Influenza and the Tuberculosis Connection

An influenza virus was not the real culprit in the 1918–19 flu pandemic. More likely, hidden tuberculosis was to blame for the devastating secondary infections and huge death toll.

Part 1 of 2

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Several Medline studies have shown that inoculating with influenza vaccine is also protective against tuberculosis. New evidence and older historical findings which explain this phenomenon bring up the possibility that influenza doesn't originate from a virus.

How a Microbe Was Repackaged and Sold as a Virus

Pfeiffer's influenza bacillus, a bacterium, was heavily isolated at one time or another in victims of the 1918–19 pandemic by practically all major research centres in the United States. It was therefore considered, by most, to be the cause of influenza until at least 1933. And Bradford, Bashford and Wilson's influenza isolates, although called "viruses" because they easily passed through a filter, were obviously minute bacteria, isolated from the blood, sputum and spinal fluid in a number of pandemic cases.[1] Bradford never named the bacteria that he and his colleagues had found. But his description of them was not inconsistent with Löhnis's minute forms of the mycobacteria, the precise name originally given to Pfeiffer's influenza bacillus, which was classified *Mycobacterium influenzae* soon after its discovery. The nearest competitor of Pfeiffer's bacillus was a vague filterable "virus".

Although the term *virus* has existed since 1898, the infectious agent it was attempting to describe was so unclear and mysterious that for many decades scientists considered it purely theoretical. Certainly, even by 1917, "influenza" was still not felt to be serious enough to be a reportable disease, and no doctor had to report it to state or local health officials. Most cases were self-limiting and gone in 10 days. Yet the great "influenza" pandemic that swept the world in 1918–19 may have been the most virulent outbreak in history, at least in terms of the swiftness of its devastation. It killed more than 20 million persons around the world, including some 550,000 in the United States—all within two years.

By 2000, Dr Andrew Noymer and Michel Garenne, demographers from the University of California, Berkeley, thought they knew just why this perceived discrepancy existed, reporting convincing statistics showing that undetected tuberculosis (TB) may have been the real killer in the 1918 flu epidemic. Aware of recent attempts to isolate the "influenza virus" from human cadavers and their specimens, Noymer and Garenne concluded: "Frustratingly, these findings have not answered the question why the 1918 virus was so virulent, nor do they offer an explanation for the unusual age profile of deaths."[2]

Somehow overlooked in today's revisionist history of the flu, the influenza bacillus or Pfeiffer's bacillus, discovered by Pfeiffer and Canon in 1892, was originally named *Mycobacterium influenzae* because it was thought to be related to *Mycobacterium tuberculosis*. Both mycobacteria stained best with carbolfuchsin and methylene blue—bacterial stains that Robert Koch himself used in the discovery of tuberculosis. Also, Grassberger[3] observed the same branching fungal forms in Pfeiffer's influenza mycobacteria as Metchnikoff[4]

had first seen in tuberculosis. Such fungal-like forms are the hallmark of the mycobacteria (their prefix "myco" means "fungal"). Mycobacteria such as tuberculosis are particularly deadly precisely because they share properties of the fungi as well as bacteria.

More recently, in 1999, Fredj Tekaia, of the Pasteur Institute, Paris, and colleagues, looking for "overall gene similarities as signatures of common ancestry", found similar genetic profiles and sequencing for Pfeiffer's bacillus (*Mycobacterium influenzae*) and *Mycobacterium tuberculosis*, lumping them together in the same "well-defined group".[5] Tekaia's diagrammatic genomic tree shows the two organisms directly next to one another. This reopened the historical argument that Pfeiffer's bacillus and tuberculosis are related.

In 1933, the very year that Smith, Andrewes and Laidlaw claimed stake to the discovery of the human influenza "virus", Stobie, in the *British Medical Journal*, still acknowledged that, rather than being a virus, the real nature of "influenza" could well be a form of *Mycobacterium tuberculosis*.[6] Stobie simply reflected the active, vigorous, yet historically suppressed debate that had been raging in medical journals for decades. He mentions cases of tuberculosis following influenza which together exhibited "a sinister type of disease which rarely responded to treatment". Enter "galloping consumption", the most devastating form of tuberculosis, then called "consumption".

Why these findings... in and after the pandemic of 1890, were ignored by the historians, scientists and practitioners of 1918 is beyond comprehension.

Obuchow Hospital, St Petersburg, Russia, 1890

In 1890, a fierce "influenza" pandemic struck worldwide, killing approximately a million people. Occurring at the end of the 19th century, this second most severe influenza ever to hit the world occurred at a time when there was fear that tuberculosis would destroy the civilisation of Europe.

Twenty-eight years later, those who survived that pandemic and lived to experience the Great Pandemic of 1918–19 tended to be less susceptible to the disease. But the lessons of 1890 were poorly understood and therefore not carried over to 1918.

Of all the forms of "influenza" known in 1890, none was more dreaded nor struck more terror into the hearts of victims and their families than that described by Wiltschur[7] as "galloping consumption". An attending physician at the Obuchow Hospital, St Petersburg, Wiltschur tells what happened when influenza punctuated previous or active cases of tuberculosis: "The [influenza] patients were, for the most part, still well nourished." This mirrored the swine flu episode of 1918, where young healthy soldiers were suddenly decimated by disease. Wiltschur continued: "Cyanosis of the face and extremities was a frequent occurrence." Patients exhibited severe difficulty in breathing (dyspnoea), an extremely high temperature not characteristic of the flu, pulmonary haemorrhages and a rapid progression of lung disease, "with death occurring in many instances unexpectedly and suddenly".

Why these findings, including the well-known rapid fatality of "galloping consumption" with its high fever, profuse haemorrhaging, brownish spots or splotches on the face, strawberry tongue and typhoid-like symptoms, documented so clearly in and after the pandemic of 1890, were ignored by the historians, scientists and practitioners of 1918 is beyond comprehension.

Historian/researcher René Dubos, of the then-named Rockefeller Institute for Medical Research, would later confirm the galloping acceleration between influenza and tuberculosis in the laboratory.[8] Dubos also assured us that "galloping consumption" was not an isolated, but a *frequent* diagnosis in the 19th century.[9]

Despite persistent myths to the contrary, in the early phase of any new TB epidemic, perhaps from a new strain, tuberculosis manifests itself as an acute disease and only much later as the chronic pulmonary tuberculosis that we know in today's western world. An example can be found in the high mortality during the 1918 "influenza" pandemic, when African Americans were brought to fight in France during World War I, large numbers of them dying from the accelerated tubercular "galloping consumption" of yesteryear. But was it only this specific group that was affected circa 1918? There has been much documentation that in certain cases, depending on the virulence of the tubercular strain, the infection can spread rapidly, causing a disease both acute and fatal, with signs and symptoms so unspecific that a proper diagnosis is impossible to make.

College of Medicine and Surgery, Philippines, 1919

At the beginning of January 1920, an important study[10] appeared in The Rockefeller Institute's *Journal of Experimental Medicine*. Physician Herbert Windsor Wade, an American investigator working out of the Department of Pathology and Bacteriology at the University of the Philippines College of Medicine and Surgery, doubted that a virus had anything to do with influenza. And Wade, working with Filipino national Dr Christobal Manalang, proved this in 1919 in their university laboratory.

Richard Pfeiffer still insisted that his organism "had the best claim to serious consideration as the primary etiologic agent [cause], and its only competition is an unidentified filterable virus".[11] Had Pfeiffer studied Wade

and Manalang's laboratory evidence, his reply would have been quite different. Pfeiffer's bacillus itself had a filterable virus-like form that could easily be mistaken for a "filterable" influenza "virus".

Wade remained a voice of scientific reason throughout the 1918 influenza pandemic, during which he personally experienced a scourge in which, depending upon which province he visited, from 40 to 95 per cent of Filipinos had contracted the disease. At least 70,000–90,000 of them had already died. Wade knew what it was like to come into a village where there were not enough living to bury the dead. Also, being far from the United States, he was not subject to the relentless censorship of the Wilson administration, both against civilians and scientists of the US Army Medical Corps itself. If he saw mycobacterial forms similar to those of TB in Pfeiffer's influenza bacillus, he could and would report them without fear of being accused of fuelling the flames of hysteria. And in documenting tuberculosis-like fungal forms in previously filtered Pfeiffer's, that is exactly what Wade did.

When the French chemist/microbiologist Louis Pasteur was on his deathbed, his conscience finally overtook him, forcing him to admit that his great rival, biochemist and MD Antoine Béchamp, had been correct and that he had been wrong. Not only were microbial forms changeable, but they depended upon the culture media or the environment in the body's terrain. So with his last dying breath, Pasteur whispered: "The terrain is everything…"[12]

Nowhere else had this become more obvious than with the mycobacteria, in which, depending upon the culture media used, either fungal or bacterial elements could be grown out. Wade's media grew out fungal forms after its spores went through a filter. They were therefore "filterable", but they were not "viruses".

In the words of microbiologist Milton Wainwright, Wade and Manalang had now committed "the ultimate pleomorphist heresy",[13] documenting that a bacterium or mycobacterium could have more than one form in its life cycle. Pfeiffer himself, when he discovered his bacillus, had seen these same forms but described them as "pseudo" influenza. Pfeiffer simply had failed to see both forms of his discovery as one and the same, central to the nature of his influenza bacillus.

It was no accident that Wade and Manalang's paper was published by The Rockefeller Institute. Simon Flexner, then director at the Institute, once followed similar acid-fast fungal forms associated with tuberculosis and found this similar association with the influenza bacillus fascinating. Flexner called his filterable fungal forms inside TB "pseudo-tuberculosis".[14]

Flexner was a key figure in the 1918 pandemic, heavily influencing Rockefeller research. He also had full status in the close-knit band of major US research operatives during the pandemic. Why had he not taken the cue and run with Wade's study? Wade had found that Pfeiffer's mycobacterial fungal forms could appear as filterable viruses. Even a 1918 B*ritish Medical Journal* editorial (1918: 2:665) had supported this.

Actually, one researcher had listened carefully to Wade. But since that researcher wasn't a member of the US scientific hierarchy, he would be ignored. His name: Dr Victor Conrad von Unruh.

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Medical Reserve Corps, NYC, 1917

Physician/researcher Victor Conrad von Unruh was born in 1868 in Dahlewitz, Germany, at a time when German medical research and science were unrivalled. He immigrated to the US, and by 1917 he had received the commission of Captain in the Medical Reserve Corps, New York City. The pandemic of 1918 was about to hit hard. Von Unruh's "A Comparative Study of the Acid Fast Bacilli"[15] had appeared in 1916, two years prior to the killing fields of 1918. His study can be found in the *Catalogue of the Library of The Surgeon General's Office – United States Army*, 1920—a reminder for posterity.

Because Pfeiffer's bacillus stained acid-fast, von Unruh, like others, had been evaluating what medical texts such as Stengel's[16] referred to as M*ycobacterium influenzae*, also known as B*acillus influenzae*. The entire influenza group was thought to be caused by this acid-fast mycobacterial bacillus, which was similar to the tubercle bacillus. Both microbes had fowl, swine and human forms.

Von Unruh never saw the need to look for a "filterable virus" or "influenza" in the thousands of hogs that died abruptly with flu-like symptoms just before the pandemic, as did virologist Richard Shope. Why should he? Shope, the American virologist credited with the first isolation of the influenza "virus", seemed oblivious to the fact that more than 60 per cent of hogs circa 1918 were tubercular from fowl TB, a fact that gave hog breeders such concern that large-scale efforts were under way to rid farms and chicken flocks of avian tuberculosis. The

situation had become so grave in hogs and cattle that by 1917, one year before the most destructive pandemic ever, the Cooperative State–Federal Tuberculosis Eradication Program, administered by the US Department of Agriculture and the Animal and Plant Health Inspection Service, had to be instituted. For in 1917, it was estimated that 25 per cent of deaths from tuberculosis in adult humans were caused by animal tuberculosis.[17] Nor did the fact that swine freely infected humans and vice versa faze von Unruh. Swine were a mycobacterial laboratory; although they held primarily fowl tuberculosis, they could also acquire bovine and human forms and freely infect people.

In "A Comparative Study of the Acid Fast Bacilli", von Unruh stresses that the many cases of influenza he had investigated contained both the resting (dormant) form of TB and the influenza bacillus. Although Pfeiffer had likewise documented chronic colonisation with his bacillus in TB patients, von Unruh saw this, and the fact that they were both mycobacteria was more suggestive of "a common ancestry or origin". Von Unruh wrote: "We have in influenza the fever, malaise, loss of weight, invasion by the organism of the same anatomical structures as in tuberculosis; we have chronic cases of bronchitis in which the influenza bacillus is constantly present; and lastly, we know that typical tuberculosis often follows an attack, however mild, of influenza."[15]

They never for a moment considered that this unknown primary cause could be tuberculosis, which not only spawned secondary infection with strep, staph and pneumococci but Pfeiffer's bacillus itself.

Such tubercular infection could in turn lead to other secondary bacterial infections. Noymer and Garenne's statement that tuberculosis was behind the many deaths in the 1918 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. Noymer and Garenne wrote: "It is highly plausible that TB infection laid the ground for the massive secondary bacterial pneumonias that killed the victims of the flu in 1918."[2]

The Reichsgesundheitsamt, Berlin, March 1882

Although it has always perplexed doctors, scientists and historians alike as to why so many microbes were involved during the deadly 1918 epidemic, the explanation was laid out clearly by the German physician/microbiologist Robert Koch, discoverer of tuberculosis, decades before. From the onset, Koch concluded[18] that other micro-organisms shared in the destructive work of the tubercle bacilli. Gaffky, Pansini, Cornet, Spengler, Schabad, Ortner and Flick, among others, agreed.[19]

Various organisms, all of which reappeared to confuse scientists in 1918, were assigned a share in the clinical picture of tuberculosis—among them the S*treptococcus* and P*neumococcus* micro-organisms and even the influenza bacillus itself. The pyogenic, pus-forming cocci were more generally suspected than other bacteria of complicating tuberculosis. In the B*ritish Medical Journal* of 28 July 1900, the following editorial appeared, dealing with the role of streptococci in tuberculosis: "It is a remarkable fact that…the bulk of the disturbing and dangerous features of tuberculosis are not due to the tubercle bacillus, but to streptococci and other pyogenic organisms." The pneumococci, staph and methycillin-resistant staph (MRSA) are other pyogenic pathogens that subsequently have been documented.

British pathologist William Crofton chafed at the ridiculous notion that in influenza, Pfeiffer's bacillus and only Pfeiffer's bacillus alone should be found in pure cultures. He asked whether the typhoid bacillus was ever found in pure culture. But because Pfeiffer's was often found with the very same infections secondary to tuberculosis, such as from streptococci and pneumocci, investigator after investigator during the Great Pandemic of 1918–19 was coming to the curious conclusion that Pfeiffer's bacillus was not the primary cause but somehow was awakened to activity by some unknown primary cause. They never for a moment considered that this unknown primary cause could be tuberculosis, which not only spawned secondary infection with strep, staph and pneumococci but Pfeiffer's bacillus itself. Besides this, the obvious: microbes like streptococci, pneumococci and staphylococci could produce epidemics, but never *pandemics*. What was the source of this confusion?

Medical Reserve Corps, New York City, July 1918

Victor von Unruh continued to see many reasons to pin a common ancestry onto the two organisms. Both the influenza bacillus and the quiescent TB formed Much's granules. Much's granules, named after their discoverer, Hans Much, passed through filters—then a major criterion for diagnosing a virus. Furthermore, both the influenza bacillus and TB were mycobacteria, with the branched fungal forms characteristic of the mycobacteria. And both could stain with "acid-fast" mycobacterial stains.

Von Unruh wrote: "Therefore my conclusion is that the

influenza bacillus is merely a weaker or dwarfed form of the real tubercle bacillus, a strain that in this case failed of better development because of a higher degree of resistance in the host. In both tuberculosis and influenza we deal with the self-same organism that in tuberculosis is fully developed, while in influenza it lacks development. In other words, we are dealing with a difference in degree only, but not in kind."[15]

Von Unruh's distinction is suggestive of a similar, well-acknowledged comparison between the damaging effects that *Mycobacterium tuberculosis* has inflicted on man for centuries and the better resistance that humans with a healthy immune system have had against bird or fowl tuberculosis (*Mycobacterium avium*), found in swine as well. Did types of fowl tuberculosis in the form of *Mycobacterium* (Haemophilus) *influenzae suis* from pigs combine additively with latent human tuberculosis to cause the deadly galloping consumption of 1918–19?

Victor von Unruh's findings could have been taken lightly, were it not for similar thoughts published in the more prestigious medical journals of his time.

Did types of fowl tuberculosis...from pigs combine additively with latent human tuberculosis to cause the deadly galloping consumption of 1918–19?

Bureau of Laboratories, 1918

Something was eating at physician/researcher/editor William Park during the carnage of 1918. Park, heading the state-of-the-art Bureau of Laboratories in New York City, was relied upon by both the US Government Health Corps and other major research centres across the country. Along with collaborators, including bacteriologist Anna Williams, Park co-authored the important teaching text, *Pathogenic Microorganisms*.

At first, Park blamed Pfeiffer's bacillus for the 1918 pandemic and reported it to the US Army Health Corps. But flustered by the secondary infections and lack of consistency in other labs in isolating Pfeiffer's, Park flip-flopped, cautioning against attributing the pandemic solely to Pfeiffer's.

By the same token, Park felt uncomfortable with the muzzling of the media and medical corps by the Wilson administration regarding the ferocity of the 1918 American epidemic in general—that is, until it was too late to deny its viciousness.

So although Pfeiffer's influenza bacillus, also known as *Mycobacterium influenzae*, was no longer at the top of his choices for the causal agent of the 1918 pandemic, it was its association with another mycobacterium, tuberculosis, which bothered Park the most. He had just re-read Flick's account of what preceded both Great Pandemics.

In 1888, physician Lawrence Flick, citing US census reports leading into the two greatest world "influenza" pandemics in history, reported that out of every 1,000,000 deaths, 242,842 males and 302,046 females died of tuberculosis. This was for all nationalities and colours. Specific subsets within these statistics revealed that among African Americans, every million deaths represented 248,179 males and 326,973 females having died of tuberculosis. Among people of Irish parentage, 309,507 males and 375,636 females died of TB for every million deaths. And among people of German parentage, its victims numbered 249,498 males and 254,958 females for every one million deaths.[20]

Although Park had seen these statistics before, their effect was not lost on him: "Consumptives [people with tuberculosis] frequently carry influenza bacilli [Pfeiffer's bacilli] for years and are particularly susceptible to attacks of influenza."[21]

In 1918, with "flu" victims dropping all around them, John B. Hawes, MD, of the Massachusetts General Hospital, and Richard Cabot, MD, of Harvard, wrote: "One of the diseases most frequently mistaken for pulmonary tuberculosis is influenza, chronic or acute." According to Hawes and Cabot, the symptoms of both diseases were often identical.[22]

Tufts pulmonologist Edward O. Otis also caught Park's attention. Otis not only mirrored Hawes and Cabot's view, he went a step further: "Often a patient gives the history of a previous attack of influenza which may have been an active outbreak of a latent tuberculous focus, which later again became inactive."[23]

Back in 1901, an editorial in the *Journal of the American Medical Association* specifically cited Liverpool physician R. Buchanan: "Dr R. J. M. Buchanan makes the not improbable suggestion that many of the so-called sporadic cases of influenza are really symptomatic of the initial infection of tuberculosis, or possibly an exacerbation of a latent tuberculosis previously unsuspected or undetected."[24]

In saying this, Buchanan had fired the shot heard around the world, and physician Walter Lindley, editor of the *Southern California Practitioner*, was quick to respond. Lindley on Buchanan: "The author, impressed by the large number of instances in which patients have referred the commencement of their ill-health to an attack of so-called influenza, conceives that many of the so-called sporadic cases of influenza are really symptomatic of the initial infection of tuberculosis or possibly an exacerbation of a latent tuberculosis."[25]

A complaint by Wakley and Wakley in the *Lancet* in 1899 regarding an epidemic of infectious fever raging in New York is also appropriate: "The name 'influenza' seems to have a strong attraction for some people. Every ache and pain, no matter where located and whether accompanied by fever or not, is at once put down as

'influenza'; every headache, every coryza [nasal congestion, common cold], every sore-throat, every attack of gastroenteritis, from whatever cause, is promptly self-diagnosed as 'influenza', and when the practitioner arrives upon the scene he will be expected to fall in with this view, and there is a great temptation to do so."[26] The authors go on to suggest that the rise in temperature and general malaise frequently met with in this New York epidemic wasn't influenza at all.

Hendrickson, who practised during the Great Pandemic of 1918–19, spoke more bluntly: "No doubt there were many cases of tuberculosis whose death certificates were labeled influenza during the pandemic owing to lack of time to make a diagnosis by the overworked physician."[27]

He reiterates that when an attack of influenza intervenes in a patient suffering from pulmonary tuberculosis, the tuberculosis process is likely to be aggravated "and very often terminates fatally in a comparatively short time". Of more vital interest, even in the cases of dormant, healed TB in which the individual has contracted the disease in childhood, he quotes Osler on influenza being "an important exciting cause" of latent tuberculosis.[28] At the same time, Hendrickson clarifies why in certain US Army barracks in 1918, measles—in marked analogy to influenza—could also cause the reactivation and rapid spread of dormant tuberculosis.

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Hendrickson, in 1918, therefore told American science all it needed to know about the pandemic, but it just wasn't listening. Epidemic measles had kicked things off in Army barracks before the influenza epidemic, during the bitterly cold winter of 1917–18. But unlike childhood measles, it was deadly, racking its victims with high fever, intractable cough, savage earaches and even brain inflammation. As with tuberculosis, the measles didn't kill by itself; rather, it killed by the very same secondary bacterial infections that dogged TB-infested lungs.

So, even prior to Pfeiffer's bacillus becoming the focus of national attention in the "influenza" epidemic, upwards of 5,000 out of approximately 30,000 barracks soldiers died of measles "complications" on American soil. At Camp Shelby, Mississippi, alone, almost half of the deaths from all reportable causes were linked to pneumonia secondary to the measles. American newspapers began to headline a measles fiasco, and Army Surgeon General William Gorgas was called to task.

Hendrickson warned American medicine that both measles and then Pfeiffer's bacillus were nothing but red herrings landing on an underlying tubercular infection. But American medicine wasn't listening.

Continued in the next issue of NEXUS...

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