A few days ago I provided critical comments on a publication from Australia (University of Melbourne) which claimed isolation and identification of SARS-CoV-2 virus [1]. I suggested that claims were not supported by scientific evidence and logic.

The present article critically evaluates a similar claim from America's Center for Disease Control (CDC) in a publication [2] entitled:

“Severe Acute Respiratory Syndrome Coronavirus 2 from Patient with Coronavirus Disease, United States”. [ CDC-publication]

The patient case report for this (above-mentioned) study is detailed in a separate publication [3], and is considered here first before we moved to the study itself.

Case Report - COVID-19 Diagnosis

A 35-year-old man who had recently returned from China presented to a clinic in Washington State with a 4-day history of cough and subjective fever. The patient visited the hospital voluntarily after hearing of a health alert over an alleged novel coronavirus outbreak in China apparently with similar symptoms to his own.

The medical examination indicated: body temperature (37.2°C); blood pressure 134/87 mm Hg; oxygen saturation 96%; chest radiography showed no abnormalities. CDC staff decided to test the patient for 2019-nCoV (subsequently renamed SARS-CoV-2) based on current CDC “persons under investigation” case definitions. The patient was discharged from the hospital, but was later called back as his PCR test returned a positive reading.

Treatment during this time was largely supportive. The patient received, as needed, 650 mg of acetaminophen every 4 hours and 600 mg of ibuprofen every 6 hours. He also received 600 mg of guaifenesin for his continued cough and approximately 6 liters of normal saline over the first 6 days of hospitalization.

In view of the potential for hospital-acquired pneumonia, treatment with vancomycin (a 1750-mg loading dose followed by 1 g administered intravenously every 8 hours) and cefepime (administered intravenously every 8 hours) was begun.

The CDC asserted that the patient’s nasopharyngeal and oropharyngeal swabs tested positive for SARS-CoV-2 via rRT-PCR assay. The stool and both respiratory specimens also tested positive by rRT-PCR for SARS-CoV-2, whereas the serum remained negative.

Treatment with intravenous remdesivir (a novel nucleotide analogue prodrug) was begun on the evening of day 7, and no adverse events were observed in association with the infusion. Vancomycin was discontinued on the evening of day 7, and cefepime was discontinued the following day.

On hospital day 8 (illness day 12), the patient’s clinical condition improved. Supplemental oxygen was discontinued, and his oxygen saturation values improved to 94 - 96% while he was breathing ambient air. The previous bilateral
lower-lobe rales were no longer present. His appetite improved, and he was asymptomatic aside from intermittent dry cough and rhinorrhea. He was afebrile, and all symptoms had resolved with the exception of his cough, which was decreasing in severity.

Considering the case history, it is not clear as to why the patient was subjected to an rRT-PCR test for SARS-CoV-2 when he was exhibiting what would normally be diagnosed as a mild flu and which would therefore be treatable with antibiotics (as per Australian response). Nevertheless because the rRT-PCR test came out positive the assumption that the patient had SARS-CoV-2 prevailed and the antiviral drug, remdesivir was (accordingly) administered.

The fundamental error here is that the patient should not have been given an rRT-PCR test for establishing the presence of SARS-CoV-2, and this is because the rRT-PCR test is not a validated test, and cannot be validated without an independently extracted physical reference sample of the virus, which has not been obtained to date. From a scientific viewpoint, and with regard to the matter at hand, no assay can be valid without an independently verified physical reference sample. Therefore the diagnosis, and the associated claim, that the patient had SARS-CoV-2 and its associated infection/disease (COVID-19), is necessarily false.

**CDC-publication**

It would be safe to assume that the authorities/experts are aware the shortcomings of the ‘PCR dialogistic process’, and (accordingly) attempt to overcome this with convoluted logic which runs as follows:

“As no physical sample of the virus is available, let us ‘create’ the virus from RNA found in the patient’s swab samples and which we presume to be RNA from the SARS-CoV-2 virus.”

And as if this were not bad enough, there is the additional problem consisting of the fact that the DNA resulting from such creativity is not the actual virus itself but merely a virus marker which is commonly, but wrongly, referred to as the virus.

To clarify, rRT-PCR never determines a virus but only a DNA (marker) of the virus. Moreover, a PCR test does not even determine actual biological DNA (from the swab samples), because it is almost invariably present in extremely small amounts which must be multiplied millions of times in order to be detected. There is therefore no definitive link between the RNA/DNA in the mucus swab and the purported virus. In short, one cannot definitively establish the presence of a virus by simply assuming that a particular RNA/DNA strand relates to a certain patient’s symptoms, but must physically isolate the virus as a whole, which will then provide us measurable quantities of DNA.

PCR is therefore not a valid diagnostic test but simply a technique for manufacturing multiple (millions and billions) copies of DNA initially synthesized via a RNA primer, the resulting measurable quantity then being used to ‘confirm’ the presence of that DNA via standard analytical chemistry tests. The limits of the technique with regard to diagnostic accuracy are well documented, and Dr. Kary Mullis, Noble Laureate and inventor of the PCR technique highlighted this on several occasions. Hence, PCR is often not recommended, or at least should not be relied upon, for diagnostic purposes.

Parallel to producing multiple copies of DNA via the PCR technique, the CDC also used swab
samples to inoculate cell culture to produce multiple copies of the (supposed) virus. This (latter) production was also monitored using the PCR technique. That is, the process involved: (1) creating multiple replicate copies of the alleged virus DNA via the PCR technique; (2) creating multiple copies of the alleged virus in a culture/media/soup from the swab sample.

The study asserts that: (1) electron microscope images of spherical particles with spikes are an indication of the presence of SARS-CoV-2; (2) observed DNA from the culture containing the spherical particles is similar to that of other corona viruses (detected with PCR-technique and evaluated with computer-generated modelling) and is assumed to be associated with SARS-CoV-2. This kind of “confirmation” treats the culture/media/soup as though it were the virus itself, which would be like saying that sugar molasses is pure sugar! This is positive (mind-set) thinking, not science: the analysis should have instead culminated in the isolation of the actual virus particles themselves, which is the standard procedure for confirming the existence of any virus, its link to disease and its clinical symptoms. Therefore, as it stands now the publication does not provide any evidence that the SARS-CoV-2 virus has been positively identified.

Further, the question remains as to the basis upon which SARS-CoV-2 is considered linked to the disease COVID-19. Just what actually is a COVID-19 disease? What are its specific symptoms and clinical (measurable) parameters? The case study as well as the CDC-publication reveals nothing in this regard, but rather, it would appear that a trendy PCR digital testing regime has substituted for real, ‘boring’ empirical science.

And there are several associated claims of similar dubious merit: (1) SARS-CoV-2 is contagious; (2) SARS-CoV-2 is 5 or 10 times deadlier than the common flu virus; (3) face-masks provide protection from the virus; (4) social distancing protects the public by stopping or reducing the spread of the virus; (5) washing hands or exposed skin surfaces provides protection from the virus; (6) lockdowns (partial or full) help reduce the spread of the virus; (7) a significant increase in positive test results (“cases”) shows a wide spread of the SARS-CoV-2 virus; (8) vaccines are under development, with various time schedules for availability, to protect patients/public from the SARS-CoV-2 virus.

In summary, the resultant declaration by the CDC of the presence of SARS-CoV-2 in the USA was based on a flawed (PCR) technique. This declaration was then further assumed confirmed by electron microscopic images of “virus-like” sphere-with-spikes particles in cell lysate or cell culture. No effort was made to isolate, identify, and characterise the particles from culture media to confirm that the particles were indeed SARS-CoV-2 and whether or not they might have represented other viruses previously catalogued. It is quite clear that the analysis was not submitted to the rigours of empirically-based science.

Experts and authorities are requested to reconsider their views with regard to the scientific method in declaring the presence of the virus, its link to any disease and its spread. The science of analytical chemistry would state that there is currently no evidence available in support of the current claims and measures regarding the virus SARS-CoV-2 and COVID-19 narrative.

References:


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