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3-24-2020 The Current Coronavirus Pandemic: It's Likely Caused By More Than One Pathogen And There Is a Missing Antidote, by Bill Sardi

By Bill Sardi March 24, 2020

The reality is, most of the cases of COVID-19 coronavirus infection have been diagnosed without confirmation by laboratory testing. And so, doctors end up finding what they are looking for – COVID-19. The singular mindset of modern medicine continues to be practiced today. COVID-19 fits a script that was created prior to this current epidemic. That is because they want to develop a single-component vaccine and inoculate the entire planet at one time (the <u>dream of a billionaire philanthropist</u>). So, better get sick to make his dream come true (NOT!). Somehow, vaccine makers were reportedly making a COVID-19 coronavirus vaccine long before the first outbreak in China.

Similarly, most virologists collectively believe the Spanish flu of 1918 was caused by an influenza virus, with some level of certainty, maybe $\underline{\mathsf{H5N1}}$. By the way, the US had a population of 103 million at the time and an estimated 550,000 Americans died of the Spanish flu, which is \sim a 0.5% death rate (half of one percent). Today the US has a population hovering around 325 million and a 0.5% death rate would result in 1,625,000 deaths. But let's recall, 1918 was an era before <u>chlorinated water</u> (eradicated cholera, typhoid and dysentery), and before <u>nutrient-fortified foods</u>. But we also have more older adults (12+ million over age 80), the primary at-risk group for COVID-19 mortality.

Logic test

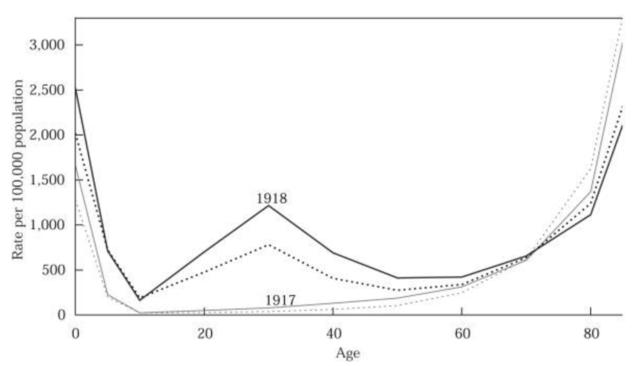
So, let's test your logic. Maybe you can a think a little less narrowly than doctors do. If a youngster runs across the road wearing tennis shoes and gets hit by a car and dies, and when we study this type of accident, we find 83.5% of kids who die in pedestrian accidents were wearing tennis shoes.

Then it is obvious the tennis shoes were a causal factor in these deaths, right? Obviously, that is flawed reasoning. The tennis shoes were involved (associated with) these deaths but not causal. This type of flawed reasoning is precisely what is going on today with the COVID-19 flasco. We have bumbling politicians managing a major disease. And doctors with blinders on.

What caused the 1918 flu pandemic?

<u>Demographers at UC Berkeley went back and analyzed</u> the facts surrounding the 1918 Spanish flu. They found the age group that was the primary target and the male/female composition of deaths fit the diagnosis of tuberculosis, not influenza. They claim tuberculosis along with influenza were behind the many millions of deaths, not H5N1 influenza alone.

So molecular biologists dug into the Alaskan permafrost to obtain samples from human bodies of the flu strains in circulation in 1918 and determined they were closest to avian strains of the flu as well as swine strains. But that didn't answer why the 1918 epidemic was so virulent, nor did it answer why young males were its predominant victims.



Age specific death rates for influenza and pneumonia combined, 1917-18.

Males (solid line); females (dotted line).

The above profile more aptly fits the epidemic now underway in <u>Italy where</u> younger immigrants (age 18-44) are dying of what obviously is not COVID-

19 but tuberculosis that was latent and erupted due to low vitamin D blood levels from the cold winter weather and the fact these migrants were darkskinned and don't synthesize vitamin D in their skin as Caucasians do.

But in Italy we also have massive death among the very old. This is explained by age and health habits, with many 80+ year-old Italians being wine drinkers and smokers, destroyers of the immune system.

Here is the chart showing the death rate of males in 1920, two years after the Spanish flu pandemic.

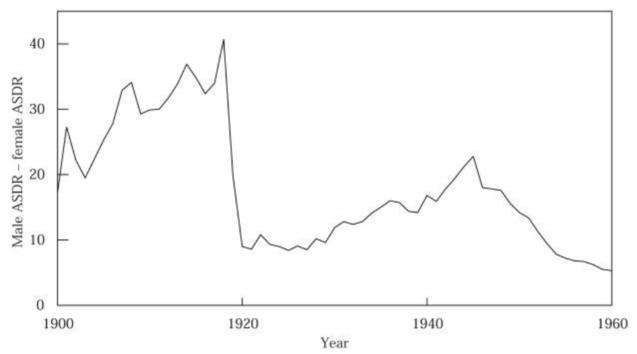


Chart interpretation: male death rate for tuberculosis plunged in 1920 because the flu had vanished. Americans had developed antibodies to the flu.

So demographers think <u>TB and influenza interacted in 1918</u>. Demographers think TB rendered it easier to contract the flu. In other words, a non-singular understanding of the 1918 pandemic.

In the words of UC Berkeley demographers:

"We conjecture that many influenza deaths in 1918 took place among the tuberculous—persons with clinical disease or latent infection with Mycobacterium tuberculosis. That the 1918 influenza virus, known to be atypical, should interact Page 3 of 7

pathologically with M. tuberculosis seems likely... Excess male flu mortality is consistent with the differential incidence of TB by sex. The fact that flu deaths had a mode in the 25–34 age group is also strongly indicative of a TB interaction; TB is a disease of adulthood, not of old age."

The <u>UC Berkeley demographers go on to say</u>:

"...The link between influenza and TB may include a third pathogen. Tuberculosis infection causes lung cavities to form, which become a breeding ground also for non-TB bacteria, including Staphylococcus aureus. This would have had the effect of priming tuberculous individuals for S. aureus superinfection in the event of coinfection with influenza."

We know there are <u>no trademark clinical features of COVID-19 infection</u>. That is why diagnosis is so difficult. COVID-19 tests are notoriously inaccurate. <u>Unreliable tests are used to confirm COVID-19 coronavirus</u>. Confronting the new coronavirus infection known as coronavirus disease 2019 (COVID-19) is challenging and requires excluding patients with suspected COVID-19 who actually have other diseases. Due to unreliable tests for COVID-19 and the need to rapidly diagnose and treat patients with in rapid physical decline, symptomology is paramount in hospital diagnosis.

In December of 2019 a <u>lower respiratory tract (lung) illness accompanied by fever of unknown origin</u> was reported in a cluster of patients in Wuhan, China. Because of prior outbreaks of coronavirus infections in China, coronaviruses were initially suspected and a newly mutated strain was identified that human populations had no immunity towards. This infectious disease was coined COVID-19 and is usually diagnosed by symptomology (dry cough, fatigue, muscle aches, fever, with chest x-rays revealing pneumonia (fluid in the lungs) and an accompanying ground-glass appearance in the lungs being a hallmark sign.

There are two symptoms of interest that pertain to COVID-19:

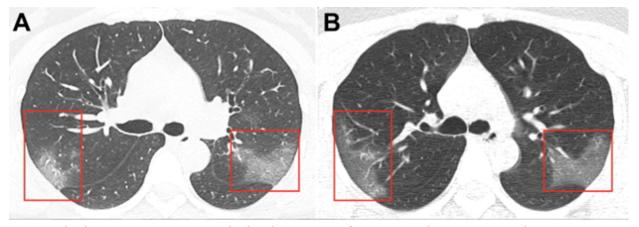
1st symptom

The hallmark, but not universal, sign – – of COVID-19 are opacities in the lower lungs that appear like ground glass in a lung scan. [The term **miliary opacities** refers to innumerable, small 1-4 mm pulmonary nodules scattered throughout the lungs.] It is useful to divide these patients into those who are febrile and those who are not. Additionally, some military opacities are very

dense, narrowing the differential - see multiple small hyperdense pulmonary nodules. These opacities represent fluid in the lung space which prevents oxygen from being transferred to hemoglobin in red blood cells, resulting in shortness of breath.

Ground-glass opacities were reported in up to 86.1% of 101 COVID-19 coronavirus cases of pneumonia. Patients with extensive ground-glass opacity (greater than 50%) are more likely to experience a longer hospital stay suffer acute respiratory failure than those with less ground-glass opacity.

Here is what ground-glass opacities look like in a lung scan (inside red boxes).



Ground glass opacities are linked to use of toxic and non-toxic drugs. More than 600 drugs known to cause lung toxicity. Many of the drugs used in the hospital to treat infectious lung diseases are toxic to the lungs! Sedatives and tranquilizers may induce pneumonia. The more medicine doctors prescribe the more likely you run into one of these hundreds of lung-toxic drugs.

The appearance of ground-glass lung opacities are far more likely among blacks who characteristically have lower vitamin D levels than Caucasians, and are less common in summer months when sunshine vitamin D levels are higher, and is <u>4.3 times more likely among individuals with vitamin D deficiency</u>.

This is just one reason why hospitalization should be avoided at all cost. Another reason is to avoid treatment errors and antibiotic resistant bacteria that live in hospitals. And because hospital rooms are dark caves where sun-starvation induces vitamin D deficiency. All patients with suspected infectious lung disease should be screened for vitamin D deficiency upon hospital admission. This isn't being done.

2nd symptom

2. Crackling sounds in the lungs are the second symptom of interest. By definition, cracklesare the clicking, rattling, or cracklingnoises that may be made by one or both lungs of a human with a respiratory disease during inhalation. They are heard only with a stethoscope ("auscultation"). You can hear crackles in an online video.

https://www.youtube.com/watch?v=LHqqvrm2j6g

Crackle sounds in the lungs are not specific for any particular infectious disease but they are widely associated with..... vitamin B1 deficiency. Here is the evidence:

- Patients with <u>beriberi</u> (<u>vitamin B1 thiamine</u>) <u>deficiency are known to have crackle sounds in their lungs</u>.
- The World Health Organization lists <u>lung crackles in the lower lungs as</u> <u>sign of thiamine deficiency</u>. B1 deficiency is also known as beriberi.
- Many hospitalized patients are placed on water pills (diuretics) to control blood pressure with no regard for the fact this class of drugs deplete vitamin B1.
- Infections were reported among 35 of 68 patients who had pneumonia. Infection may be the presenting manifestation of vitamin B1 deficiency.
- Cases of pneumonia were frequently reported in <u>prisoner of war camps</u> when <u>beriberi developed</u>.
- A 73-year old man with <u>beriberi breathing had crackles in both lungs</u> which resolved with vitamin B1 therapy.
- Vitamin B1 helps to <u>limit the growth of the bacterium that causes</u> tuberculosis.
- Patients with <u>tuberculosis are more likely to have low vitamin B1 blood</u> levels.

 One published scientific report refers to <u>thiamine (B1) as a "super</u> antibiotic."

Nicotine and alcohol use deplete essential nutrients like vitamin B1 and vitamin C. In China, 68% of men are smokers and 46% drink alcohol.

What may be missed by acute care physicians is that fever with pneumonia among some patients may not be induced by a germ but may be induced by a vitamin B1 deficiency that results from loss of control of body temperature by the hypothalamus in the brain. Thiamine deficiency can result in high fever. Vitamin B1 injections may eradicate infections. A fever accompanied by vitamin B1 malnutrition may emanate from dysfunction of the hypothalamus in the brain with accompanying lack of nitric oxide, a transient gas in the blood circulation needed to quell infections.

Many coronavirus-infected patients are alcoholics. Alcohol induces vitamin B1 deficiency. In many cases of pneumonia <u>no bacterial or viral infection is identified</u>. Vitamin B1 therapy should be a standard therapy for any patient with lung disease and a history of alcohol or tobacco consumption should call for vitamin D testing upon hospital admission.

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