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Questions Raised By the "New" Coronavirus: Too Many "Experts"–Too Little Thought

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Abstract

History has a tendency to repeat itself, and pandemics/epidemics are no exception. Case in point, the common ground between the present "novel" 2019 coronavirus (AKA COVID-19), the SARS (Severe Acute Respiratory Syndrome) and MERS (Middle East respiratory syndrome) outbreaks before that, and the Great Pandemic of 1918, long ago. The present COVID-19, did not occur in a vacuum. By December of 2018, Liu et al., proclaimed tuberculosis to be an epidemic throughout China, an epidemic which still rages on. China harbors the second largest burden of tuberculosis in the world–a disease which often begins with flu-like symptoms, and a disease whose bacilli are laden with RNA bacterial viruses called mycobacteriophages. Quietly, by 2016, the World Health Organization acknowledged that despite advances, the TB bacillus, which Koch was forced to refer to as "the TB virus", is once again the deadliest pathogen in the world. Here we compare all 4 pandemics/epidemics with some surprising results and similarities.

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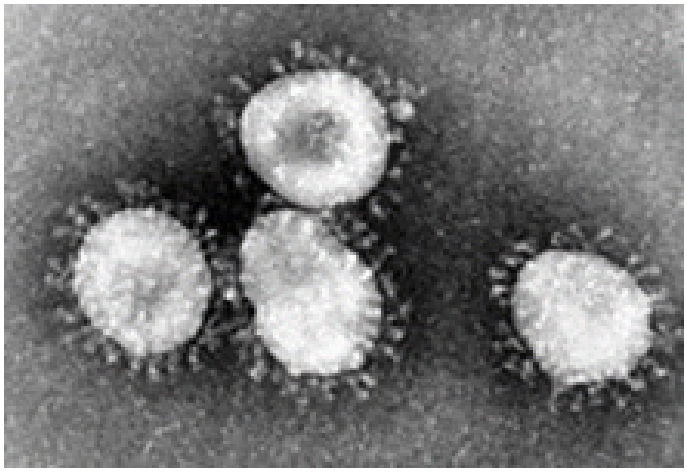

***Figure 1**: Electron micrograph of a coronavirus*

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Abbreviations : SARS: Severe Acute Respiratory Syndrome; MERS: Middle East respiratory syndrome; PCR: Polymerase Chain Reaction; CDC's: The Centers for Disease Control and Prevention; ARDS: Acute Respiratory Distress Syndrome; CWD: Cell-wall-deficient; MAC: Mycobacterium avium complex

Introduction

Diagnosing a viral disease is no easy matter. Just to name a few instances, Lyme disease, mycoplasma pneumonia and Legionnaires' disease were all thought to be viruses. That is, until their respective bacteria were found. SARS itself, often compared with COVID-19, was misdiagnosed as avian influenza A (or 'bird flu'), the human metapneumoviruses (hMPV), and then a chlamydia-like bacterial-like organism taken from patients during what later came to be known as the Guangdong outbreak, where death came within hours. But none of these pathogens, thought to be SARS, could be confirmed in most labs outside of China. Finally, on April 16, 2003, a "novel" coronavirus, never before seen in humans or animals was proclaimed by WHO officials to cause SARS. Up to that point human strains of coronavirus were only associated with mild disease and never known to kill precipitously. Still major problems in diagnosis existed with SARS, problems which have carried over to the "new" and "novel" coronavirus. Many cases were being diagnosed by doctors simply by their symptoms, a fool's errand when defining a "flu-like" illness. And in those cases where patients were being tested, it was mostly through antibody-antigen tests, filled with inaccuracy. This left only a minority that were tested by the method called PCR (Polymerase Chain Reaction), which has had its own problems.

SARS coronavirus was present only in about half of the SARS cases and antivirals such as ribavirin and oseltamivir (Tamiflu) were not working on SARS culture plates, nor were they very successful in the clinics or hospitals either. That passenger viruses do exist has been abundantly documented, as when the HTLV-1 virus was mistakenly attributed to be the cause of AIDS. And so the question which never went away loomed: were the coronaviruses merely traveler viruses from a yet to be determined stealth cause?

Even during and after the 1918 Great Influenza Pandemic, inconsistencies abound. In 1933 researchers claimed they had discovered human influenza "virus". So, what was the flu virus of 1918? Historically, in 1892 the flu was originally named *Mycobacterium influenzae* because it resembled tuberculosis. Both of these pathogens stained similarly on a lab slide. Staining is one method of identifying types of bacteria. Also, it was eventually found that *Mycobacterium influenzae* and *Mycobacterium tuberculosis* have similar genetic profiles.

CDC's (*The Centers for Disease Control and Prevention*) diagnostic coronavirus test is a real-time reverse transcriptase-polymerase chain reaction (PCR) test that provides "possible" detection of COVID-19. According to the CDC, a positive test result suggested "likely infection", presumed to be contagious. As for negative results, however, US regulators warned that negative results don't necessarily preclude infection. If consistently negative results don't preclude an infection with coronavirus, then there could be more behind the disease than just coronavirus. Curiously, Kary Mullis, the inventor of the polymerase chain reaction (PCR) technique, said that such tests cannot detect free, infectious viruses at all; they can only detect proteins that researchers believe, in some cases wrongly, are unique to the virus being tested for. The tests can detect genetic sequences of viruses, but not viruses themselves. [1]

Real and Present Danger

Liu emphasized that historically there has been no record of a severe TB outbreak in China, unlike the situation in Europe where the epidemics of 18th to 19th centuries raged. China might have dodged the bullet with past European-like epidemics, but increasing urbanization might just have finally caught up with it. [2]

Noymer and Garenne's statement that tuberculosis was behind the many deaths in the 1918 Great Influenza Pandemic was specifically based upon the well-known concept that the secondary bacterial infections that cropped up in 1918 were common in TB-infected lungs. [3] Pearl similarly saw a definite relationship between the explosiveness and fatality of that killer "influenza" pandemic in 40 American cities and the existing death rates for pulmonary tuberculosis and related disease.[4] In Nishiura's study not only was TB shown to be associated with influenza death, but there was no influenza death among controls without TB. Nishiura concluded: "*Should a highly fatal influenza pandemic occur in the future, testing the role of TB in characterizing the risk of death would be extremely useful in minimizing the disaster*…" [5]

Tiny, difficult to diagnose, viral-like, cell-wall-deficient forms of mycobacteria such as *Mycobacterium tuberculosis* were documented in the Great Pandemic of 1918 by Wade and Manalang, who saw them in a germ once called *Mycobacterium influenzae* (Pfeiffer's bacillus, *Hemophilus influenza*). At one point in the pandemic this germ was thought by the majority of US investigators to be the cause of the Great Pandemic. [6] In December of 1918, a *British Medical Journal editorial* [7] spoke of Wade and Manalang's filter passing "pseudoinfluenza", concluding that it should have satisfied both virologists and their bacteriologist rivals. "Those who accept this hypothesis may still maintain their belief in Pfeiffer's organism and may assume that it has a filterable [virus-like] stage" as well. But such reasonable thoughts never stuck in the minds of researchers.

Is It a Virus?

Actually, antiviral medicines were not found to provide benefit in either the SARS or MERS strains of coronavirus, which broke out in 2002 and 2012, respectively. Yet this habit of administering antivirals has been clung to stubbornly in the treatment of the 138 Coronavirus-infected patients hospitalized in Wuhan described in Wang., *et al*'s recent *JAMA* study. [8] On the other hand, all of these 138 patients, and most of the 99 patients in Chen's current *Lancet* study [9] received antibiotics, some of which have significant anti-mycobacterial as well as antibacterial activity. Antibiotics have no effect against viruses. And although it is claimed that antibiotics are and were simply being used to quell "secondary" bacterial infections in the new pandemic/epidemic, the fact is that antibiotics have proven universally to be of great help worldwide to the vast majority of novel coronavirus COVID-19 victims, with or without secondary infections.

The herding together or quarantining potential COVID-19 is common. Yet Thomas M. File Jr., president of *the Infectious Diseases Society of America* was concerned that the close proximity of quarantine could expose people "to other infections that are even more easily spread than coronavirus, like tuberculosis, which is airborne, and bacterial infections that can spread among dense populations." [10]

Certainly Dr. File remembered that in 1990, a new antibiotic-resistant tuberculosis outbreak took place in a large Miami municipal hospital. Soon similar outbreaks broke out in three New York City hospitals from which it spread to city prisons. Like the origin of SARS, the infection spread in nosocomial manner – from patient to patient and from patient to staff. As in Florida multi-drug-resistant strains made the New York TB cases almost impossible to treat and the majority of sufferers died, many within weeks. By 1992, resistant tuberculosis had appeared in seventeen US states, with mini-epidemics in Florida, Michigan, New York, California Texas, Massachusetts and Pennsylvania, and was reported by not the American, but the international media, as out of control.

During this SARS coronavirus outbreak, Wong *et al*, writing in the *Journal of the Chinese Medical Association* warned: "Preoccupied with the diagnosis of SARS (Severe Acute Respiratory syndrome) in a SARS outbreak, doctors tend to over-look other endemic diseases, such as tuberculosis" [11], a warning usually ignored.

The chronicicological timetable of the present Wuhan viral pandemic suggests nothing new or novel. This coronavirus outbreak started in December 2019 after 41 people presented with pneumonia of no clear cause. The Wuhan winter is from December to February. Yang's Wuhan study from 2004 to 2013, described the annual TB surge there as being fueled by increased transmission in the winter; peaking in March, with a second smaller peak in September. [12] Such a tubercular Spring (March) peak is identical with that from other countries, such as Japan, Kuwait, India and Spain. Yang attributed this increased winter transmission to indoor crowding, subsequent vitamin D deficiency, and even air pollution. The increasingly severe air pollution in Wuhan, powered by the influx of foreign companies and the increased use of incineration for waste disposal, resulted in a visible haze so thick that it reduced peripheral vision as far back as June of 2012 –a haze with inhalable particulate matter, highest in the winter, and, according to Yang, of a particulate size able to harbor *Mycobacterium tuberculosis*.

Those Who Cannot Remember the Past are Condemned to Repeat It

Wuhan's economy was on fire, with new companies moving in weekly. With this came a greatly increased demand for poultry and swine, two staples of the Chinese diet, along with the expansion of farms to raise them and the inevitable tons of waste that this brought. Even as far back as 2015, there were five major waste incineration plants in Wuhan with many more scheduled to be built. [13]

That was just the beginning. By July, 2018, fourteen large pig breeding farms in Wuhan, with a combined annual pig production of 1.5 million pigs pooled investments with the intent to slaughter 2 million pigs per year in Wuhan. China is the world's biggest consumer of pork, and alone accounts for more than half of the world's pig population. That is until another "virus", said to be African Swine Fever, spread throughout China–which had no cure and a near zero survival rate for infected pigs, and which, by August 2019, virtually wiped out 40% of China's entire pig population, including those in Wuhan. Essentially one-quarter of the world's pigs died in one year. China then did what it had to do and began to cull thousands of pigs to control the outbreak in 2018. But how many dead animals, including those in Wuhan were buried and how many incinerated is an open question. Burning pigs and pig excrement was a sure recipe for visible haze. The Chinese government soon came up with incentive programs for livestock farmers to sell their manure to use as fertilizer, which was only successful to one extent or another. During one time-interval study, from 1953 to 1968, the incidence of *Mycobacterium avium* (Fowl tuberculosis) in a pig population was an astonishing 81%. [14]. As reported by some workers, *M. avium* isolates from swine represent a major threat to human beings. And the similarity of the IS1245 RFLP patterns of the human and porcine isolates indicates close genetic relatedness, suggesting that *M. avium* is transmitted between pigs and humans.

Fort Funston 1918, Kansas USA

Similar events occurred at Fort Funston in Kansas circa 1918, thought by many to be the birthplace of the onset of the Great Influenza Pandemic of 1918.

It was only with industrial development that the US TB epidemic traveled to the Midwest and Kansas, and finally the West. [15] Yet all-in-all, by 1918, it could be said, in so far as tuberculosis was concerned, that the world was a super-saturated sponge ready to ignite and that among its most vulnerable parts was the very American Midwest where the 1918 unknown pandemic hit, in rural Haskell country, Kansas, in the midst of an infectious pig slaughter of unknown cause, a few hundred miles from Camp Funston, today Fort Riley.

It had to be more than a coincidence that by the autumn of 1918 thousands of Midwest pigs died, seemingly from the same flu-like illness and in the same Haskell County location in which the worst human pandemic in history, which would

kill between 20 and 100 million people was about to begin. US Inspector and veterinarian J.S. Koen, for lack of another term, and with no evidence other than a hunch, quickly called this unknown disease in pigs "swine influenza'', even as it killed pig after pig.

That thousands of pigs died in the Autumn of 1918 was problematic in that bird or fowl TB also called Avian tuberculosis or *Mycobacterium avium* routinely infects birds as well as hogs and sometimes cattle –but could, under the right conditions also infect man. So pigs had involuntarily become the living laboratory thru which three of the main types of tuberculosis (human, cow and fowl) could mutate through genetic exchange by their viral mycobacteriophages, much in the same fashion as has been attributed to the ''influenza virus''. The stage was set for disaster.

Unknown at the time, but pertinent since Kansas lies squarely in America's' "dustbowl'', were the results of a previous European experiment wherein guinea pigs exposed to organisms like Avian tuberculosis got little or no lung disease. However, when these mycobacteria were placed in dust aerosols with particulate matter, guinea pigs came down with progressive, fatal lung disease [16], not unlike what was occurring in the pandemic of 1918 as well as with the present Wuhan air pollution with its particulate matter haze. Thus, though it was long proposed that infection with fowl tuberculosis required some ''defect'' in the human immune system, that defect could be as simple as dust tying up the body's immune system. Certainly a previous tubercular infection, common in 1918, with or without accompanying ''chronic bronchitis'', could immunosuppress. [17,18]

Fort Riley, Kansas was a sprawling establishment housing 26,000 men and encompassing an entire camp, Camp Funston. Within that camp, thousands of horses, hogs, mules and chickens produced in excess of a stifling nine tons of manure each month. And the accepted method for its disposal was to burn it, even against driving wind. State Veterinarian W.J. Butler would report at the 28th Meeting of the *United States Live Stock Sanitary Association*: "I consider contaminated manure and stagnant water the most important factors in the spread and propagation of tuberculosis". [19] Wang's recent JAMA study suggested that COVID-19 may spread fastest on cruise ships and in hospitals where workers re-use gear contaminated with feces to try to conserve supplies. [7]

And so on Saturday, the 9th of March, 1918, a month which just happened to coincided with the annual peak surge for tuberculosis in Wuhan, China, a threatening black sky forecast the coming of a major dust storm towards Fort Funston.. When this storm struck, combined with the ashes of over 9 tons of burning manure, a stinking, stinging yellow haze resulted. The sun was said to have gone black in Kansas that day. Two days later, on March 11th, company cook Albert Gitchell reported to the Funston infirmary saying he had "a bad cold" with flu-like symptoms. Among his symptoms were a headache, a sore throat, muscle aches, chills and fever. He also reported cleaning pig pens on March 4th, one week before feeling sick. Gitchell would never recover from this, his last illness. And by noon of March 4th, a hundred men joined him at the Army infirmary he had walked into. Within a month 1,000 men were sick and approximately 50 dead. [20] Camp Funston was having a deadly epidemic.

These deaths were highly unusual, but nothing like what would return in the fall, when the disease would come back with a vengeance, seeming to gain strength through human passage. Camp Funston in March, Camp Devens in September (a month that also coincides with the second annual TB peak surge in Wuhan), then across the country and the world, leaving an estimated 50-100 million dead globally, at least 600,000 to a million of them American, in the span of less than a year –the most destructive plague that man had or has ever witnessed.

"Wuhan Pneumonia"

Chan has characterized the present Wuhan Coronavirus pandemic as at times progressing to an "atypical" pneumonia. Such references to atypical pneumonia were used by Rist in 1929, when he found in almost 50 per cent of 300 consecutive

hospital pneumonia admissions, "atypical" tubercular pneumonia. [20] And Farber and Clarke reported 100 cases which were admitted to a general hospital for non-tuberculous pneumonia, which were found to be of a tubercular cause. [21]

In his own post-mortem of the Great Pandemic, Major Milton Hall, M.D. spoke of inflammatory diseases of the respiratory tract freely using the word "influenza" to explain what happened in 1918 while at the same time admitting that the cause of such influenza was entirely unknown. 'Atypical' was the same description used by Dr. Hall for 'influenza' pneumonia, something he traced as being far more susceptible in troops coming from "southern and southeastern" parts of the United States, states with the highest incidence of pulmonary tuberculosis in the country. [22]

Lessons from SARS

Much as with Wuhan pneumonia, in patients exposed to or coming down with SARS, [23] lower lobes were preferentially affected, along with perihilar enlargement, intralobar septal thickening and ground-glass opacities [24], all present in mycobacterial lung disease. Even on a clinical and laboratory basis it seems that SARS, the Novel Coronavirus and miliary blood-borne tuberculosis find parallels. Fever, leukopenia, thrombocytopenia, lymphopenia and hypertransaminasemia can all be present in each. [25] With regards to fever, in Shi's study of the major causes of fever of unknown origin at Peking Union Medical College Hospital in the past 26 years, by far the most common cause of Fever of Unknown Origin (FUO) was tubercular disease (479 cases, 48.0%). [26]

The coronavirus kills by way of the Acute Respiratory Distress Syndrome (ARDS). With ARDS, difficulty of breathing results from inflammation with subsequent flooding of the alveolar spaces through fluids gathering at these sites, blocking the proper exchange of oxygen. The number of TB cases in which people in the Orient die of adult respiratory distress syndrome (ARDS) has been on the rise for some time, the same ARDS that often provokes a 'crazypaving' appearance at thin-section CT. [27] Chen cautiously suggested that the percentage of blood-borne tuberculosis as a potential cause of ARDS 'might not be very low'. [28] Roger., *et al,* however favor suspecting tuberculosis in all cases of acute respiratory failure of unknown origin. [29] ARDS caused by miliary TB is associated with just as high a fatality rate as ARDS caused by either SARS or the Wuhan coronavirus. [30]

Yet perhaps one of the most puzzling features to virologists regarding the new coronavirus as with SARS and MERS is that compared with adults and teenagers, it seems to have a less aggressive clinical course in younger children [31]; puzzling until one reads bacteriologist Dubos's observation that tuberculosis is rarely severe between the ages of 5 and 12, [32] the very age span spared by the 2019-nCOV coronavirus.

As an explanation for the precipitous death in a matter of hours to a few days recorded in some with fatal coronavirus, there was the comparable acute blood-borne miliary form of virulent "galloping" tuberculosis ("galloping consumption") at the time of the time of the 1890 and 1918 flu Pandemics, a disease which according to McCall Anderson, then Professor of Clinical Medicine at the University of Glascow, could kill within hours to a few days, even without influenza. Such galloping consumption also began with high fever and pneumonia in one or both lungs. [33]

Finally, that bats came to the forefront, as a possible vehicle of the new novel coronavirus is no surprise. Already associated with the outbreaks of the SARS and MERS coronavirus (Middle Eastern respiratory syndrome or MERS-CoV)- bats have been recognized as the natural reservoir for over 100 other viruses including MERS, Ebola virus, Marburg virus, Hendra virus, and Nipah virus, to name a few. But there again bats can also carry mycobacteria from the *M. tuberculosis* complex and its viral-like CWD forms as well. [34-36]

Conclusion

The preferred form of tuberculosis and fowl tuberculosis, according to Nobel nominee Lida Mattman [37], is their hard-to-spot, cell-wall-deficient (CWD) forms. Tiny and viral-like, the diagnosis of these forms requires special stains, special culture media, and often mycobacterial growth stimulants – materials which most labs simply do not carry. CWD forms can and often do revert back to the classical acid-fast bacillus of TB. But when they do not, according to Mattman, detection of CWD-tuberculosis is about 50%; and for the Mycobacterium avium complex (MAC), highly implicated in this paper, classical sputum smears may be positive only in 16% of the cases.

Considering that a disease like TB and the mycobacteria killed one billion people between 1850 and 1950 alone, not to mention the 1.7 million it now kills each year – there is really no need to look for exotic viruses, which we still have not proved are not merely passenger viruses, to explain or scrutinize a deadly "viral" pneumonia with similarities to TB and the mycobacteria beyond coincidence.

Conflict of Interest

The author has no conflict of Interest to report

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