designed based on published SARS-CoV-2 DNA sequence.” In other words, they did not purify the virus and chemically characterize the structure of the viral particles to extract the genetic material and perform a full sequence to obtain the entire genome. They simply relied on previously published primers.

The authors show photos of electron microscopy and state that they observed Vero cells infected with SARS-CoV-2. But, as mentioned before, without virus purification, it is impossible to say for sure what is being observed under the electron microscope. It might be a pathogenic virus, but it could also be endogenous. There is no way to simply look at something under a microscope and assert with confidence that it is the exact virus you are looking for. This is not a minor point. Remember the HIV papers that we referenced in “Problems with Step 4” in Section 2 [27][28].

At the end of the study, the authors state that “Currently, the diagnosis of COVID-19 is based on gene detection via real-time RT-PCR.” But this is in contradiction to what the US FDA and Korean RT-PCR test kit manufacturers say. According to official sources, RT-PCR cannot be used as the sole basis for diagnosis of COVID-19 [5].

This has been a very disappointing study. The authors purposely use the words “causative agent” four times in the study without even the slightest bit of evidence to support that claim. In fact, the papers the authors reference outright deny causation.

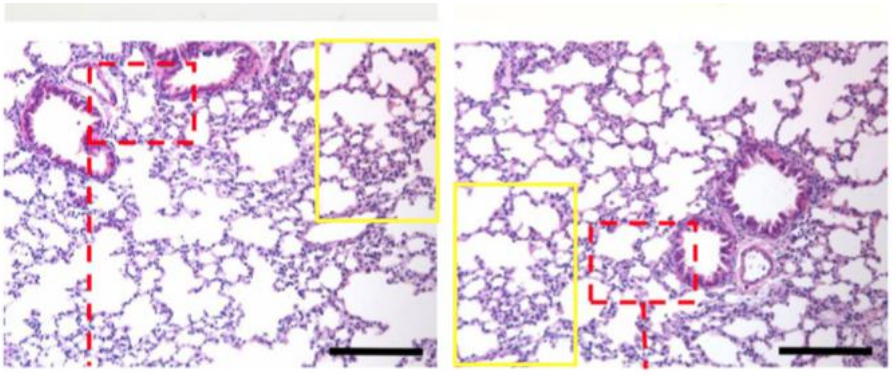
This study is either the product of the most extreme form of incompetence or is based on a willful desire to deceive its readers. We cannot help but think that this study was driven by political agenda. This study was prepared by a government agency and was most likely used to justify the creation of drastic public policies, including contact tracing, forced testing of asymptomatic people, criminal fines for refusing testing, and imprisonment for breaking forced quarantine. The people of South Korea and the world should demand that the Korean CDC be held accountable for this study.

Study 4: The Pathogenicity of SARS-CoV-2 in hACE2 Transgenic Mice [52]

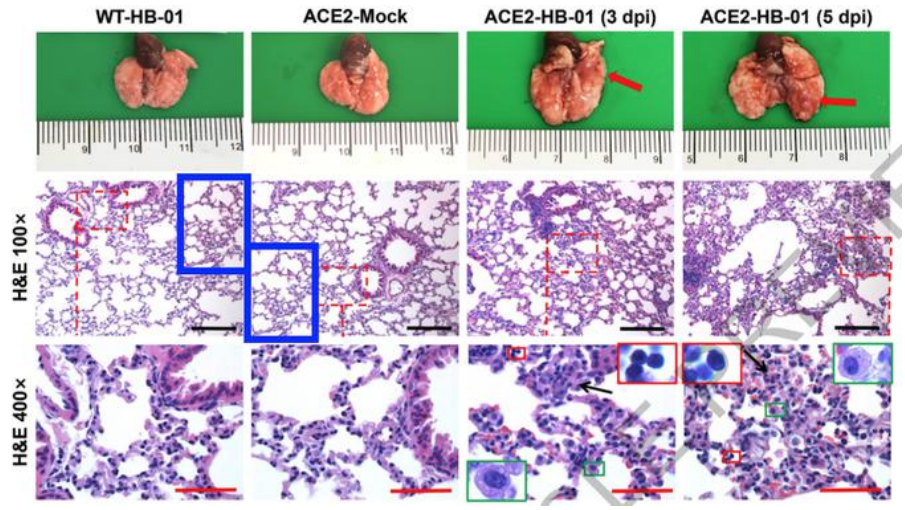
https://www.nature.com/articles/s41586-020-2312-y

This study is interesting because it actually claims to completely fulfill Koch's postulates when combined with previous studies. In particular, the authors' state that "Taken together, our results demonstrated the pathogenicity of SARS-CoV-2 in mice, together with the previous clinical studies, completely fulfills the Koch's postulates and confirmed SARS-CoV-2 was the pathogen of COVID-19." However, the previous clinical study that they refer to is the Zhu N et al. paper that we examined in Study 2 of this section [48]. Zhu N et al. clearly say that their work did not fulfill Koch's postulates. Also, when we examined Zhu N et al. it became evident that the virus was not purified according to the gold standard. If Zhu N et al. had in fact purified the virus according to the gold standard, the authors of our current study could have simply used pure virus to inoculate the mice. Instead they adopted the familiar but faulty process of obtaining impure materials, adding antibiotics and serums, and culturing them in Vero cells.

This paper has generated some controversy. In particular, there is some evidence that the authors of this study manipulated photos in the study. Regarding this, Guillem Pratx, assistant professor at Stanford Radiation Oncology, found some overlapping regions in the photos [53]:

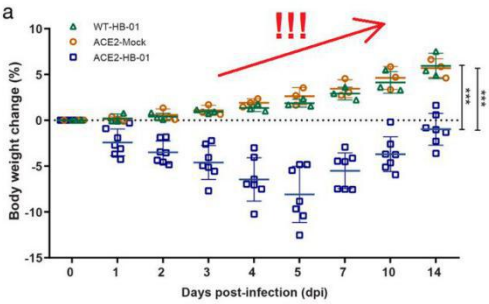


These overlapping photos were also noticed by Elisabeth Bik, PhD. She commented on Twitter that "This is not great. A @Nature paper about SARS-CoV-2 pathogenicity with a possible overlap in photos representing different treatment groups. HT @RuneLinding who asked me to look at the paper" [54].



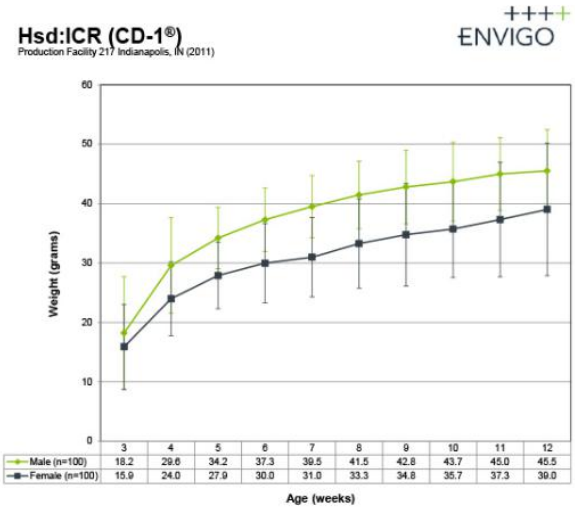
When commenting on a different study, she reveals her frustration by stating that "I reported 100s of papers 5y ago, and most have not been acted upon. Journals do not appear to care for good science" [55]. Sadly, we concur. The Journals do not appear to care for good science and our current study is no exception.

Otto Kalliokoski, a researcher at the University of Copenhagen, also commented on Twitter that "Since everyone is already asking questions about this paper, I will add in the one thing that stood out to me. Figure 1A does not look like the growth curve of 6-11 months (!) old mice" [56].



Animal experiments
For the animal experiments, specific pathogen-free, 6-11-month-old, male and female transgenic hACE2 mice were obtained from the Institute of Laboratory Animal Science, Peking Union Medical College, China. Transgenic mice were generated by microinjection of the mice ACE2 promoter driving the human ACE2 coding sequence into the pronuclei of fertilized ova from ICR mice, and then human ACE2 integrated was identified by PCR as previous described[19]. the hACE2 mainly

According to Kalliokoski, "The body weight of ICR mice will plateau around week 10 of their lives. To have mice put on 5 % body weight in 14 days, in such a consistent way, (despite being a mix of male/female) at age 6-11 *months* seems weird" [57]. He provides the following figure as evidence:

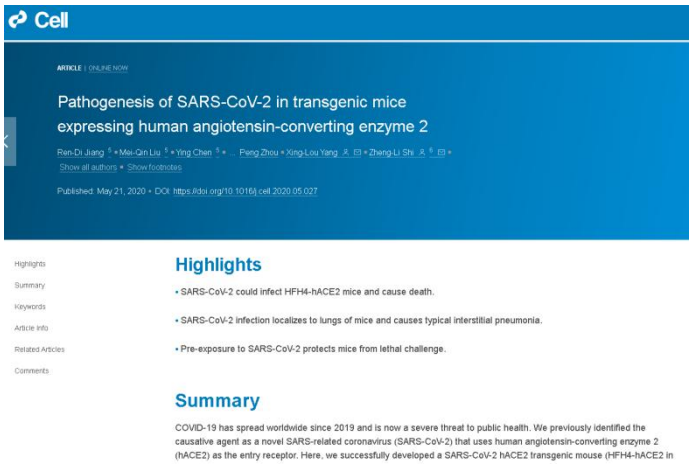


https://www.envigo.com/model/hsd-icr-cd-1-

In response, Anne Sperling, PhD, professor of immunology at the University of Chicago, said " I noticed that too when I skimmed the paper. We weigh mice all the time in me lab, the controls NEVER gain significant weight in such a short time" [58].

It should be clear that this study is highly problematic.

Study 5: Pathogenesis of SARS-CoV-2 in transgenic mice expressing human angiotensin-converting enzyme 2 [59]



https://www.cell.com/cell/fulltext/S0092-8674(20)30622-X

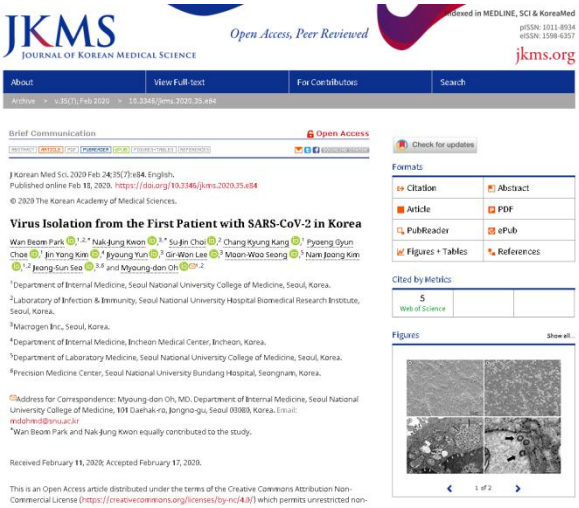
The content of this study is very similar to the content of Study 4. This study does not claim to have fulfilled Koch's postulates. However, the authors do say that "We and other groups quickly identified the aetiologic agent as a severe acute respiratory syndrome related coronavirus (SARSr-CoV), named SARS-CoV-2" and refer to three studies. These three studies include a study by Gorbalenya AE et al. that does not even deal with the issue of causation [60] and two other studies that we have already examined, namely Zhou P et al. and Zhu N et al. [47][48]. Since those latter two studies directly deny fulfillment of Koch's postulates, the authors' statement of identifying the aetiological agent is unfounded. Their statement is also curious due to the fact that Zhou P was one of the contributors of the study.

Similar to what we said in Study 4, if the three studies mentioned by the authors had in fact isolated the virus according to the gold standard, the authors could have simply used pure virus to inoculate the mice. Instead they adopted the familiar but faulty process of obtaining impure materials, adding antibiotics and serums, and culturing them in Vero cells.

Since the authors failed to fulfill postulates (1) to (3), their attempt to fulfill postulates (4) to (6) is completely hopeless.

In the summary section of the study, the authors say that " Our results show that the hACE2 mouse would be a valuable tool for testing potential vaccines and therapeutics." They reiterate this in the discussion section by saying "In summary, a human ACE2 mouse model that partially simulated the pathology of COVID-19, will be a valuable platform for testing vaccines and other potential therapeutics." We believe that these statements expose one of the motivations behind this study, namely to facilitate development of vaccines. However, this worries us greatly. It has become apparent that faulty SARS-CoV-2 science became the basis for public policies such as usage of facial masks, social distancing, contact tracing, and so on. But what the authors are doing is magnifying the effect of this faulty science by using it to create more faulty science that could become the basis for future public policy regarding vaccines. Here is a serious question: How can a vaccine be created for a virus that has never been properly purified? By adopting this faulty science approach, the results will inevitably lead to ineffective and unsafe vaccines, such as has been the case for pneumococcal, SARS-CoV, and MERS-CoV vaccines [61].

Study 6: Virus Isolation from the First Patient with SARS-CoV-2 in Korea [62]



https://jkms.org/DOIx.php?id=10.3346/jkms.2020.35.e84

This study claims to have isolated SARS-CoV-2. However, similar to the other studies we have seen so far, they did not isolate the virus according to the gold standard. The study references the Zhou P et al. [47] study as the first isolation of the virus. We examined the Zhou P et al. paper in Study 1 of this section. Zhou P et al. stated clearly that they did not fulfill Koch's postulates.

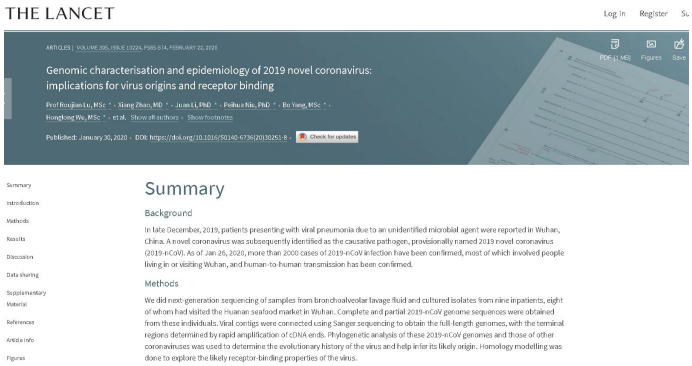
As far as trying to isolate a virus, the authors of this study simply acquired an impure oropharyngeal sample, cultured it in Vero cells, and waited to see cytopathic effects. After observing cytopathic effects, they examined the specimen under an electron microscope. There is no mention that they did any filtering.

After using electron microscopy, they proceeded to sequence the genome. It is always important to remember that they are not sequencing pure virus. The previous steps did not isolate the virus according to the gold standard. They are simply sequencing genetic materials of unknown origin. As mentioned before, it could be the virus, or it could be a slew of other things.

It is also important to note that it is no great feat to produce a genome that is basically identical to previous sequenced genomes in other independent studies. The reason for this is because they started their study with RNA that already tested positive for SARS-CoV-2 via RT-PCR testing. The primers and probes used in the RT-PCR test were created based on the genome previously obtained in those other independent studies. The authors simply produced more of the same RNA through culturing it.

The authors noted that some portions of their sequenced genome differed with the sequenced genome of the Korean CDC (for information on the Korean CDC study, see Study 3 of this section). Do you remember what we said in Section 2 about genetic material being able to alter itself? The authors of this study basically admit the same thing when trying to explain the reasons for the differences in the genome. One of the potential reasons they give is that "These mutations may occur by cell culture-adaptation in that our culture isolate was obtained after first blind passage." There are so many factors at work during this whole process. As we have repeatedly said, simply taking impure materials, culturing them in Vero cells, and seeing cytopathic effects is proof of nothing. There are just too many other factors at work. Nothing can be proved until the virus is purified according to the gold standard. None of the papers we have looked at so far have done this, and this paper is no exception.

Study 7: Genomic characterisation and epidemiology of 2019 novel coronavirus: implications for virus origins and receptor binding [63]



https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(20)30251-8/fulltext#back-bib37

The authors of this study don't really add much to the discussion, but since this paper is referenced in studies 8 and 9, we thought it would be appropriate to examine it briefly.

As far as trying to isolate the virus, the authors say "Special-pathogen-free human airway epithelial (HAE) cells were used for virus isolation. Briefly, bronchoalveolar lavage fluids or throat swabs from the patients were inoculated into the HAE cells through the apical surfaces. HAE cells were maintained in an air-liquid interface incubated at 37° C. The cells were monitored daily for cytopathic effects by light microscopy and the cell supernatants were collected for use in quantitative RT-PCR assays." The authors never said that they actually observed cytopathic effects and provided no electron microscopy images at all. It also seems that they did no filtering. As stated several times before, this faulty process of simply inoculating cells with with impure materials does not correspond to isolation according to the gold standard.

The authors spend a lot of time discussing the genomic characterization of SARS-CoV-2 since this was the main topic of their paper. However, since the authors did not purify the virus, we will not examine this section of the paper. We have covered the topic of genome sequencing in Section 2 and in studies 1, 3, and 6 of this section. Please refer to that material if interested.

In the discussion section of the paper, the authors curiously say that "In conclusion, we have described the genomic structure of a seventh human coronavirus that can cause severe pneumonia." The authors did not prove causation at all, so it is strange that they would say this. The authors reference the Zhou P et al. [47] and Zhu N et al. [48] papers, but both of those papers deny proving causation. In order to prove that SARS-CoV-2 is the causative agent, it is first necessary to isolate it according to the gold standard. After that, it is necessary to do animal experiments to show that the purified virus can cause comparable disease. The authors did neither of these steps and did not reference any papers that did them.

Study 8: Enhanced isolation of SARS-CoV-2 by TMPRSS2-expressing cells [64]

https://www.pnas.org/content/117/13/7001.long

This paper basically takes the same approach to isolation as all of the other studies have. However, it does provide us with some interesting information, so we will briefly examine it.

This study starts off by claiming that SARS-CoV-2 has been isolated previously. It references Zhou P et al. [47], Zhu N et al. [48], and Lu R et al. [63] papers, which we have already examined in studies 1, 2, and 7 of this section. None of these papers isolated SARS-CoV-2 according to the gold standard.

The authors of our present study also follow the same faulty process of isolation, not purifying the alleged virus appropriately. However, instead of using VeroE6 cells, they use VeroE6/TMPRSS2 cells to show that cell lines using the TMPRSS2 enzyme are more susceptible to SARS-CoV-2 infection.

To show this, they inoculated VeroE6/TMPRSS2 cells with nose swabs and sputum and monitored the cell cultures for cytopathic effects (CPE). Their proof that the VeroE6/TMPRSS2 cell line is more susceptible to SARS-CoV-2 infection was simply that it had lower RT-PCR cycle counts (Ct) than other specimens. But as we have stated before, Ct is not a reliable way to estimate viral load [42][43][44][45].

The authors of this study do reveal something interesting. When performing next-generation sequencing (NGS), they stated that " Unexpectedly, the NGS data showed contaminated mycoplasma sequences (Mycoplasma hyorhinis and Mycoplasma arginini) from VeroE6/TMPRSS2 cells. CPE in VeroE6 cells persistently infected with SARS-CoV was enhanced by infection with Mycoplasma fermentans, but whether a similar situation exists for SARS-CoV-2–related CPE in this cell line is unclear." How did the Mycoplasma fermentans bacterium suddenly appear on the scenes? This provides further proof of what we said in Section 2 about the many factors at work during this whole process. As we have repeatedly said, simply taking impure materials, culturing them in cells, and seeing cytopathic effects is proof of nothing. It is impossible to prove the existence or pathogenicity of any alleged virus, including SARS-CoV-2, using these faulty procedures.

Study 9: Isolation and characterization of SARS-CoV-2 from the first US COVID-19 patient [65]

https://www.biorxiv.org/content/10.1101/2020.03.02.972935v2

This paper has not yet been certified by peer review. Also, some of the authors of this study are researchers at the US Centers for Disease Control and Prevention (CDC).

The authors start off their abstract by saying that the etiologic agent of pneumonia in Wuhan China was identified as SARS-CoV-2. When looking at their references, they mention Zhu N et al. [48] and Lu R et al. [64]. We examined these papers in studies 2 and 7, respectively. Neither of these papers isolated the virus according to the gold standard. Furthermore, Zhu N et al. clearly state that they did not fulfill Koch's postulates. Lu R et al. did say that SARS-CoV-2 "can cause severe pneumonia" but they provided no proof at all to validate their claim. The US CDC authors only use the words "etiologic agent" once in the abstract, so it might have been a mistake, unlike the authors of Study 3 who used the words "causative agent" rather purposefully four times, despite providing no proof and referencing papers that actually denied that claim.

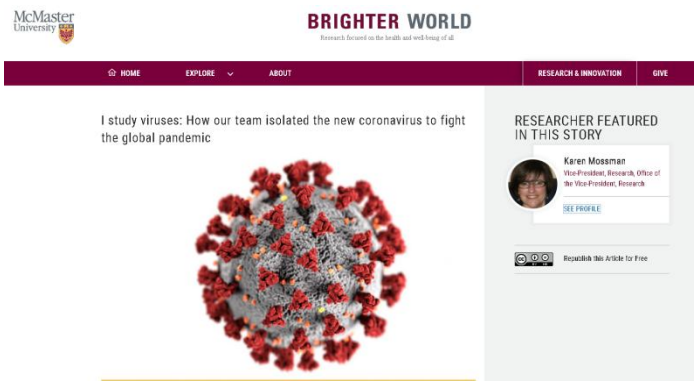
As far as trying to isolate SARS-CoV-2, the authors followed the same faulty procedures that previous studies have. They simply used nasopharyngeal (NP) and oropharyngeal (OP) swabs, added antibiotics and serum, used the specimen to inoculate Vero E6, Vero CCL-81, HUH 7.0, 293T, A549, and EFKB3 cells, and waited to see if there were any cytopathic effects. The authors noted that "No cytopathic effect was observed in any of the cell lines except in Vero cells," but also added that there was replication in all cells except for EFKB3. In other words, SARS-CoV-2 seems quite picky about its living environment. It will cause trouble in some environments, but live peacefully in others.

The paper makes no mention of filtering. Also, remember that simply seeing cytopathic effects or particles under an electron microscope proves nothing. Please review Section 2 and the previous studies of sections 3 and 4 for more information on why the procedures adopted by the authors are so problematic.

The authors did genome sequencing, but since they did not purify the virus, we will not examine this section of the paper. We have covered the topic of genome sequencing in Section 2 and in studies 1, 3, and 6 of this section. Please refer to that material if interested.

This study has been rather disappointing. We had supposed that a study being sponsored by the US CDC would at least attempt to prove that SARS-CoV-2 is the causative agent of the alleged COVID-19 disease. Rather, the study simply follows in the footsteps of every other paper on this subject. The virus was not purified and no animal experiments were done. However, since this paper still has not been certified by peer review and has apparently not been used in creating public policy, we cannot condemn it in the same manner as we did Study 3 of this section.

Study 10: I study viruses: How our team isolated the new coronavirus to fight the global pandemic [66]



https://brighterworld.mcmaster.ca/articles/i-study-viruses-how-our-team-isolated-the-new-coronavirus-to-fight-the-global-pandemic/

This is not a published paper. It is an article that was posted on a website operated by McMaster University in Canada. The author is a professor of pathology and molecular medicine and Acting Vice President of Research at McMaster University.

The author of this article starts by saying that "The emergence of a new coronavirus in a market in Wuhan, China, in December 2019 set in motion the pandemic we are now witnessing in 160 countries around the world." This is quite a claim! Earlier we complained about the unwarranted use of the words "causative agent," but "set in motion the pandemic" is simply an outrageous claim to make, especially in light of the fact that SARS-CoV-2 hasn't even been purified yet. What actually "set in motion the pandemic" was drastic public policy that adopted wide-scale RT-PCR testing to create massive amounts of false positives through non-validated use on asymptomatic people [5]. As mentioned in the introduction section of this paper, SARS-CoV-2, if it even exists, produces no symptoms that differ from the common cold or seasonal flu [1], produces no symptoms at all in nearly 80% of infected people [2], and has a cumulative hospitalization rate basically on par with the seasonal flu [3]. The "pandemic of panic" was not set in motion by SARS-CoV-2. That much has become clear.

As far as trying to isolate the virus, the article doesn't go into great detail, but makes clear that they used the same faulty procedures as the previous studies we examined. However, the author does reveal some interesting information.

She states, "It isn't obvious what particular environment [the] virus has adapted to, so it can be hard to grow it successfully in the lab...We can use tricks to draw out a virus. Sometimes the tricks work and sometimes they don't. In this case, [we cultured] the virus on immunodeficient cells that would allow the virus to multiply unchecked. It worked."

Do you remember what we said in Section 2 when mentioned some general criticisms about how virologists go about their work? They can literally spend days and weeks preparing the ideal lab conditions to achieve their desired goal. Even using immunodeficient cells is acceptable. Anything is possible in a virologist's lab! Simply assume there is a virus, do whatever it takes to achieve cytopathic effects in cultured cells, and declare victory as the heroes of the day!

She also states, "Since specimens from patients are also likely to contain other viruses, it is critical to determine if a virus growing in the culture is really the target coronavirus. Researchers confirm the source of infection by extracting genetic material from the virus in culture and sequencing its genome."

Again, do you remember in Section 2 when we mentioned all of the viruses living in the human body? These countless viruses also live in the specimens that are inoculated into the cell cultures. These countless viruses are also present when those cell cultures experience cytopathic effects (CPE). The author of this article says

that researchers confirm the source of infection (i.e., CPE) through genetic sequencing, but she fails to mention that the researchers cheat to achieve their goal. The specimen had already tested positive for SARS-CoV-2 via RT-PCR testing before being inoculated into the cell cultures. Therefore, it is no surprise that it will test positive for SARS-CoV-2 after cell culturing. RT-PCR is an ultra-sensitive test that is capable of detecting the tiniest amounts of RNA in a specimen. In other words, simply testing the cell cultures using RT-PCR is not an acceptable way to confirm the source of the CPE. It could have been the other viruses that caused the CPE. Remember that only about 6,000 viruses have been described in detail, but millions of them exist in the body [20][21]. It isn't hard to imagine that one of these unknown viruses, for which there are no RT-PCR tests available, could have been the culprit behind the observed CPE and not SARS-CoV-2.

The author of this article did not isolate SARS-CoV-2 according to the gold standard. Therefore, her claim that SARS-CoV-2 "set in motion the pandemic" is unfounded. However, the article did provide us with some interesting information that further proved the matters we discussed in Section 2.

Conclusion

In this paper, we showed that the science surrounding SARS-CoV-2 is very faulty. In fact, it is so faulty that the entire existence of SARS-CoV-2 as a pathogenic virus is highly questionable. We believe that we have provided enough evidence to prove our claim.

In particular, we did the following:

1. Identified the "gold standard" for determining the cause of an infectious disease
2. Identified how to isolate (i.e., purify) the virus according to the "gold standard" and described some shortcomings of this method
3. Examined claims of satisfying the standard for other coronaviruses
4. Examined claims of satisfying the standard for SARS-CoV-2

We looked at 10 papers on SARS-CoV-2 and showed that none of them isolated the virus according to the gold standard and none of them proved that SARS-CoV-2 is a causative agent of disease. In fact, the two papers [47][48] that were referenced the most by the other papers [50][52][59][62][63][64][65] both directly denied fulfilling Koch's postulates.

As we mentioned in Study 10 of Section 4, SARS-CoV-2 is being blamed for all the chaos in the world right now, but the real culprits of the chaos are poor public policies. COVID-19 produces no symptoms that differ from the common cold or seasonal flu [1], produces no symptoms at all in nearly 80% of infected people [2], and has a cumulative hospitalization rate basically on par with the seasonal flu [3]. Since the alleged symptoms of COVID-19 are the same as the common cold or seasonal flu, it is impossible to detect by mere clinical diagnosis. The only reason it is being detected at all is because wide-scale RT-PCR testing is being used to create massive amounts of false positives through non-validated use on asymptomatic people [5]. According to Dr. John Ioannidis, Professor of Medicine at Stanford University School of Medicine, "If we had not known about a new virus out there, and had not checked individuals with PCR tests, the number of total deaths due to 'influenza-like illness' would not seem unusual this year. At most, we might have casually noted that flu this season seems to be a bit worse than average" [4].

The policymakers who have enacted drastic public policies that have ruined the economy and terrorized people throughout the world need to be held accountable.

Here is what needs to happen:

The people of the world need to turn off their televisions. They need to start using their minds and their common sense. They need to use their five senses and look around and see that there are no dead bodies lying in the streets. They need to to realize that more than 95% of deaths attributed to COVID-19 were actually due to pre-existing medical conditions and old age [67]. They need to realize that a huge medical fraud has been perpetrated on them. They need to realize that their freedoms have been stripped away from them for no good reason. They need to realize that they have been lied to by their leaders, news media, and healthcare workers. They need to wake up and take lawful action immediately to hold the relevant people accountable and restore all the freedoms that they have lost in the name of COVID-19.

Acknowledgment

We would like to thank David Crowe from theinfectiousmyth.com [68]. This paper would not have been possible without his writings and help. We would also like to acknowledge the contributors at the Piece of Mindful blog, especially Mark Tokarski, Fauxlex, and Stephers [69][70][71]. Some of their content, comments, and insight were used in this paper.

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